Sri Lakshmi Narayana Institute of Medical Sciences

Date: 03.05.2019

From
Dr. M Kalasree
Head Of Department Incharge
Department of Anaesthesia
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research
Puducherry

To
The Dean,
Sri Lakshmi Narayana Institute of Medical Sciences
Puducherry

Sub: Request for Permission to conduct value-added course: Fluid Therapy

Dear Sir,

With reference to the subject mentioned above, the department proposes to conduct a value-added course titled: Fluid Therapy for undergraduates from July -Dec 2019. We solicit your kind permission for the same.

Kind Regards

Dr. M KALASREE

Head of Dept. Anaesthesiology
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry - 605 502

FOR THE USE OF DEANS OFFICE

Names of Committee members for evaluating the course:

The Dean: Dr JAYALAKSHMI

The HOD: Dr. M KALASREE

The Expert: Dr CHANDRASEKAR

The committee has discussed about the course and is approved.

Dean

Subject Expert

HOD

Dr. JAYALAKSHMI, BSC.,MBBS.,DTCD.,M.D.
DEAN
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Ageram Kudapakkam, Post,
Villianur Commune Puducherry-605 502.

DEPARTMENT OF ANAESTHESIOLOGY
SRI LAKSHMI NARAYANA INSTITUTE
OF MEDICAL SCIENCES
OSUDU, KUDAPAKKAM, PUDUCHERRY-605 502.

Head of Dept. Anaesthesiology
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry - 605 502

OFFICE OF THE DEAN

Sri Lakshmi Narayana Institute of Medical Sciences

OSUDU, AGARAM VILLAGE, VILLIANUR COMMUNE, KUDAPAKKAM POST,
PUDUCHERRY - 605 502.
[Recognised by Medical Council of India, Ministry of Health letter No. U/12012/249/2005-ME (P -II) dt. 11/07/2011]
[Affliated to Bharath University, Chennai - TN]

Circular

07.06.2019

Sub: Organising Value-added Courses: Fluid Therapy - reg

With reference to the above mentioned subject, it is to bring to your notice that Sri Lakshmi Narayana Institute of Medical Sciences, **Bharath Institute of Higher Education and Research**, is organizing **"_Fluid Therapy"** course in July- Dec 2019. The course content is enclosed below."

The application must reach the institution along with all the necessary documents as mentioned. The hard copy of the application should be sent to the institution by registered/ speed post only so as to reach on or before 15/06/2019. Applications received after the mentioned date shall not be entertained under any circumstances.

Dean

Dr. G. JAYALAKSHMI, BSC.,MBBS.,DTCD.,M.D.,
DEAN
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Ageram Kudapakkam. Post,
Villanur Commune Puducherry-605 502.

Encl: Copy of Course content

COURSE PROPOSAL

Course Title: Fluid Therapy

Course Objective:

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1. To enable the students to learn about normal fluid and electrolyte balance and to understand the various abnormalities associated with its imbalance and measures to correct it.

2. To know about the newer available fluids and newer regimens of fluid correction

Course Outcome:

On successful completion of the course the students will have a basic understanding of how to calculate fluid deficit in patients and to decide on replacement strategies for the deficit.

Course Audience: IV MBBS

Course Coordinator: Dr. M KALASREE

Course Faculties with Qualification and Designation:

1. Dr Kalasree: Associate Professor.

2. Dr Chandrasekar: Assistant Professor

Course Curriculum/Topics with schedule (Min of 30 hours)

S.No	Date	Topics	Time	Hours	Faculty
1	14.07.2019	Introduction	2-4PM	2	Dr Chandrasekar
2	21.07.2019	Goals of fluid therapy	2-4PM	2	Dr Kalasree
3	28.07.2019	Composition of body fluids	2-4PM	2	Dr Chandrasekar
4	04.08.2019	Fluid requirements	2-4PM	2	Dr Kalasree
5	11.08.2019	Electrolyte requirements	2-4PM	2	Dr Chandrasekar
6	18.08.2019	Renal regulator mechanisms	2-4PM	2	Dr Kalasree
7	25.08.2019	Replacement strategies	2-4PM	2	Dr Chandrasekar
8	01.09.2019	Crystalloids and colloids	2-4PM	2	Dr Chandrasekar
9	08.09.2019	Achievement of successful fluid resuscitation	2-4PM	2	Dr Kalasree
10	15.09.2019	Indicators of successful fluid resuscitation	2-4PM	2	Dr Chandrasekar
11	22.09.2019	Electrolyte imbalance	2-4PM	2	Dr Kalasree
12	29.09.2019	Complications	2-4PM	2	Dr Chandrasekar
13	06.10.2019	Group discussion	2-4PM	2	Dr Kalasree
14	13.10.2019	Management of electrolyte deficiencies	2-4PM	2	Dr Chandrasekar
15	20.10.2019	Assessment	2-4PM	2	Dr Kalasree

REFERENCES

1) Miller’s Anaesthesia- 8th edition.

2) The ICU book – Paul marino - 4th edition.

3) Morgan and Mikhail's Clinical Anaesthesiology - 5th Edition

VALUE ADDED COURSE

1. Name of the program & Code

Fluid Therapy, ANAES 11

2. Duration & Period

30 hrs: July 2019- December 2019

3. Information Brochure and Course Content of Value Added Courses

Enclosed as Annexure- I

4. List of students enrolled:

Enclosed as Annexure- II

5. Assessment procedures:

Multiple choice questions- *Enclosed as Annexure- III*

6. Certificate of Participation:

Enclosed as Annexure- IV

7. No. of times offered during the same year:

1 Time JULY 2019-DEC 2019

8. Year of discontinuation: 2019

9. Summary report of each program year-wise

Value Added Course -JULY 2019-DEC 2019					
Sl. No	**Course Code**	**Course Name**	**Resource Persons**	**Target Students**	**Strength & Year**
1	ANAES 11	Fluid Therapy	DR. M KALASREE	IV MBBS	20

10. Course Feed Back

Enclosed as Annexure- V

DEPARTMENT OF ANAESTHESIOLOGY SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCES OSUDU, KUDAPAKKAM, PUDUCHERRY-605 502

RESOURCE PERSON **COORDINATOR**

DR. CHANDRASEKAR **DR. M KALASREE**

Head of Dept - Anaesthesiology,
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry - 605 502.

Annexure I

FLUID THERAPY

TOPICS:

Composition of body fluids

Principle of fluid therapy

Crystalloids

Special fluids

Colloids

Guideline of fluid therapy in hypovolemia andmonitoring

Methods to calculate the rate of infusion

Water regulation

Sodium regulation

Potassium regulation

Calcium and magnesium regulation

Fluid therapy in hypovolemic shock

Fluid therapy in DKA

Fluid therapy in children

Composition of body fluids

Total body water:

Total body water content.is about 60°/o of body weight in an young adult male and about 50°/o in an young adult female. Since fat contains less water, an obese person will have proportionately less body water as compared to a lean person. In new born infants the proportion of body water in relation to weight is as high as 80°/o, which declines with age.

Distribution of body fluid :

Out of total body water two third (40o/o of body weight) is intracellular fluid

(ICF) and one third (20% of body weight) is extracellular fluid (ECF)

Oral (or l.V.) fluid intake and urine output are important measurable parameters

of body fluid balance. To determine daily fluid requirement of body we need to

know insensible fluid input and loss as summarized below :

Insensible fluid input= 300 ml water due to oxidation.

Insensible fluid loss= 500 ml through skin

= 400 ml through lung

= 100 ml through stool

Fluid loss - Fluid input - 1000 - 300 ml - 700 ml.

NORMAL DAILY INSENSIBLE FLUID LOSS= 700 ML

Fluid loss

(Abnormal)

= 500 ml. through moderate sweating

= 1.0 - 1.5 liter through severe sweating/high fever

= 0.5 - 3.0 liter through exposed wound surface

(burns) and body cavity (laparotomy)

Units of measurements

It is important to understand basic terminology used to measure concentration and composition of body fluids and their inter relationship.

Ions

· An ion is an atom or group of atoms with an electric charge.

Anion : When ion has a negative electric charge it is called anion (i.e. er HC0 3 -).

Cation : When ion has a positive electric charge it is called cation (i.e. Na+, K+, Mg+2).

Cation- "t"- + positive charge

Different ways by which solute concentrations can be measured are milligram per decilitre (mg/di), milliequivalent per litre (mEq/L) or niilliosmoles per litre or per kg (mOsmol/L or mOsmol/kg).

Equivalent and milliequivalent:

Equivalent is a relative term, it refers to mole of ionic charges.

Equivalent : An equivalent is the atomic weight in grams, multiplied by

the valence.

For ions which carry a single charged mole equals an equivalent (i.e. Na+, K+, c1·, H•). But if the ion carries charge that is greater than one, numbers are no tonger equal. For example a mole of calcium ion (Ca+2) equals two equivalents.

So equivalents = moles x valence

Comparision of normal value of serum electrolytes concentration in mEq/L and mmol/L

Molecules must be quantified in moles (e.g. 'a mole of glucose') because they carry no charge. However in practice they are usually measured in .mg or gram because of simplicity and convenience. To conve~ from mg/di to mmol/L the following formula can be used

mmol/L =

mg/di x ·1 O

Atomic weight

Ions can be quanUfied as either"moles or e·quivalents.

As concentration ·of most *of* the molecules ·and ions are very low in serum, their measurement is convenient in mmol or mEq rather than moles or equivalents. In day to day· work we use millimeter which .is

·. '1/1,000 of meter. :In same. way .mmol·or mEq is 1/1,000 of. mole or

equivalent. If we look at value. of serum potassium. it, is 0.004 mole orequivalent/L. But after conversion it is 4 mmol/L or mEq/L, which is very simple and convenient value to use in practice .

PRINCIPLES OF FLUID THERAPY

Advantage:

.*Y* Accurate, controlled and predictable way of administration.

L

Immediate response due to direct infusion in intravascular

compartment.

/. Prompt correction of serious fluid and electrolyte disturbances.

Indications :

Fluid therapy is widely used for restoration of fluids and electrolytes, as a

drug carrier and for nutrition. Most common and important indications are :

Conditions when oral intake is not possible e.g. coma, anesthesia,

surgery.

. Severe vomiting and diarrhoea.

. Moderate to severe dehydration and shock,

. Hypoglycemia where 25°/o dextrose is life saving.

.Treatment of critical problems : Shock, anaphylaxis, severe asthma

cardiac arrest and forced diuresis in drug overdose, poisoning, urinary

stone

Disadvantages :

Possible only in hospitalized patient under skilled supervision .

.Improper selection of type of fluid used can lead to serious problems .

Improper volume and rate of infusion of fluid can be life threatening.

Improper technique of administration can lead to compfications.

Contraindications :

. l.V. fluid should be avoided if patient is able to take oral fluid.

.

Preferable to avoid l.V. fluid in patient with congestive heart 'failure or volume overload.

Complications :

1.Local:

Haematoma, infiltration and infusion phlebitis.

2. Systemic :

Circulation overload with rapid or large volume infusion espe cially in patients with cardiac problem.

Rigors, air embolism and septicaemia.

3. Others :

~uidC'onta~i~ation, fungus in I. V. fluids, mixing of incompatibl e, drugs.

improper technique of infusion, 1.v. set or l.V. catheter related problems

and human error related problems

CLASSIFICATION OF I. V. FLUIDS

I. V. fluids can be *divided* into three groups.

1. Maintenance fluid 2. Replacement fluid 3. Special fluid

Maintenance Fluids :

Maintenance fluid replaces fluid lost from lungs, *skin, -urine* arid faeces. These losses are poor in salt so this maintenance *fluid* should be hypotonic to plasma sodium. Routinely used maintenance fluid is 5% dextrose, dextrose with 0.45°/o NaCl solution (dextrose with half isotonic saline).

Replacement Fluids

Formulate~ to correct body fluid deficit caused by losses such as gastric drainage, vomiting, diarrhoea, fistula drains, intestinal oedema, oozing from trauma, infection, burns etc. Commonly used replacement fluids are Isotonic saline, DNS, Ringer's lactate, lsolyte-M, P and G.

Special Fluids

.Special fluids_ are . used for the special indications such as

CRYSTALLOIDS

5%DEXTROSE

Composition :

One litre of fluid contains :

Glucose 50 grams'

2. Pharmacological Basis :

5°/o-dextrose (D-5°/o) corrects dehydration and Supplies energy. After consumption of glucose, remaining water is distributed in all compartments of body proportionately. Therefore D-5°/o is the best agent to correct intracellular dehydratign. D-5o/o is selected when there is need of water but not electrolytes.

5°/o-dextrose solution (50 gm dextrose per litre) provides 170 Kcal/L.

1 gm of hydrous dextrose supplies 3.4 Kcal.

Indications :

1. Widely used fluid for prevention and treatment of dehydration due to inadequate water intake or excessive water loss.

2. Cheapest fluid fo provide adequate calories to bodt

3. For pre and post-operative fluid replacement.

. For l.V. administration of various drugs.

5. For treatment or prevention of ketosis in starvation, diar rhoea,

vomiting and high grade fever.

6. Adequate glucose infusion proteets the liver against toxic substances.

correction of hypernatremia due to pure water loss(e.g. diabetes

-insipidus)_. Hypernatremiadue to salt poisoning or excessive use

_of electrolyte solution needs infusion of 5%-dextrose with frusemide.

_!Q. promote Na excretion and correction .of hypernatremia..,

4. contraindications

· Cerebral oedema

Neurosurgical procedures:

3. Acute ischaemic stroke : Glucose containing fluid should not be used after acute ischaemic stroke as hyperglycemia aggravate~ cerebral ischaemic brain damage.

4. Hypovolemic shock : 5°/o-dextrose is not the right fluid to select because it does not substantially increase intravascular volume. Moreover, fast replacement by large volume of D-5o/o can lead to hyperglycemia and osmotic diuresis leading to increased urine .. output. s.o correction of dehydration w·ill be delayed.

5. Hyponatremia and water intoxication : By providing . electrolyte free water 5°/o-dextrose worsens both conditions.

6. Hypernatremia : Fast infusion of 5°/o-dextrose· rapidly corrects severe hypernatremia, but this correction occurs slowly in brain cells, so swelling of hypertonic brain cells occur. This may lead to serious or permanent neurological damage. Moreover, rapid

infusion of dextrose induces osmotic diuresis, which can aggravate hypernatremia. So correction of hypernatremia should be done gradually with D-5°/o or with low sodium containing fluids.

7. Blood transfusion :

Dextrose

solution

and whole blood should not

be administered thr~ug~ the same l.V. line as haemolysis and clumping can occur.

8. Uncontrolled diabetes and severe hyperglycemia.

5. **Precautions** :

Inverted sugar solution

1. Composition :

One litre of fluid supplies :

Inverted sugar 100 gm

2. Pharmacological Basis :

Inverted sugar is a nequimolar mixture, which contains half dextrose and half fructose. Fructose is considered to be metabolized ih the absence of insulin. Therefore, it can be utilized mo,r,e rapidly than dextrose, especia lly in a diabetic patient. However, glucose is a metabolic product of fructose and requires the presence of insulin for its metabolism.

3. Indications :

1. Treatment of nausea, vomiting including vomiting *of* pregnancy.

2. In patient with liver disease it provides glucose, prevents glycogen depletion and exerts protein sparing effects.

4. Adverse effects :

Large dose of fructose can cause lactic acidosis, hyperuricemiaand hypophosphatemia.

Contraindications :

Hereditary fructose intolerance.

Cautious use in patients with impaired kidney function or severe

Less effective in treatment of hypoglycemia.

Generally more than 25 gm of fructose per dayis not

2.. Pharmacological Basis :

Sodiun1 chloride is present chiefly in extracellular fluid maintaining osn1olal-ity of ECF. So isotonic saline is used to provide major extracellular electrolytes.

During von1iting and diarrhoea along with water loss, there is a substantial loss of sodium chloride. 0.9isotonic saline is very useful to correct both fluid and electrolyte deficit.

As isotonic saline is distributed chiefly in extracellular fluid, it will increase the intr~vascularvolume substantially. There.tore, isotonic saline is a very useful l.V. fluid to raise blood pressure in the patient with hypovolemic shock.

3. **Indications** :

1. Water and salt depletion as in diarrhoea, vomiting, excessive diuresis or excessive perspiration.

2. Treatment of hypovolemic shock.

3. Treatment of alkalosis (e.g. vomiting) with dehydration.

In severe salt depletion or hyponatremia when rapid correction of sodium is necessary.

Miscellaneous :

• Initial fluid therapy in diabetic ketoacidosis.

• Treatment of hypercalcen1ia.

• Fluid challenge in prerenal ARF.

• Irrigation for washing of body fluids .

.As a vehicle for certain drugs and can be given safely with blood

Hypertonic saline 3°/o NaCl is used in treatment of hyponatremia due to SIADH or water intoxication (along with frusemide), as it provides greater sodium in lesser fluid volume (Na concentration 513 mEq/L in 3°/o NaCl versus 154 mEq/L in 0.9°/o Na.Cl). 3°/o. NaCl is available as 250 ml bottle commercially.

4. Contraindications :

1. Cautious use or avoid in . hypertensive or preeclamptic patients and in patients with oedema due to congestive heart.failure,. renal

disease and cirrhosis.

2. Careful administration to very young or elderly patients.

Dehydration with severe hypokalemia : With severe hypokalemia there is deficit of even ICF potassium so infusion of isotonic saline, without additional potassium supplementation, will aggravate electrolyte imbalance of ICF.

Dextrose with half strength saline

(5°/o-O,extrose with 0.45 °/o NaCl Solution)

1. Composition :

One litre of fluid contains :

Sodium

77 mEq

Glucose

50 gm

Chloride

77 mEq

Each 100 ml contains : Glucose

5.0 gm and Sodium chlo'ride 0.45 gm

3**. Indications** :

1. Fluid therapy in paediatric patients.

2. Treatment of severe hypernatremia: As it corrects hy~ernatremia

gently, it avoids cerebral oedema, and is therefore sa e .

. 3. As maintenance fluid therapy.

4. Early post operative period.

4**. Contraindications** :

1. Hyponatremia.

2. Severe dehydration due to diarrhoea and vomiting where there is need for larger salt replacement.

Dextrose Saline (DNS)

(5o/o Dextrose with 0.9°/o NaCl Solution)

Composition :

One litre of fluid contains :

Glucose

50 gm

Chloride

154 mEq

. Sodium

154 mEq

Each 100 ml contains : Glucose 5.0 gm and Sodium chloride 0.90 gm

Pharmacological Basis :

This fluid has advantage of both 5°/o-dextrose (to provide energy} and isotonic saline (to provide salt). So DNS is_ useful to supply major extracellular electrolytes. (sodium and chloride) and energy a'iong with fluid to correct dehydration.

Like 0.9% isotonic salin_e it rapidly corrects NaCl deficit of ECF. As DNS is distributed chiefly in ECF compartment, unlike D-5o/o it does not correct intracellular dehydration.

As bNS increases only ECF volume, it can be considered in treatment oLdehydration with hypovolemic shock. But like D-5%, faster infusion of large volume of DNS will lead to large glucose load (> 25 gm/hr)J leading to hyperglycemia induced osmotic diuresis. So in presence of incompletely or partially corrected shock patient will have increased urine output.

Unlike D-5o/o DNS is not hypotonic (due to NaCl) and hence it is compatible with blood transfusion.

3. Indications :

1. Correction of salt depletion and hypovolemia with supply of energy.
2. Correction of vomiting or nasogastric aspiration induced alkalosis and hypochloremia along with supply of calories.
3. Fluid compatible with blood transfusion.

4.. Contraindications :

1. Anasarca : Cautious use in anasarca of cardiac, hepatic and renal disease.
2. Hypovolemic shock: Not preferred in severe hypovolemic shock,

when rapid replacement with larger volume of fluid is required.

Rapid infusion of DNS can cause hyperglycemia and osmotic diuresis even in presence of fluid deficit. So the simple logic tp use DNS for supplying salt solution to correct shock and providing energy simultaneously is not correct.

Ringer's lactate (RL)

Each 100 ml contains :

Calcium

Bicarbonate

3mEq

28 mEq

Sodium lactate 320 mg, Sodium chloride 600 mg, Potassium chloride

2. **Physiological Basis** :

" Because of high sodium concentration p'30 mEq/L), Ringer~ lactate rapidly expands intravascular volume and so it is very effective in treatment of severe hypovolemia. Ringer's lactate is the most physiological fluid as its electrolyte content (i.e. Na, Kand Ca) is nearly similar to the free concentration in plasma. So even larger amount of RL can be infused rapidly without risk of electrolyte im~alance.

Sodium lactate in Ringer's lactate is metabolized in liver to bicarbonate . As RL provides bicarbonate, it is useful in correction of metabolic acidosis. As Ringer's lactate not only supplies all electrolytes but also provides bicarbonate, it is very useful in many surgical conditions .

3. Indications :

Correction of severe hypovolemia rapidly with large fluid volume.

. For replacing fluid in postoperative patients, burns, fractures, peritoneal irrigation etc.

Diarrhoea induced hypovolemia with hypokalemic metabolic acidosis is effectively treated. with RL. RL is the fluid of choice for initial treatment of diarrhoea induced dehydration even in paediatric practice.

4. In diabetic ketoacidosis, RL provides glucose free water, so corrects metabolic acidosis with added advantage of supplying potassium.

5. For maintaining normal ECF fluid and electrolyte balance during and after surgery ..

5. In vomiting or continuous nasogastric aspiration : Her~ hypovolemia is associated with metabolic alkalosis. As AL.provides bicarbonate, it wrn worsen metabolic alkalosis and, therefore, is riot preferred .

Along with blood transfusion : Calcium in HL binds with the citrate anticoagulant in blood transfusion. This can inactivate the anticoagulant and promote the formation of clots in donor blood .

. For this reason simultaneous infusion of RL and blood product in one l.V. line Is contraindicated.

The calcium in RL binds with cert~in drugs (i.e. amphotericin, thiopental, ampicillin., doxycycline etc.), and reduces their

. bioavailability and efficiency.

lsolyte-G

1. Composition : .

One litre of fluid supplies :

Glucose

50 gms

Sodium

65 mEq

Potassium

17 mEq

100 ml of fluid contains :

Chloride

Ammonium

.150 mEq

69 mEq

: NaCl 0.375 gm, KCI 0.130 gm, NH4CI 0;370 gm, Glucose 5.000 gm, Sodium metabisulphite 0.015 gm

2. Physiological Basis :

During vomiting or continuou~» nasog astric aspiration there is loss of gastric juice. Gastric juice contains 60 mEq/L so dium, 1 o mEq/L potassium and 130 mEq/L chloride along with acidic content. So vomiting or continuous nasogastric aspiration will lead to hypochloraemic, hypokalaemic metabolic alkalosis .

2. · Pharmacological Basis :

lsolyte-M is the richest source of potassium (35 mEq/L), so.very useful

to treat hypokalemia. However, always ensure good urine output or normal renal status before its infusion.

Proportion of electrolytes in lsolyte-M is almost similar to the maintenance requirements of the body. Additionally, it corrects acidosis and supplies energy. So this fluid fulfills the needs of body electrolytes, pH maintenance, caloric supply and water replacement and, so it is the ideal fluid for maintenance fluid therapy and, therefore, named

As concentration of sodium is low (40 mEq/L) in lsolyte-M, it should be avoided in hyponatremia. It is not the preferred I. V. fluid in patients with significant salt and water depletion.

3. Indications :

1. For parenteral fluid therapy, it is the ideal maintenance fluid.

2. To correct hypokalemia secondary to diarrhoea, bilious vomiting, prolonged infusion of potassium free l.V. fluids, ulcerative colitis etc.

4. ContraindiC.ations :

1. Renal failure : Cautiously used or totally avoided in presence of significant renal failure (ARF or CRF) due to potential risk of hyperkalemia.

2. Hyporiatreniia and water intoxication : As Na+ concentration of lsolyte-M is much low (40 mEq/L), it should be avoided.

AdrenoC"ortical insufficiency : These patients have abnormally high potassium concentration and, therefore, should not receive fluids with high potassium.

Burns,: In patient~ with severe burns potassium concentration may

. be ~bnormally

high

due to

tissue destruction and acidosis.

Moreover

· such patients require fluid with high sodium concentration such as

lsolyte-P

Acetate ·

HPO .

4

Magnesium

I :

23 mEq

3 mEq

3 mEq

Glucose 5.0 gm, Sodium lactate 0.260 gm, KCI 0.130 gm, MgCI 0.031

gm, Di_basic potassium phosphate 0.026 ~m, Sodium

metabfsulphafe 0.021 gm.

2. Pharmacological Basis : -

lsolyte-P is designed to suit maintenance fluid requiremen_t .of children.

It provides electrolytes, maintains pH, supplies calories and replaces

water deficit. As c'ompared to adults, children need more water and

· s·ame electrolytes. So lsolyte-P provides almost double water but same -electrolytes as lsolyte-M. Roughly lsolyte-P has half concentration of electrolytes compared to lsolyte-M. lsolyte-P can be used in adults when there is chiefly water loss and only small loss· of eiectrolytes (e.g.· hypernatremia)

3. **Indications** :

1. Chiefly used as. maintenance fluid in infants and children to provide daily water and electrolytes.-

4**. Contraindications** :

Hyponatremia: Among all sodium containing l.V. fluids, lsolyte-P has least concentration of sodium (20 mEq· /L)

Renal failure : Cautiously used in renal failure due to high concentration of potassium (20 mEq/L). ' -

Hypovolemic shock : lsolyte-P is not the suitable l.V. fluid to correct hypovolemic shock (as with diarrhoea or vomiting) . due· to following reasons :

(a) Because of low Na concentration, ability of lsolyte-P to correct mtravascular volume and hypotension is poor.

(b) In oliguric child, high K+ conce~tration (20 mEq/L) is not ·safe.

(c) Rapid infusion of large volume of lsolyte-·P can · c·ause hyperglycemia and osmotic ·diuresis even in child with fluid deficit, which is not desirable.

lsolyte-E

(Extracellular replacement solution)

1 ~ · : **Composition** :

One litre of fluid supplies :

Glucose

50 gms

.Sodium

140 mEq.

Potassium

1 O mEq

Chloride

103 mEq

Each 100 ml contains : ·

Acet.ate

Calcium

Magnesium

Citrate

47 mEq

5 mEq

3 mEq

. 8 mEq

Glucose 5.0 gm, NaCl 0.5 gm, Sodium acetate 0.64.0 gm, KCI 0'. 075 gm,

Sodium Citrate 0.075 gm, MgCI 0.031 gm, Sodium metabisulphate

·0.020 gm.

Pharmacological Basis :

lsolyte-E is extracellular replacement solution .. lsolyte-E has electrolytes similar to ECF except that it has double the concentration of potassium and acetate (which will get converted into bicarbonate). Patients on long term fluid therapy may develop magnesium deficiency. lsolyte-E is the only 1.V. fluid available which will correct magnesium deficiency. So lsolyte-E provides all ECF electrolytes, additional potassium and acetate for maximum Capacity to correct metabolic acidosis, supplies energy and replaces water deficit.

contraindicatin

Vomiting or continuous nasogastric aspiration will lead to metabolic alkalosis due to loss of H+ ions in gastric juice. As lsolyte-E provides maximum bicarbonate (acetate 47 mEq/L) among all commercially available l.V. fluids it will significantly aggravate metabolic alkalosis.

Metabolic alkalosis due to diuretics or sodium bicarbonate.

SPECIAL FLUIDS

Sodium Bicarbonate (NaHC03)

. **Composition** :

1. Commonly available and routinely used preparation is Injection sodium bicarbonate 7.5°/o, 25 ml ampoule.

2. Each ampoule contains 22.5 mEq sodium and 22.5 mEq bicarbonate.

2**. Indications** :

1. Treatment of metabolic acidosis.

2. For cardiopulmonary resuscitation and shock.

MiJd to mod~rate metabolic acidosis can be treated with Ringer's lacta1e or lsolyte-E in addition to treatment of underlying disorders. But in severe metabolic acidosis or in critically sick patients, conver$ion of acetate or lactate to bicarbonate may be impaired. So effective way to treat is with sodium bicarbonate.

\

(i) Metabolic acidosis suppresses cardiac contractility. So if it is severe and not treated well, it can lead to hypotension.

(ii) Persistent metabolic acidosis will consume bone buffers and cause osteoporosis and rickets. Release of bone calcium will lead to hypercalciuria and may lead to nephrocalcinosis and nephrolithiasis.

(iii) Acidosis induced reduction in urinary citrate concentration · (which is inhibitor of stone formation) also aggravates the stone formation . .

Amo unt of sodium bicarbonate required depends upon severity *of* acidosis.

Amount'of, · NaHCO~ Required (in mEq/L)

= 0.5 x Weight in Kg x (Desired HC03 - Actual HC03)

Do not correct metabolic acidosis rapidly or completely.

Desired, value of HC03 is usually 10-1'5 mEq/L, and riot normal

· value of 24 mEq/L

(iv) . Never treat acidosis .without treating the .etiology. Common

r etiologies are hypotension, diabetic ketoacidosis, diarrhoea,

.sepsis, hypoxia, postoperative catabolic patients, uraemia etc.

(v) In presence of renal failure, treatment with sodium bicarbonate may cause tetany or pulmonary oedema. So the safer treatment will be dialysis, if acidosis and renal failure are severe.

(vi) Never correct acidosis without correcting associated hypokalemia. By correcting acidosis, NaHC03 will shift ·potassium from . the extracellular compartment into the intracellular compartment. It will aggravate hypokalemia which can be life threatening. This is very important in treatment of diabetic ketoacidosis.

(vii) Do not mix lnj. calcium with lnj. NaHC03 in the same syringe or line: Combination can precipitate calcium carbonate as white crystals.

(viii) Avoid mixing of lnj. NaHC03 with inotropes.

Complications :

Over shoot, post treatment metabolic alkalosis.

Hypokalemia : Due to shift of potassium from ECF to ICF.

Volume overload : 25 ml of 7.5°/o sodium -bicarbonate contains 22.5 mEq sodium. (Normal requirement is 60-100 mEq/day). So aggressive treatment with large volume of sodium bicarbonate will lead to sodium overload. If renal status is normal it will be excreted. But if renal function is poor or if large volume is infus~d rapidly, it can lead to volume overload, pulrnonafY _oedema and hypernatremia. Hypocalcemia - Tetany : Decrease in pH will lead to decreased ionic calcium. I~ patient with chronic renal failure where pre-existing hypocalcaemia is likely, rapid alkalization can lead to tetany. 1.V. calcium gluconate will improve hypocalcemia-induced tetany, with additional advantage of counteracting acidosis induced suppression of cardiac contractilitis

5. Contraindications :

1.' Respiratory alkalosis, metabolic alkalosis and hypokalemia.

2. Correct dehydration, hypokalemia, and hypocalcemia prior to or alorig with sodium bicarbonate treatment.

3. Cautious use in congestive heart failure, chronic renal failure, cirrhosis of liver or hypertension.

Injection Potassium Ghloride

1. Composition :

lnj. Potassium Chloride 15% 10 ml ampoule contains :

1 ml = 150 mg potassium chloride = 2 mEq potassium

·1 Amp. = 1 O ml = 1.5 gm potassium chloride = 20 . mEq potassium

2. Pharmacological Basis :

Potassium is chiefly intracellular cation, but its presence in ECF isvery important . for neuromuscular regulation. .

.Ability of kidney to retain potassium is incomplete (unlike sodium) so

about 20 mEq potassium is lost daily even in presence of hypokalemia .

$0 to avoid hypokalemia, K+ supplementation is required in patient

·on maintenance fluid therapy.

Moreover in many conditions where sodium and potassium both are

lost (Le. diarrhoea, vomiting, diuretic therapy etc.), kidney retains

sodium at the cost of potassium with resultant hypokalemia: Potassium

chloride is added .to various potassium free 1.V. fluids ~s potassium .

·supplement.

indications

potassium in an adult is .about 60 mEq/day. Improper use of injection

potassium chloride can cause hyperkalemia, which can be

dangerous and can cause sudden death due to cardiac arrest.

2. Added to potassium free peritoneal dialysis fluid to maintain proper

potassium level.

3. During cardiac bypass surgery for achieving cardiac stand-still after preparation of heart-lung bypass.

Basic Rules in Use of Injection Potassium Chloride

Never give d~rectl.V. potassium chloride injection.

Always use injection potassium chloride diluted in infusion.

Never add more than 40 mEq potassium/litre

Never infuse more than 1 O mEq potassium/hour. .

Never add potassium chloride in lsolyte-M.

Monitor serum K+ level closely and, if possible, also by ECG monitor during infusion of high dose potassium.

4. Contraindications : .

1. Cautious use in renal failure as hyperkalemia is a potential risk.

2. · Never use injection potassium chloride without kndwing potassium status.

Injection 25o/o-dextrose

Composition :

Available as 25 ml ampoule and 100 ml infusion ·bottle.: . .

100 ml· of 25°/o.;.dextrose contains 25 gm glucose. :.

2. Pharmacological Basis :

Dextrose supplies energy and prevents catabolism. 25°/o-dextrose is a concentrated form, so it is useful when faster replacement of glucose is needed (like in hypoglycemic coma).

When patient is on fluid restriction (i.e. CHF, volume overload, cirrhosis, oliguric renal failure) 25°/o-dextrose provides larger glucose in smaller volume to provide nutrition.

3. Indications :

1. Rapid correction of hypoglycemia or hypoglycemic coma.
2. To provide nutrition to patient on maintanence fluid therapy.
3. For the treatment of hyperkalemia, with 1 O units of regular insulin, 25°/o-dextrose 100 ml is infused to prevent hypoglycemia.

4. Contraindications :

1. 25°/o-dextrose is contraindicated in dehydrated patient with anuria, intracranial or intraspinal haemorrhage and in delerium tremens.
2. To be avoided in diabetic patient unless there is severe hypoglycemia.

5. Caution :

Rapid infusion of 25°/o-dextrose can cause glycosuria secondary to hyperglycemia. So in absence of hypoglycemia, 100 ml of 25°/o-dextrose ~ho.uld be infused slowly over a period of 45-60 minutes.

colloids

·Colloids are large molecules so when infused into the vascular space · they are retain-ed within the vascular system unlike crystalloids. So colloids _: are more effective than crystalloids as plasma volume expander. Colloids are about three times more potent than crystalloid fluids for increasing :·· Vascular volume and supporting the cardiac output. So in patient with ·- <h'aemorrhagic shock, when plasma or blood is not available immediately, _ infusion of ·colloids to correct circulatory fluid volume is vital and often life ~vih,g. However, to maintain adequate capacity to carry oxygen, blood transfusion is ·required subsequently. The potency of colloid fluids as Albumin is a physiological plasma protein. Chief function of albumin is to maintain plasma oncotic pressure (responsible for 75°/o of the oncotic pressure of plasma). Other function of albumin is binding and transport of low molecular substances like bilirubin, hormones, certain drugs etc. Heat-treated preparation of human serum albumin is commercially available in a 5°/o solution (50 gm/L) and a 25°/o solution (250 gm/L). As sodium load is small, 25°/o albumin is also called salt poor albumin.

1. Pharmacological basis :

A 5°/o albumin solution (50 gm/Lor 5 gm/di) has a colloid osmotic

pressure of 20 mm of Hg (which is similar to that of plasma), and expands the plasma volume to roughly the same as volume infused. Approximately half of the infused volume of 5°/o albumin stays in the vascular space. The oncotic effects of albumin last 12 to 18 hrs. The 25°/o albumin solution has a colloid osmotic pressure of 70 mm of Hg and expands the plasma volume by 4 to 5 times the volume infused. Thus infusion of 100 ml of 25°/o albumin can increase the plasma volume 400-500 ml.

2. Indications :

Plasma volume expansion : When rapid volume expansion is required, as in acute hypovof emic shock, burns and severe acute albumin loss, albumin is an *ideal* solution for replacement.

b. Correction of hypoproteinemia : Jn liver disease, diuretic resistant nephrotic syndrome or malnutrition, infusion of 25% albumin is *very* effective for short term management.

c. As an exchange *fluid* : In therapeutic pfasmapheresis, albumin is used as an exchange fluid to replace *removed* plasma .

Adverse effects :

Adverse effects are rare and include nausea, vomiting, febrile reaction and allergic *reaction* including anaphylactic shock.

Precautions and contraindications :

Fast infusion wiff rapidly increase circulatory *volume* with resultant

vascular *overload* and pufmo nary oedema

Infusion of albumin solution is contraindicated in patient with

severe anaemia or cardiac failure. It should be *given* with caution to patients with low cardiac reserve

or cardiac insufficiency.

Dehydrated patients may require additional fluids along with

albumin infusion.

Albumin *solution* should not be used for parenteral nutrition.

The amount of albumin solution administered will depend upon the

clinical condition of the patient and his response to treatment.

For adults an initial infusion of 25 gm of albumin is suggested. (e.g. 5~0

ml of a 5% solution or 100 ml of a 25% solution). A suggested rate. IS

1 to 2 m'- *per* minute {5% albumin) or 1 ml per minute *(25%* afbumrn)

a~though high rates may be needed in the treatment of shock.

Dextran

polymers produced by bacteria (Leucohostoc)

1, Pharmacological basis :

1. Plasma volume expansion :

Both forms effectively expand intravascular volume but dextran is not a substitute for whole blood because it has no oxygen carrying property. It is not a substitute for plasma proteins because it has many limitations including lack of clotting factors. Dextran-40 as 10°/o solution produces greater expansion of plasma volume than dextran 70 as 6°/o solution. But comparative duration of expansion with dextran 40 is shorter due to its rapid renal excretion.

2. Improvement of microcirculation &prevention of thromboembolism : Low molecular dextran improves microcirculation independently of simple volume expansion. It minimizes the sludging of blood that may accompany shock and prevents intravascular aggregation of RBC and improves microcirculation in conditions or procedures associated with impaired circulation.

Indications :

1. Correction of hypovolemia : For short term rapid expansion of plasma volume in conditions such as shock or impending shock from burns, surgery, haemorrhage or trauma.

2. Prophylaxis of deep vein thrombosis and postoperative and post traumatic thromboembolism.

3. To improve blood flow and microcirculation in threatened vascular gangrene.

Side effects:

1. Acute

renal failure : Rapid renal excretion of dextran 40 · t

2· Hypersensitivity reaction : As dextran is a potent antigen,

sensi.tivity . reactions are known to occur, but with better

manufacturing techniques incidences have decreased (

3Dextran may interfere with blood grouping and cross matching

4. Contraindications :

1. Severe oligo-anuria and renal failure.

2. Known hypersensitivity to dextran.

3. Sever~ CHF or circulatory overload.

4. Bleeding disorders such as thrombocytopenia, hypofibrinogenemia

etc.

5. ·. Severe dehydration.

5. Precautions :

1. Dextran should be administered with caution in patient with

a.

lmpair~d renal function or oliguria

b.

Active haemorrhage.

c. Chronic liver disease

d. Patient at risk of developing pulmonary oedema or CHF .

2. The haematocrit should not be allowed to fall below 30.

3. Correct dehydration before or at least during dextran infusion to maintain adequate urine flow arid prevent ARF.

4The anticoagulant effect of heparin is enhanced by dextran.

5. As d~xtran m~y interfere with blood grouping and cross matching,

6 .Along with de>e~raninfusion, patient may requirf3 blood, coagulation

Basic Principles of Fluid Therapy and Pharmacology of I. V · Fluids Administration :

Dextran-40

It is given by l.V. infusion as 10°/o solution in 0.9o/o NaCl or so;

0

glucose. The dosage depends on the pa~ient's ~eed: Adult person with shock usually requires 500 ml of rapid I. V. infusion. In the first 24 hours total dose should not exceed 20 ml/kg. Dextran-40 can be given subsequently in dose of 10 ml/kg/day upto 5 days.

Regimen for thromboembolism

Day-1 : 500-1,000 ml over 4 to 6 hours.

Day-2 : 500 ml over 4 to 6 hours.

Up to 1 o days : 500 ml over 4 to 6 hours on alternate day.

Regimen for surgical prophylaxis :

500 ml of dextran given preoperatively and daily postoperatively

for 3 days.

. Dextran-70

Dextan-70 is *given* l.V. as 6°/o solution. The total dose should not exceed 20 ml/kg *in* the first 24 hrs and 1 O ml/kg on subsequent days.

Gelatin Polymers (Haemaccel}

Haemaccel (500 ml plastic bottle pack 3.5o/o solution).

It *is* a sterile, . pyrogen free, colloidal plasma volume substitute, which contains a polymer of the degraded gelatin with electrolytes.

1. Composition :

Each litre contains : Polymer from degraded gelatin 35 gm

(Molecular weight 30,000-35,000Sodium

145 mEq

·Chloride . 145 mEq

Calcium

Potassium

12.5 mEq

5.1 mEq , .

2.Indications :

· ·· i. · ·For · rapid expansion of intravascuar volume and hypote~sionin shock, burns, trauma and intra or postoperative blood loss.

ii. Prophylactic use in major surgery to reduce total volume of fluid replacement.

iii. For priming of the heart lung machine.

3. Advantage :

It doesnot interfere with coagulation, blood grouping and cross matching.

It remains in blood for 4-5 hours and expands plasma volume by about 50°/o of infused volume.

4. Precautions :

L It contains no preservative, so ensure clear solution before infusion.

ii. Gelatin is plasma expander like dextran so needs almost similar precaution.

iii. It contains calcium, so it should not be mixed with citrated blood as calcium may cause clotting.

5. Side effects :

Hypersensitivity reaction (flushing, urticaria, rigor etc.)

ii. Bronchospasm and fall in blood pressure.

Hetastarch (Hydroxyethyl Starch)

Hetastarch is a synthetic colloid available as 6°/o solution in isotonic saline.

Hetastarch is a: starch that is composed of more than 90% esterified amylopectine. Esterification retards degradation, which leads to longer plasma expansion. 6°/o hetastarch, which is normally used, has molecular

Pharmacological basis :

After LV. hetastarch infusion, molecules with lower molecular weight , (about40°/o . of dose) are readily excreted in urine in 24 hours. Larger ·. Hetastarch does not interfere with blood grouping or crossmatching.

Indications

Hetastarch is less expensive than albumin .

. Plasma volume expansion greater than 5°/o albumin.

Expands plasma volume for a longer period, effect lasts for about 24 hours.

Disadvantage :

.

Increase in serum amylase concentration during and 3-5 days after discontinuation of hetastarch. So serum amylase concentration can not be used to diagnose acute pancreatiti1s during this period.

. Like other colloids, it has no oxygen carrying capacity, so *one* should not allow haemotocrit to fall below 30°/o after an infusion. .

Adverse effects :

Hetastarch is not antigenic. However allergic or sensitive reactions . can occur i.e. vomiting, feverishness, urticaria wheezing etc

Anaphylactic reactions are extremely rare . '

6. Contraindications :

Contraindications are similar to dextran, chiefly bleeding disorders, congestive heart failure or impaired renal function.

7. Administration :

The dose of hetastarch depends on the patient's needs. The usual adult dose of hetastarch 6% solution is 500 ml to 1 litre. The total daily dose should not exceed 20 ml/kg

Pentastarch

Pentastarch is a low molecular weight derivative of hetastarch that is available as 3°/~, 6°/o and 10°/o solution in isotonic saline. Pentastarch differs from hetastarch in having a lower degree of e sterification. As pentastarch contains smaller but more numerous starch molecules, it has a higher colloidal osmotic pressure. So it is more effective as a volume expander than hetastarch. 10°/o pentastarch can increase plasma volume 1.5 times of the infused volume.

Indications, contraindications and side effects are similar to hetastarch.

GUIDELINE FOR FLUID THERAPY IN HYPOVOLEMIA

Clinical evaluation of volume depletion :

Mild (<2 litres in adult)

Thirst

Concentrated urine

Moderate (2-3 litres in adult)

As above, plus :

Dizziness, weakness.

Oliguria (<400 ml/day)

Postural hypotension >20 mni Hg systolic.

Low JVP

Severe (>3 l_itresin adult).

Systolic BP 100mm Hg

Tachycardia (not in elderly), low pulse volume

Cold extremities, poor capillary return.

Reduced skin turgor (doughy feel)

It is usually difficult to estimate volume deficit in a hypovolemic patient. If patient's normal weight prior to fluid deficit is exactly known, loss

of weight will reflect the extent of fluid deficit. Above mentioned clinical criteria can provide only rough idea about severity of fluid deficit.

If the haematocrit (Hct) value prior to fluid deficit is known and there is no blood loss or haemolysis, following formula can be used to calculate extracellular fluid deficit.

ECF deficit (L) = 0.2 x lean body weight x (Current Hct - 1)

Normal Hct

As extent of fluid deficit in a hypovolemic patient cannot be calculated precisely, patient should be closely monitored clinically and by

laboratory data to assess adequacy of fluid repletion.

. Fluid selected for initial volume replacement in patient with hypotension due to hypovolemia is isotonic saline. Saline corrects hypovolemia effectively by replacing lost sodium and water. As simple water or 5%-dextrose infusions are salt free, they are less effective in correction of hypovolemia and can lead to hyponatremia, and are therefore avoided in initial therapy (also see Chapter No. 4). Colloids are preferred when. hypovolemia coexists with hypoalbuminemia (burns, nephrotic ., syndrome, cirrhosis of liver, etc.)

For subsequent fluid therapy, selection of fluid for replac-ement-varies from patient to patient. The type of fluid lost, serum electrolyte status, '.: acid base balance and renal status or coexisting disorders~ all.mustbe". ·,,: taken into account for appropriate selection.

CH. 2 :· Bas.ic ,Principles of Fluid Therapy and Pharmacology of l.V. Fluids

. Immediate aim of fluid therapy in hypovolemia is to get the patient out of danger and to induce positive fluid balance.

Hypovolemic ·patient with hypotension or shock requires . rapid fluid replacement. In patient with shock approximately 1 to 2 litre of fluid should b_e given in first hour to restore adequate tissue perfusion as _quickly as possible, under close medical supervision.

In hypovolemic patient who is haemodynamically stable, gradual repletion is_ preferrable, since it will restore normovolemia while minimizing the risk of volume overload and pulmonary oedema.

The aim of fluid therapy is not just to administer fluids but to induce positive fluid balance. If a patient with diarrhoea losses average 300 ml/hour fluid and I. V. fluid is infused at the same rate, there will not be correction of fluid deficit. On the contrary, as urine output and insensible losses are not considered, inspite of infusion of fluid, fluid deficit will worsen. So fluid therapy can successfully correct hypovolemia only if it provides 50 to 100 ml extra fluid in addition to losses, as summarized below.

Effective rate of fluid replacement per hour

= 50 to 100 ml

+ Urine output per hour

+ Ongoing loss (such as diarrhoea or tube drain) per hour.

Monitoring fluid therapy

flu'id therapy needs careful monitoring with frequent

Patient receiving

Suggest correction of hypovolem1a and adequate

Parameters

fluid replacement are :

.

.

1 Weight

2. -: Skin and tongue

.

3Sensorium : Improvement of anxiety and restlessness_.

4. · Urine output : Urine output >30-50 ml/hour in adu!ts or >0.5 ~o 1.0 ml/kg/hour in · children in absence of glyc~suna o~. osmo~1c diuresis. Increased urine output with decreasing unne spec1f1c gravity and osmolality are other dependable parameters.

5. Pulse rate : ·Correction of tachycardia to pulse rate less than 11 O/min in young adults as well as change from low volume collapsing pulse to bounding pulse.

6. Blood pressure : Patient with shock, hypotension or orthostatic hypoten.sion becomes normotensive.

7. . Haematocrit : Decreasing haematocrit in absence of blood loss or ha~molysis.

8. Blood urea an.d Serum creatinine : Both will become normal. High ratio (>20: 1) of blood urea:serum creatinine will become normal (10:1).

9. Urinary Na : Increase in urinary Na excretion (> 25 mEq/L). If the urinary Na remains under 25 mEq/L the kidney is sensing persistent volume depletion and patient requires additional fluid replacement.

10. Metabolic acidosis : Improvement of acidosis with improved peripheral perfusion.

11. CVP or PAWP: Low central venous pressure (CVP) or pulmonary arterial wedge pressure (PAWP) will become normal with adequate fluid replacement.

METHODS TO CALCULATE RATE OF INFUSION

Methods to calculate rate of fluid infusion

Calculation of the rate of fluid infusion for conventional (routine) l.V. sets, is not mentioned in most of the medical text books. Due to lack of such guidelines, we often feel that the calculation of fluid volume to be infused is a tedious and a complicated exercise. The methods that we are using and have found to be simple, practical and user friendly are mentioned below.

1 Calculation for routine l.V. set

When fluid is infused with routine 1.V. set these methods calculate rate of infusion quickly with reasonable accuracy.

The methods consist of :

1. RULE OF TEN

2. RULE OF FOUR

For routine I. V. set 15 drops = 1 ml. ',.

. Simplest and best method is to follow _' _LJLE OF TEN". -. --- -

RULE OF TEN", by multiplying fluid volume in litres to be infused i~ 24 hours with ten, it will give us drop rate per minute.

I. V. fluid in litre/ 24 hrs. x 1 O = Drop rate/minute

2.0 litre in 24 hours = 2.0 x 10 = 20 drops/minute

3.5 litre in 24 hours = 3.5 x .10 = 35 drops/minute

Q. From drop. rate of infusion, how tq calculate fluid volume in 24 hrs.?

A. As per "RULE OF TEN", roughly the is drop rate divided by 10 will give us volume in ·litre in 24 hrs .

Drop rate per minute ·+ 10 -

15 drops/min = 15/1 O =

.·. 20 drops/min = 20/1 O -

I. V. fluid in litre/ 24 hrs.

1 .5 litre *I* 24 hrs

2 ·litre *I* 24 hrs

As this rough calculation is 96 °/o perfect, at end of 24 hrs. 4% less fluid will be infused.

: Simplest and best method is to follow "RULE OF FOUR"

. As per "RULE OF FOUR"; by dividing ·fluid volume (in ml) to be infused in one hour by four will give us drop ·rate per minute.

Volume in ml *I* hour + 4 -

. Drop rate *I* minute

60 ml *I* hour = 60 + 4

=

15 drops *I* minute

· 200 ml *I* hour = 200 + 4 -

50 drops *I* minute

As per "RULE OF FOUR", drop rate per minute multiplied by four will give fluid volume in one hour.

Drop rate *I* min x 4 -

1 o drops *I* min = 1 o x 4 _

80 drops *I* r:nin = 80 x 4 -

Volume in ml *I* hour

40 ml *I* hr

320 *ml I* hr -

Perfect method to calculate fluid volume from drop rate in 24 hrs.

Drop rate x 96 =volume in ml per 24 hour.

10 drops *I* min = 10 x 96 =

960 ml *I* 24 hrs

20 drops *I* min = 20 x 96 = 1,920 ml *I* 24 hrs

Drop rate calculation for any parameters

Volume to be infused {in ml} = Drop rate *I* minute

Duration of infusion in hours x 4

Example : If 660 ml. fluid is to be infused in 3 hours.

660 = 660 = 55 drops *I* minute

3x4

12

53

Caution : If due to manufacturing defect, routine I. V. set fails to provide 15 -drops per each ml of fluid, these methods (rule of ten and four) will not be accurate in fluid delivery. So always counter check calculated rate of fluid admininstration with actual fluid delivery.

2

Calculation for micro drip l.V. set

For micro drip set 1 ml = 60 drops.

. Number of micro drops per minute = volume in ml/ hour

Micro drop rate *I* minute = Volume in ml *I* hour

35 micro drops *I* minute = 35 ml *I* one hour

50 micro drops *I* minute -

50 ml *I* one hour

Similarly :

Volume in ml *I* hour . -

Drop rate per minute

30 ml *I* one hour . = 30 - micro drops *I* minute ·

45 ml *I* one hour.'-.:'= .. ·45 micro dropsiininute

WATER REGULATION

Water Regulation

In a normal person water in the body is balanced by adjustment in input and urine output.

Response to water deficit

Water intake is regulated by thirst, stimuli for which are :

i)

Dehydration

ii) Fall in BP and

iii) Increased solute concentration (osmolality).

Water excretion is tightly regulated chiefly by antidiuretic

hormone (ADH).

Regulation

Fluid deficit increases osmolality (due to increased serum sodium

or decrease circulating blood volume~ which stimulates thehypothalamusfor ADH release. ADH acts on the kidney at distal tubules and collecting

duct and increases water permeability. The result will be that as much as 18 litres of water is reabsorbed under the influence of ADH. ADH mediates increased water reabsorption and decreases urine output, which preserves body water.

Response to water excess

i. Decrease ADH

.When amount of water in the body increases secretion of ADH

will decrease, so water reabsorption by collecting duct will

decrease and there will be increase in urine volume.

ii. Increase ANP

Volume expansion will also lead to increased secretion of atrio

natriuretic peptide-ANP (due to atrial stretching) which promotesdiuresis and natriuresis. So decreased ADH and increased ANP will decrease water reabsorption and increase urine output and thereby maintain required water status in the body.

SODIUM REGULATION

Sodium Regulation

1 Physiological basis

Sodium is the major ECF cation (sodium value 140 mEq/L ECF vs 25 mEq/L intracellular).

Total body sodium is about 5,000 mEq in a normal adult person.

85-90°/o sodium is extracellular.

Major function of sodium is to mJintain ECF volume and therefore maintain blood pressure.

ECF volume is reflectio~ of total body sodiu m content (amount).

Daily requirement of s odium is about 100 mEq or 6 gm of sodium chloride.

2.*Fluid and Electrolyte Disorders*

·Excess salt is excreted chiefly by kidney. Loss .of ·sodiufl1 · sweat is poor (30-65 m Eq/L).

Response to sodium deficit ·

Deficiency of sodium in bo9y will lead to hypovolemi~ and ~ activates Angiotensin-11 and Aldosterone. .

By acting on the kidney, angiotensin-11 helps to increa '. sodium absorption at the proximal tubules and aldosterone:~ l the collecting duct.

In a state of so di Lim deficit, abso.rption ·ot Na under : aldosterone control is so perfect that almost no urinary Na '. loss occurs. So by almost complete absorption of Na kidney : helps to prevent sodium loss.

Renal priority :

To reclaim sodium under aldosterone influ.ence, initially l potassium and if needed later on H+ is lost in the urine.

. D·ue to body's priority to reclaim Na > H >· K, during abnormal ; loss of all electrolytes (like in diarrhoea) hypokalemia is the I commonest abnormality.

1. When there is excess amount (content) of sodium it will lead ~ . to increased ECF volume, which will lead .to decreased : Angiotensin-11, Aldosterone and increase in ANP.

ii. Decreased angiotensin-11 and aldosterone level will lead to :

. decreased renal reabsorption of sodium.

iii. lnc~eased ANP will lead to natriuresis and diu.resis.

.

Hence net result is increased urinary excretion of sodium. ThU5 \

extra sodium will be lost.

HYPONATREMIA

Hypon.atremia i~ defined as plasma sodium. less than, 135 . mEq/L.

Hyponatremia is not uncommon in ahospitalized patient (incidence 1.5 to 2.5°/o), but is rarely s.een in. an ambL1.lato.ry .patient (if present, r~flects a chronic disease status). :)

Hyponatremia is clinically an important entity because :

Acute, severe hyponatremia has substantially high morb'd.

and mortality.

2. Rapid correction of chronic hyponatremia can lead to neurologi

even death.

3. Etiology and treatment is not as simple as that of other elec1rolYte deficit.

A common understanding is that,

the deficit should be treated with supplementation

e.g. potassium deficit is corrected with K+ supplementation.

But in case of hyponatremia the treatment may be contrary to this common

understanding, thus

Hyponatremia = Sodium deficit

So salt replacement is required in all

is a wrong concept.

Serum sodium reflects the relative proportion of sodium and water.

Hyponatremia usually means water overload and not sodium deficit.

Hyponatremia can occur with normal, low or even high total body sodium.

So basically hyponatremia can be dilutional (water excretion lesser than water intake and so needs fluid restriction as the most important treatment) or due to sodium loss (needs sodium and fluid supplementation)

Pseudo hyponatremia,

A. Normal osmolality

B. High osmolality

Hypoosmolar hyponatremia (true hyponatremia)

A. Hyponatremia with ECF volume depletion

(Patient dehydrated, reduction in total body sodium exceeds reduction in total body water)

1. Extrarenal loss (Urinary sodium < 15 mEq/L)

Vomiting, diarrhoea, peritonitis.

2. Renal loss (Urinary sodium > 20 mEq/L)

Excessive diuretics, S?tlt losing nephropathy, diabetic

ketoacidosis, cerebral salt wasting syndrome.

B. Hyponatremia with hypervolemia, increased ECF volume

(Patient oedematous - Increase in total body water exceeds increase in total body sodium)

Urinary sodium< 20 mEq/L: CHF, cirrhosis and nephrotic syndrome.

Urinary sodium > 20 mEq/L : Renal failure

C **Hyponatremia with normal ECF volume**

(Patient normovolemic, increased total body water)

SIADH, post operative pain, hypothyroidism, glucocorticoid deficiency, psychogenic polydypsia, drug induced.

Clinical Features :

The severity of symptoms depends upon the severity of hyponatremia and the rate at which the plasma sodium concentration is lowered. So acute and severe hyponatremia is symptomatic but chronic and mild hyponatremia is well tolerated. The very young and elderly patients are more symptomatic.

Headache

Muscle cramps

Muscular weakness

Convulsions

Nausea

Vomiting

Confusion

Coma

Death '

Lethargy

Ataxia

. It is not the reduction of ECF Na. that .glves rise to symptoms and signs in hyponatremia. ·1ristea·d, it is the · increa_se in volun:ie. of ICF and particularly increase in the volume of the brain cells which leads to signs and symptoms. · ·

Hyponatrernia Jead_s to hypoosmolality of ECF, so. water moves into .. c~lls (ICF) and the cell swells. ln -.tissue such as skel~tal muscle this is of little consequence. When the cells of the brain swell, .this produces an in,creas~ in intracranial pressure because the brain is an enclosed space Witti ·a fixed volume. Increase in intracranial pressure causes decrease in cerebral blood flow leading to hypoxic brain damage. In severe cerebral oedema, swelling_ of brain exceeds about 5-8°/o o.f. brain volume. As th ~ skull is ·a· fixed closed space, s'uch increa~ed brain cell volume and ·resui'tant significant *r(se* in the intracranial pressure can · ·1ead to"· herniation. Herniation is characterized by unequal or fixed dilated pupils, hypo\ientilation, cardiovascular instability, ·urinary .or faecal incontinence or respiratory arrest.

.

HYPONATREMIA

Oedema

Normovolemia .

No salt

Water restriction

Water restriction

Loop diuretics

Removal of the drugs responsible for hyponatremia :

Diuretics, Chlorpropamide or l.V. Cyclophosphamide.

Management of physical stress, postoperative pain.

Specific treatment for adrenal insufficiency, hypothyro_idis~,
nephrotic syndrome, CHF, uncontrolled diabetes or ketoacrdosrs,
salt losing nephropathy etc.

Thes;e patients require fluid and salt supplementation which can be done with l.V. isotonic (0.9°/o) NaCl (or even oral salt .containing water) at rate appropriate for the ~stimated volume depletion. Intake of simple 'water or I. V. fluid with 1ow, Na (0.45°/o NaCl, lsolyte-M :

5<%'-dextrose, 'etc.·) should be· restricted until the plasma sodium i~ within the normal range, because it will aggravate hyponatr~r:nia , Diuretics induced hyponatremia is treated with saline with potassium supplementation (30 to 40 mEq/L)

. .

2. Hypona'tremia with hypervolemia (O.edematous state)

Therapy is difficult in oedematous state since . sodium· supplementation will worsen fluid overload. Such patients are · treated with diuretics·, salt restriction, fluid restriction (intake< urine output) and correction of potassium· deficit in addition · to ,the etiological treatment.

3. Hyponatremia with euvolemia

Hyponatremia with, nprmal or ti_igt:i ECF volume has impaired water excretion with nornia·I or high tbt~I body sodium. In such patients fluid restri.ction is the 'rl1osf Important .. freatment. Adequate restriction of fluid intake will gradually: increase serum sodium concentration.

c. Basic ·Princip t·es of Correction of Hyponatretnia

Treatment of hyponatremia should balance.· ttl'e. ·risk· of hypotonicity due to hyponatremia and.the'·risk of. therapy.

How rapiolY. hyponatrert:lia should be . corrected is a . dilemma. Patier)t~ with severe hyponat:erni_a (110-115 m,Eq/L) ~re at risk

developing severe and potent1ally_ 1rreversible neurological da~~g and sortietimes even death.

So it is important to balance the risk of hyponatremia against the ·risk of correction. To avoid these problems controlled and limited cofrection is advised after proper selection of patients who need treatment for hyponatremia .

This severe ne~rologicaldisorder is characterized by dysarthria, dysphasia,- flaccid paresis or coma. Diagn"osls is confirmed by C.T. scanning or more acc"urately by MRI.

. Pati.ent with seizures or other severe n.eurological .symptoms due to .hyponatremia needs prompt treatment. , In thi~ etting, risk of untreated hyponatremia and cerebral ed~ma is greater than the potential harm of rapid correction. So rapid correction is ipdicated in acute (<48 hours) symptomatic or severe (serum:· Na <120 mEq/L) hyponatremia

·on the other hand, chronic·(> 48 hrs) and mild hyponatremia,

with minimal neurological sympto:ms are at iittle risk due to hyponatremia. However, these patients can develop demyelination

foll.owing rapid correction. So t~ere is no necessity to correct these patients rapidly and they should be treated using slower acting therapy such as fluid restriction .. Pay particular attention to premenopausal women, elderly and YOl:Jng _chiidren" as they ·are more symptomatic and need early treatme~ · · ·

General guidelines for treatment are :

Chronic asymptomatic hyponatremia :

· The targeted rate of plf!sma sodium .should *not* be greater than '.0.5 to 1 _.*9* mEq/L/hour. ..

the plasma.sodium by es~ tha.n .1 O _to .12 m~q/ L on th.e first day and less than 18 mEq/L over the first two days. As per recent recommendations a 'targeted rate of correction should not exceed 8 mEq/L on any day of treatment. !' '

iiLIf the rate of correction . is faster or rise in serum sodium is > 25 mEq/48 hours or correction is made until normonatremia (serum sodium 140 mEq/L) is achieved there is high risk of central pontine myelinosis.

Acute hyponatremia with severe neurological symptoms :

These patients require · rapid correction of plasma Na with

hypertonic saline. Initial rate of rise of Na concentration should be 1.5-2 mEq/L/hr for the first 3 to 4 hours or until the severe neurological symptoms improve. Besides this initial rapid correction rise in the plasma sodium concentration should not exceed 10-12 mEq in first 24 hours. Patient with seizures also require immediate anticonvulsant drug therapy and adequate ventilation .

A. Regardless of the initial rate of correction, chosen acute treatment should be interrupted once any of the three end points is reached.

1. Patient's symptoms are abolished.

2. A safe plasma sodium (generally 120-125 mEq/L) is achieved

or

3. A total magnitude of correction of 20 mEq/L is achieved. It is necessary to correct hyponatremia accurately to a safer range, rather than correcting completely to normonatremia.

. Conventional method :

Conventional method of correction of hyponatremia is as follows

Na requirement = (Desired Na - Actual Na) x Total body water

This conventional method is complicated because :

It only calculates the amount of Na required to raise Na.

After calculating the requirement of Na, it is confusing to

select Na containing fluid and determine its infusion rate.

Newer method :

This method directly calculates

Change in serum sodium concentration for

given infusate

(select type and volume of Na containing fluid and calculate

expected change in Na).

2. For given target *bf* ch~ng e in Na concentration, this formula can

directly calculate the volume o'f selected Na containing fluid.

Change in serum sodium concentration:=

I nfusate Na/L - Serum Na

Total body water (L) + 1

.or

1nfusate (Na.+ K)/L - Serum Na

Total. body water (L) + t

Total body water

= 0.60 x body weight (kg) in children and non elderly man

= 0~50 x body weight (kg) in non elderly woman and elderly man

= 0.45 x body weight (kg) in elderly woman

For correction of hyponatremia, we need to determine

1. Goal of' correction

: Determine how much Na is to

be

raised and in how much time.

2. select _the ~ppn)priatetype_ of _Na c~ntaining 1.V. fluid for

correction of hyponatremia considering the clinical condition.

Example· mentioned below will provide simple guideline for

calculation.

Example for .correction of hyponatremia :

45 year male (with 60 ~g weight), after appendicectomy received

3 litres of

/o dex~rose/day along with liberal oral intake.

On the third day patient became confused and developed

convulsions

TREATMENT PLAN: Infusion of 3°(o NaCl, l.V. fruse.mide 20 mg and

:water . rete.ntion. For convulsions ph.enytoin ~nd: ,diazepam *may* be

given as per requirement

Calculation of expected change of ~a with pne litre of 3°/o Na91

Change in Na = lnfusate Nall - Serum Na./Total body water (L) + 1

= 513-11 O *I* 0. 6 x 60 + 1

= 403 *I* 36 . + 1 = 403 . *I* 37

= 10.9 mEq/L

INITIAL GOAL: To raise Na .by 4 mEq/L in the initial 4 hours .

To raise 10.9 mEq/L of Na, 1,000 ml of 3°/o NaCl is required. So to raise 4 mEq/L of Na, amount of 3°/o NaCl required is 366 ml (4/ 10.9 x 1,000 = 366 ml)

So total 366 ml of 3°/o hypertonic saline is required to raise serum Na concentration by 4 mEq/L in the initial 4 hours.

Therefore, the required rate of infusion of 3% NaCl is 366/4 = 92 ml/hr

AFTER 4 HOURS : Serum Na was 115 mEq/L with no convulsion f. and mild improvement in sensorium.

Subsequent plan : Determine new goal _to ra!s~ serum Na and i calculate volume and rate of infusion of 3% NaCl for given period as mentioned above (if goal is to raise 4 mEq Na in next 8 hours: rate of infusion of 3% NaCl will be about 47 ml/hour).

POTASSIUM REGULATION

Potassium Regulation

1. Physiological basis

Potassium is a major intracellular catio·n. Total body potassium is about 3,500 mEq. Out of this 98°/o is intracellula~ and just 2o/o is extracellular. The normal serum · potassium concentration is· *3:5* to 5.0 mEq/L vs intracellular concentration of 150 mEq/L. Normal requirement of K+ is 50-80 mEq/day. Potassium plays an important role in cell functions and neuromuscular transmission so it is required for normal function of cells and all muscles.

2. Regulation .

Chief regulation of potassium is through renal excretion:.

Almost all filtered potassium is reabs~rbed. K+ i9n _excreted is the . one secreted at distal tubules . and .collecting duct. . . . . ,

K+ secretion (and therefore excretion) is controlled by aldost · > distal fluid · and sodium ·delivery, serum **H+** ion and ser~rone, rn K' '.·- :·

concentration.

iii. K+ secretion (and therefore excretion) is increased due to h' aldosterone level, incre~sed distal fluid and sodium deliv~~h alkalosis and hyperkalem1a.

iv. Effect of acid base balance : Metabolic acidosis increases seru potassium level while metabolic alkalosis reduces serum K+ leve~

v.

Glucose insulin infusion pushes potassium inside the cell leadin to lowering of serum potassium level.

9

3. How renal regu_lation of potassium and sodium differs ?

Unlike sodium, absorption of K+ is not complete. About 20 mEq K+ is lost everyday even in absence of potassium intake. So hypokalemia frequently occurs in the patient who is on potassium free maintenance fluid therapy.

4. Correlation of serum and body potassium :

It is important to remember that whenever body potassium increases serum potassium rises proportionately and may reach to a dangerous level rapidly. But when there is deficit in total body potassium, reduction in serum potassium is not proportionate because it is partly compensated by shift from intracellular compartment. So hyperkalemia suggests increased total body potassium but level of hypokalemia may under estimate total body potassium deficit.

DISORDERS OF POTASSIUM CONCENTRATION

Disorders of potassium are hypokalemia and hyperkalemia.

HVPOKALEMIA

Hypokalemia is defined as persistent reduction of serum potassium (K+)

below 3.5 mEq/L.

Etiology

Common causes of hypokalemia are summarized in Tab.le No. 3.10 · CH, 3 : Fluid and Electrolyte Disorders

!_able No. 3.1 O : Causes of hypokalemia

1. Poor Intake

Low dietary intake or potassium free l.V. fluids

2. Non Renal Loss

(Urinary

potassium

excretion

< 20 mEq/d

3. Renalloss

Magnesium deficiency, Amphotericin B, Bartter's syndrome

4. **Redistribution** (shift of K+ into cell)

Metabolic alkalosis

Insulin, 82 adrenergic agonist (i.e. salbutamol)

Hypokalemic periodic paralysis

II. **Clinical Features**

.

Clinical picture varies greatly and seldom occurs unless serum

potassium is less than 3 mEq/L. The manifestations of hypokalemia

are mainly neuromus~ular and cardiac.

Fatigue, myalgia and muscular weakness of the lower extremity are common complaints.

Smooth muscle involvement may result in constipation, ileus or urinary retention.

More severe hypokalemia leads to progressive weakness. hyporef.lexia,hypoventilation (due to respiratory muscle involvement) and virtually complete paralysis.

Hypokalemia leads to increased risk of arrhythmia especially in patients on digitalis treatment.

Hypokalemia can lead to polyuria due to nephrog~nic di~betes insipidus. Hypokalemia can cause increased am~ornag~nes1s a~d can precipitate hepatic encephalopathy in patients with hepatic failure.

ECG changes

Do not correlate well with the serum potassium

Early changes :

Flattening or inversi~n. of T waves

Prominent U waves

ST segment depression

Prolonged QT interval

Severe potassium depletion :

Prolonged PR interval

Decreased voltage

Widening of QRS complex

Ventricular arrhythmia

Ill. oragnosis of etiology of hypokalemia

A. History : History of poor K+ intake, abnormal losses due to diarrhoea, vomiting, diuretiG therapy, or transcellular shift of K+ due to insulin, NaHC03 or salbutamol can identify cause

of hypokalem

8. Urinary K+ excretion: In patients with K+ deficit appropriate response is to excrete K+ less than 25 mEq/day. Measurement of urinary K+ excretion is very useful to establish etiological diagnosis ol hypokalemia.

1. Hypokalem1a with low renal K+ excretion (< 25 mEq/day)

It suggests poor intake of K+, diarrhoea, excessive sweat or previous K+ loss due to vomiting or diuretics.

2 High urinary K+. excretion besides K+ deficit.

'd

The ECF volume status, blQod pressljre and associated.aci.

base disorders are useful for arrivi.ng at of etiological diagnosis

.hypokalemia

A. **Therapeutic goals**

1. Prevention of hypokalemia ·

2. To p·revent life threatening complicatfons.

(arrhythmia and respiratory failure)

3. To correctthe potassiumdeficit

4. To minimize on-going losses ·

5. To treat underlying etiology

B. ·Prevention of potassium depletion ·

Normal potassium intake of about 60 mEq/day ·is. sufficient to prevent hypOkalemia. But patients receiving digit81is, !Ong term · diuretics or la'rge doses of steroids should · receive potassium Supplement. Conditions where prevention of hypokalemia is of . special importance are digitalis therapy, hepatic failure, previous myocardial infarction or IHD and diabetes mellitus.

Postoperative patients on parenteral fluid 'therapy shouid receive 40-50 mEq/day of potassium to preverit hypokaleri1ia.

The degree of total body potassium depletion does not correlate with serum potassium. No potassium supplement

Increased oral intake of potassium rich food

Add potassium sparing diuretiqs or decrease dose of diuretics

3 to 3.5 mEq/L :

Treatment in selected high risk patients

(Risk of arrhythmia e.g. CHF, digitalis therapy, history

of acute myocradial infarction or IHD.) ..

< 3 mEq/L :

Needs definitive treatment

E. Precautions before initiating potassium supplements

1. In oliguria-anuria, avoid or supplement K+ caut_iously.

2.Patient's receiving potassium sparing diuretics, ACE

inhibitors and patients with renal failure are at high risk of

developing hyperkalemia, so potassiu m supplementation should

b~ done cautiously.-

3.Digitalis therapy.: In patients on digitalis, potassium enters the

cell at a slower rate so there is a risk of transient hyperkalemia with

faster I. v·. infus.ion. So rate of infusion should be <20 mEq/

4.hour.

Continuous ECG monitoring and frequent ·serum potassium

level estimation is advisab.le. if the rate of infusion is > 20

mEq/hour in any patient

The amount of potassium required to correct potassium deficit cannot

be determined by any fixed formula.

When the average deficit of potassium is about 200-400 mEq,
50-
100 mEq/day of potassium slowly, but adequately corrects deficit.
With severe hypokalemia· or with high rate of ongoing loss, larger ·
dose may be required.
The deficit should be corrected ·slowly over a period of days. It may take weeks to correct severe potassium loss. Failure to increase serum potassium after sufficient dose and duration of potassium supplement raises the possibility of associated magnesium deficiency.

Potassium chloride (KCI) is usually the preparation of choice and will promote correction of hypokalemia as well as of metabolic alkalosis (vomiting and diuretics lead to both hypokalemia and alkalosis).
Potassium bicarbonate and citrate tend to alkalinize the patient and would be more appropriate for hypokalemia associated with chronic diarrhoea or distal RT A.
Oral potassium administration is safer than 1.V. route because
I. V. route carries high risk of hyperkalemia.
H. Oral potassium supplementation

Oral potassium is a safer mode of corr~ction of hypokalemia as there is minimal risk of h~perkalem1a. .

In mild to moderate hypokaler:nia (serum potassium 3 to 3.5 mEq/L) average dose of potassium chloride is 60 to 80 mEq/~ay (20 mEq, 3_ 4 times) along wi.th treatment of underlying disorder (such as vomiting or diarrhoea).

Potassium chloride solution, available in the market contains 20 mEq potassium per 15 ml solution (~gm KCl=13 .. 4 mEq of potassium).

KCI tablets available contain 8 mEq potassium per. tablet.

•

Oral potassium preparation may frequently cause G.I. irritation and therefore the patient is advised to take potassium chloride solution with proper dilution in a glass of water, after food.

•

Oesophageal or small bowel erosion and stricture are uncommon side effects.

l.V. Potassium Therapy

l.V. potassium supplementation carries higher risk of hyperkalemia. So l.V. potassium supplementation should be reserved for severe symptomatic hypokalemia (K+ <3 mEq/L) or for patients who cannot ingest oral potassium.

·No rule is absolute, but common guidelines for l.V. potassium

therapy are as follows :

Always monitor l.V. potassium therapy closely with continuous ECG monitoring and frequent serum potassium estimation.

Avoid l.V. potassium, till urine output is established .

Don't give> 10-20 mEq/hour .

Don't give> 40 mEq/Litre .

Don't give> 240 mEq/day .

Never give inj. KCI directly intravenously, it can cause sudden hyperkalemia and instant death from cardiac arrest.

Never add KCI to lsolyte-M .

Remember that hypokalemia is safer than hyperkalemia, so avoid over enthusiastic treatment.

Rapid I. V. correction can cause dangerous hyperkalemia even in potassium depleted patients.

Tre~t~ent of acidosi.s with l.V. NaHC0

may aggravate or

prec1p1tate hypokalem1a (due to intracellular shift of potassium).

In severe hypokalemia, KCI should be mixed with isotonic .

saline. Don•t use D-5°/o as diluent because dextrose stimulated insulin release will shift pota~sium intracellularly and hence initially aggravates hypoka~emia. Here transient reduction of serum potassium can be 0.2 to 1.4 mEq/L if 20 mEq K+/L of D-5o/o is infused.

•

Diabetic ketoacidosis and nonketOtic hyperosmolar hyperglycemia are amongst the commonest indications .of l.V . . potassium therapy.

Depending upon etiology readymade available l.V. fluid_s can be used to provide potassium supplement e.g. lsolyte-G in vomiting or continuous nasogastric _aspiration, lsolyte-M for parenteral fluid therapy etc.

To make tailor made potassium chloride infusion, 1 oo mEq of potassium. (5 ampoules of 1 O ml, 15°/o KCI ampoules) is mixed in 1 litre of isotonic saline. Infusion of this saline at rate of 100 ml/hour (25 macrodrops or 100 microdrops) will deliver 1 o. mEq KCI per hour.

•

Potassium concentration> 40 mEq/L can cause phlebitis and should be infused only into large (femoral or subclavian) veins.

•

Average rise in potassium level is 0.25 mEq/L, when 20 mEq is given during one hour. Usually 80°/o of the administered dose enters cells.

As soon as cardiac rhythm retu rns to normal or the respiratory muscle strength is restored to normal, l.V. potassium drip is gradually tapered and discontinued and oral KCI is initiated.

.Potassium rich food : Fruit .juices, coconut water, ba~ana, juicy fruits, dry fruits, chocolate, coffee, soup, salt substitutes

e.g. Lona salt.

lnj. Potassium chloride : Most widely available and used is inj. KCI 15°/o, 10 ml ampoule.

10 ml of 15°/o KCI = 1.5 gm KCI = 20 mEq of potassium

1 ml of 15°/o KCI = 2 mEq of potassium

Potassium concentration of commonly used l.V. fluids is summarized in Table No. 3.12.

Table No. 3.12 : Potassium concentration of l.V. fluids

1.V. Fluid
lsolyte-M
lsolyte-P
lsolyte-G

lsolyte-E

Ringer's

Lactate

-

Potassium

35.0

20.0

17.0

10.0

4.0

(mEq/Litre)

HYPERKALEMIA

Introduction

Serum potassium level greater than 5.5 mEq/L is considered hyperkalemia. Hyperkalemia is not as common as hypokalemia and it's clinical manifestations are fewer but more severe than hypokalemia. Mild hyperkalemia (serum potassium 5.5 to 6.5 mEq/L) is of slight concern, but moderate to severe hyperkalemia can be life threatening.

Etiology

Common causes of hyperkalemia are summarized

Clinical Features

Hyperkalemia is often asymptomatic until plasma potassium

concentration is *above* 6.5 to 7.0 mEq/L and may lead to fatal cardiac arrhythmia hence it is called a silent killer.

Vague muscular weakness is usually the first symptom of hyperkalemia. Severe hyperkalemia can lead to hyporeflexia, gradual paralysis affecting initially legs, then trunk and arms, and at last f~ce and respiratory muscles. Paralysis usually spares the muscles supph_ed by cranial nerves and patient remains alert and apprehensive until cardiac arrest and death occurs.

Electrolyte Disorders

1. Increased intake
2. . V. fluid containing potassium

Potassium containing drugs .

Tissue Breakdown

Bleeding into soft issue, G.I. tract or body cavities .

Haemolysis, Rhabdomyolysis

Catabolic state

Shift of potassium out of cell

Tissue damage (ischemia or shock), severe exercise

Metabolic Acidosis

Uncontrolled diabetes due to insulin deficiency

Aldosterone deficiency

Hyperkalemic periodic paralysis, succinyl choline

Impaired Excretion

Acute renal failure or chronic renal failure

Potassium sparing diuretics, ACE inhibitors, NSAID,

heparin, cyclosporine

Reduced tubular excretion : Addison's disease

hyporeninemichypoaldosteronism and amyloidosis

iv.

Effective circulatory volume depletion

Pseudo hyperkalemia

Traumatic haemolysis during blood drawing

Thrombocytosis, marked leucocytosis

105 .

The patient may complain of tingling around lips or in fingers but is more likely to present with slow or irregular pulse rate or collapse due to dangerous bradyarrhythmia.

Diagnosis

Diagn·osis of hyperkalemia depends on clinical susp1c1on, serum potassium measurement and characteristic ECG changes.

Serum potassium greater than 5.5 mEq/L is diagnostic. If all patients with ARF and CRF are excluded the incidence of hyperkalemia will be rather insignificant. If the etiolog~ is not readily apparent and patient is asymptomatic, pseudohyperkalemia should be excluded.

Diagnosis of etiology of hyperkalemia

Clue to other etiological . factors should be sought in the history and . la.boratory analysis. So -to establish etiological diagnosis of hyperkalemia, ex'clude moderate to severe renal failure, c;trugs causing hyperkalemia, pseudohyperkalemia, ECF volume depletion and severe metabolic acidosis as underlying causes . .

The urinary potas_sium excretion rate or transtubular potassium gradient' (TTKG) . are most widely used ·to differentiate renal (hyp~aldosteroni sm) from extra re.nal causes of hyperkalemia.

.

. Renal causes can be further differentiated by administration of mineralocorticoid (0.005 mg fludrocorUsone) :

(a) Increased K+ excretion (urinary K+. excretion > 40 mEq/day _and TTKG > 7) favours diagnosis of aldosterone deficiency (Addison's diseases)..

(b) No increase in K+ excretion is suggestive of aldosterone resistance (pseudo hyperaldosteronism, cyclosporine, K+ sparing diuretics).

. Transtubular potas~ium gradient (TTKG) is a rapid and simple test designed to evaluate the driving force for net K+ secretion. TTKG is useful to establish etiological diagnosis of K+ disorders, as it differentiates renal (abnormal K+ secretion) from non renal causes

of hypo or hyperkalemia.

EMERGENCY TREATMENT

Potentially fatal hyperkalemia (serum potassium> 7.5 mEq/L, profound weakness, absence of P wave, QRS widening or ventricular arrhythmia on ECG) needs urgent treatment. It is based on the following principle :

Cation exchange resin (Keyxalate)

Peritoneal dialysis or haemodialysis

Calcium Gluconate

Calcium gluconate injection is available as 10°/o solution in 1 O ml ampoules.

The usual dose is 10-20 ml infused over 5 to 10 minutes .

It is the most rapid treatment available and the effect begins within minutes but is short lived (30-60 minutes). The dose can be repeated if no change in the ECG is seen after 5-10 minutes.

Calcium administration decreases membrane excitability, and protects the myocardium from toxicity due to potassium.

It should be remembered that calcium does not lower serum potassium level, so definitive treatment should be planned.

Calcium can exacerbate or precipitate digitalis induced arrhythmia .

As a result calcium should be avoided if patient is on digitalis therapy or, if necessary, should be used with great care.

Insulin and Glucose

with insulin to prevent hypoglycemia.

Caution : Hyperglycemic patients should not be given glucose along with insulin.

Administer 25 to 50 grams of glucose together with 10-20 units of regular insulin. Dose of insulin is reduced to 50°/o in patients with severe renal impairment.

Initial bolus of glucose insulin should be followed by continuous infusion of 5°/o-dextrose at 100 ml/hour to prevent late hypoglycemia.

If effective, the plasma potassium concentration will fall by 0.5 to 1.5 mEq/L. This effect begins in 15 minutes, peaks at 60 minutes, and may last for approximately 4 to 6 hours.

Sodium Bicarbonate infusion

Alkali therapy with l.V. NaHC03 will shift potassium into the cei'ls .

Sodium bicarbonate 7.5o/o, 50-100 ml (45-90 mEq) is given as bolus l.V. slowly over 10-20 minutes followed by l.V. NaHC03 drip. Onset of its effect is within 5-1 O minutes and effect lasts for 1-2 hours.

The injudicious use of large amount of alkali can cause excessive calcium binding to albumin and provokes tetany.

Care should be taken to avoid contact between calcium gluconate and sodium bicarbonate in the needle, syringe or infusion set, as

it will precipitate into chalky deposits.

l.V. sodium bicarbonate is most likely to be useful in severe hyperkalemia with metabolic acidosis.

Patients with CRF-ESRD seldom respond to this therapy and may not tolerate the sodium load and the resultant volume expansion.

Beta adrenergic agonists

B agonists such as salbutamol promote cellular uptake of p~tassium and effecitvely lower serum potassium level.

Salbutamol is given in a nebulized form or parenterally. Dose recommended is 20 mg in 4 ml of saline by nasal inhalation over 1 O minutes, or 0.5 mg by 1.V. infusion.

• · Insulin and B agonists exert additive effect. 1.V. salbutamol is preferred in E§Ro requiring a rapid lowering of serum potassium. However nebulization is preferred in ESHD patients associated with CAD, because heart rate is less elevated with nebulization than with l.V. therapy.

Calcium, . glucose-insulin, sodium bicarbonate and 82 .. agonists are temporary measures. It does not remove excess potassium from the body. Measures to remove potassium are diuretics, c~tion exchange resins and dialysis .

. 5 . . Loop and thiazides diuretics often in combination may. enhance

potassium excretion, if renal function is adequate.

6. Cation exchange r~sins

Cation exchange resins such as sodium polystyrene sulphonate (Keyxalate) promote the exchange of sodiuni for potassium in G.I. tract. Each gram binds 1 hlEq of potassium and releases 2-3 mEq of sodium.

• When given orally the usual dose is 25-30 grams mixed with 100 ml *20°10* sorbitol 3-4 times daily (sorbitol prevents constipation). It can also be given as retention enema consisting of 50 grams Of resins and 50 ml of *70°10* sorbitol mixed in 150 ml of water every 4-6 hrly. It is retained for about 60 minutes after which rectum is washed with clear water. The rectal route is faster and more reliable.

Each enema can generally lower the plasma potassium concentration by 0.5 to 1 mEq/L within 1-2 hours and effect will last for 4-6 hours.

Adve~se ~ffects of resins include anorexia, nausea, vofniting and const1pat1on. As sodium is released in exchange of potassium an~ absor?ed by G.I. tract, resins should be used cautiously in patients with CHF or volume overload.

7. **Dialysis**

The most rapid and effective way of lowering the plasma potassium concentration is haemodialysis (removal rate 35 mEq/hour). Dialysis should be reserved for patients with renal failure and those

with severe life threatening hyperkalemia unresponsive to more conservative measures.

Peritoneal dialysis also removes potassium but is only · 15 to 20°/o as effective as haemodialysis. . . · ·

Monitoring

Repeated ECG or serum potass.ium determination shoufd be ~sed to check the effectiveness of therapy.

NON EMERGENCY TREATMENT

In mild to moderate hyperkalemia. and for prevention of recurrence of severe hyperkalemia following measures are useful : ·

. .

Dietary potassium restriction : Avoid fruitjuice, coconut water and food rich in potassium.

. Avoid medications like :

Potassium sparing diuretics, NSAID and ACE inhibitors (_all reduce ·renal potassium excretion).

B-blockers (decrease ECF to ICF shift of potassium) ..

Loop or thiazides diuretics to increase renal excretion of potassium.

Cation exchange resins : Dose required is 15-20 gram Keyxa~ate, 2-4 times/day. .

Glucocorticoid (hydrocortisone)

therapy.

Treatment of diabetic ketoacidosis.

Correct metabolic acidosis : Metabolic acidosis is generally associated with hyperkalemia. So treat with sodium bicarbonate (600 mg tablets, 2-3 times/day) or sodium citrate (Shohl's solution, 10-15 ml three times daily).

CALCIUM AND MAGNESIUM REGULATION

CALCIUM

Calcium is essential for bone formation and neuromuscular function. If calcium intake is truly inadequate, bone mineralization may be impaired in children and bone loss will be accelerated in adults.

Distribution : The body of a normal adult contains 1 .2 to 1 .4 kg of calcium, so it is the most abundant cation in the body. Out of this about 99% is present in the bone, 1°/o in the cells of soft tissue and 0.15% in ECF.
Serum Calcium : Normal value is 10 ± 0.5 mg/di. About 40o/o of this calcium is bound to albumin (protein) and 50-55% is in ionized form. The remaining calcium is complexed with the anions of organic acids such as phosphate, bicarbonate, citrate, lactate or sulphate.
• Ionized calcium : The ionized-free form is the physiologically active form of calcium and measures 4.8 mg/di. The total serum calcium does

not always reflect the level of ionized calcium. Hypoproteinemia leads to reduction in protein bound and total serum calcium but the ionized calcium remains unchanged. In such cases hypocalcemia may be wrongly diagnosed.

Correction of total serum calcium in hypoalbuminemia : For corrections add 1 mg/di to serum calcium for each 1 gm/di reduction in serum albumin below 4 gm/di.

Regulation : Parathyroid hormone (PTH) and vitamin D (1,25(0H)2D3) are the main factors that maintain normal serum ionized calcium.

Fine control of serum calcium is achieved by their action on bone, intestine and kidney.

Role of PTH : PTH increases serum calcium by stimulating bone reabsorption, increasing renal calcium reabsorption and promoting conversion of vitamin D to its active form (1,25(0H)20 3). Serum calcium CH. 3 : Fluid and Electrolyte Disorders

regulates PTH secretion by a negative feedback mechanism. _ Hypocalcemia stimulates and hypercalcemia suppresses PTH release.

Role of vitamin D : Active form of vitamin D - Calcitriol (1,25(0H) 2 D) increases serum calcium by promoting intestinal calcium absorption and plays a major role in bone marrow formation and reabsorption. Calcitriol is a potent suppressor of PTH. Synthesis of calcitriol is stimulated by both PTH and hypophosphatemia, and is inhibited by increased serum phosphate.

Hypercalcemia

Hypercalcemia can have variable presentation. It can present with serious illness such as malignancy or may be diagnosed accidentally by laboratory testing in asymptomatic patient in primary hyperparathyroidism.

Etiology

Primary hyperparathyroidism and malignancy are the two most common causes responsible for hypercalcemia in more than 90°/o of the patients. Common causes of hypercalcemia as per their basic pathophysiologi~al mechanism are summarized in Table No. 3.16.

Clinical Features

Variable clinical features can be

1. Secondary to underlying disorders.

Secondary to hypercalcemia.

Clinical features of hypercalcemia are related to severity and rapidity of onset. Mild hypercalcemia is generally asymptomatic. Features of severe hypercalcemia are :

CNS Symptoms : Weakness, fati_gue, depression, confusion, stupor or coma.

. GI Symptoms : Constipation, anorexia, n~u.sea and vo~iting. Abdominal pain may result from hypercalcem1a induced peptic ulcer disease or pancreatitis.

Renal Symptoms : Polyuria, nocturia and stone formation. 114

Cardiac abnormalities ·: .Patients with· hypercalcemia a.re more prone· to digoxin _ toxicity. ECG shows shortened QT interval.

Increased. bone turnover

(1) Primary hyperparathyroidism

(2) Sec~ndary hyperparathyroidism

(3) Malignancy (lung, breast, ·kidney,. multiple myeloma)

(4) Thyrotoxicosis

(5) Lithium therapy

II

Increased intestinal absorption

(1) Vitamin D intoxication

(2) Milk Alkali. syndrome .

(3) Granulomatous disease (i.e. sarcoidosis)

Ill Decreased renal excretion

(1) Familial hypocalciuric hypercalcemia

(2) · Thiazide diuretics

(3) Acute adrenal insufficiency

Diagnosis

It is important to diagnose the two most common disorders, primary hyperparathyroidism and malignancy (which accounts for more than 90°/o of total hypercalcemia), from other less common causes of hypercalce~i a.

I

History and physical examination .

(a) Patient with primary hyperparathyroidism is usually a~ymptomatic. Hypertension is common in primary hyperparathyroidism .

.

(b) . If hYpercalcem.ia is present fcir more than 6 months without obvious cause, primary hyperparathyroidism is most certain.

(c)

(e)

In . m~lignancy,. symptoms of mal.ignancy bring the patient to the physician and hypercalcemia is diagnosed by laboratory investigations.

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Hypercalcemia With renal stone favours long duration, so malignancy is unlikely. ·

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Use of vitamin. D, calcium, antacid and lithium therapy may be underlying cause of hypercalcemia. CH. 3 : Fluid and Electrolyte Disorders

1.1 s

(f) A chest X-ray should be obtained to rule out pulmonary malignancies and granulomatous disorders.

11

Investigation

Useful laboratory tests are electrolytes, BUN, serum creatinine, phosphate, serum protein electrophoresis, intact PTH level etc.

(a) The presence of high serum chloride and low serum HCO in a ratio >33 to 1 is suggestive of primary hyperparathyroidis~.

(b) A low serum chloride, high serum HCO and elevated BUN and creatinine are characteristic of milk alkaIT syndrome.

(c) In patients with high total protein with reversed AG ratio, suspect multiple myeloma. Serum protein electrophoresis showing a monoclonal spike is suggestive of multiple myeloma.

(d) If increased intact PTH, most common cause is primary hyperparathyroidism. A low plasma phosphate is found in primary hyperparathyroidism.

As a general rule, primary hyperparathyroidism is the etiology in outpatients who are asymptomatic with a serum calcium concentration lower than or at 11 .0 mg/di. On the other hand, malignancy is often the cause in symptomatic patients with an abrupt onset of diseases and serum calcium concentration higher than or at 14 mg/di.

Treatment

Volume restoration, expansion and saline diuresis are the most useful and effective measures to correct hypercalcemia.

o.9°/

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NaCl is infused to correct dehydration, for volum~ expansion and natriuresis, which leads to. urinary excretion of calcium. patient may need 4-6 litre of saline to achieve this goal. So use cautiously in elderly and heart failure patients to avoid pulmonary oedema.

Frusemide : After volume expansion forced diuresis can increase urinary calCium excretion. Avoid de.hydration, hypokalemia and hypomagnesemia during treatment with frusemide. Avoid thiazide diuretics because it impairs urinary calcium excretion.

Haemodialysis : Reserved for treatment of patients with severe hypercalcemia and little or no renal function. ·· ~

B Measures to inhibit bone resorption

(1) Bisphosphonates : Pamidronate is the potent and most widely used bisphophonate in treatment of hypercalcemia due to bone reabsorption. For severe hypercalcemia pamidronate 90 mg 1.V. over 4 hours causes a fall in calcium, which is maximal at 2-3 days, and the effect lasts for a few weeks

(2) Plicamycin (mithramycin) : It acts by inhibiting bone resorption. Currently it is used sparingly due to its high toxicity and because of better effectiveness of bisphosphonates. Mithramycin should be avoided in patients with severe hepatic, renal and marrow disorders. Its effect begins in 12 hours and peaks at 48 hours. The dose can be repeated at 3 to 7 days intervals.

(3) Calcitonin : It inhibits bone resorption and increases urinary calcium excretion. Because of its rapid action it is useful for urgent therapy of life threatening hypercalcemia along with rehydration

and saline diuresis, but it is not useful for long term therapy.

(4) Gallium Nitrate : It inhibits bone resorption and corrects hypercalcemia. It is not often used due to 5 days duration of infusion, potential for nephrotoxicity and availability of safer and effective agents.

C Measures to decrease intestinal absorption

(1) Glucocorticoids : It decreases intestinal absorption and increases urinary excretion of calcium in pharmacological doses.

Alternatively ketoconazole and hydroxyl chloroquine can be used.
Glucocorticoids are effective in hypercalcemia due to vitamin D intoxication, sarcoidosis and malignancies (multiple myeloma, leukaemia, Hodgkin's disease etc.), but do not alter calcium level in primary hyperparathyroidism or in a normal person.
(2) Oral phosphate : It inhibits calcium absorption and promotes calcium deposition in bone and soft tissue. It should be used only if the serum phosphorus level is less than 3 mg/di and renal function is normal. Summary of treatment options tor hypercalcemia is shown in Table No. 3.17 CH. 3 :fluid and Electrolyte Disorders

D Specific treatment

(1) Discontinue drugs responsible.
(2) Surgical treatment of primary hyperparathyroidism.
(3) Specific treatment for malignancy, thyrotoxicosis etc.

Hypocalcemia

1: Etiology

Hypoalbuminemia is the common cause of hypocalcemia with normal ionized calcium. True hypocalcemia is the result of decreased calcium absorption from the gastrointestinal tract or decreased calcium reabsorption from bone, due to abnormalities of either PTH or vitamin D (calcitriol). The most common causes of hypocalcemia are shown

in Table No. 3.18.

2. Clinical Features

Symptoms of hypocalcemia vary with degree and rate of onset and are due to increased neuromuscular excitability. The patient may complain of weakness, circumoral and distal extremity parasthesia, muscle spasm, carpopedal spasm, tetany and mental changes such as irritability, depression and psychosis. On physical examination patient may have increased deep tendon reflexes or signs of latent tetany (Chvostek

sign, Trousseau•s sign).

Chvosteksign is a facial twitch $1icited by tapping on the facial nerve just anterior to earlobe, just below the zygomatic arch with the mouth slightly open.

Trousseau's sign is the development of wrist flexion metacarpophalangeal joint flexion, and hyper extended fingers' and thumb flexion when a BP cuff is inflated above systolic pressure for 3 minutes.

The ECG may show prolonged QT interval. Digitalis effect may be reduced.

Severe hypocalcemia may cause lethargy, confusion, laryngeal spasm, seizures or reversible heart failure.

Chronic hypocalcemia due to hypoparathyroidism may cause

cataracts and calcification of basal ganglia.

Hypoalbuminemia

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Hypoparathyrodisim

(a) Post surgical - Hungry bone syndrome

(b) Hypomagnesemia

(c) Idiopathic

Defect in vitamin D metabolism

(a) Nutritional, lack of exposure to sunlight

(b) Malabsorption and drugs (e.g. anticonvulsants)

(c) Liver disease, renal failure

(d) Vitamin D dependent rickets

Miscellaneous

(a) Metabolic or respiratory alkalosis

(b) Sepsis, toxic shock syndrome

(c) Acute pancreatitis, burns

(d) Massive transfusion of citrated blood

Severe acute hyperphosphatemia

Tumorlysis syndrome, ARF, rhabdomyolysis

Diagnosis

Detailed history and physical examination may give clue for underlying etiology of hypocalcemia. Hypoalbuminemia is a common cause of decreased total serum calcium. However the common causes of true hypocalcemia are magnesium deficiency, re.nal failure, metabolic alkalosis and complications of parathyroid surgery. Important investigations required for etiological diagnosis of hypocalcemia are serum albumin, serum HC03 , serum CH.

Acute Management

Symptomatic hypocalcemia should be treated as emergency with 10°/o calcium gluconate (90 mg elemental calcium/1 O ml) 10-20 ml l.V. slowly over 1 O minutes. Severe symptomatic hypocalcemia may require infusion of 60 ml of calcium gluconate in 500 ml of 5°/o dextrose. Calcium concentration of the drip is 1 mg/ml and its requirement is 0.5 mg to 2 mg/kg/hour.

If 1.V. calcium does not relieve tetany, rule out (and correct) hypomagnesemia. In treatment of metabolic acidosis with hypocalcemia (e.g. CRF), correct hypocalcemia before correction of acidosis.

When citrated blood is transfused rapidly, hypocalcemia can occur. So for every 4 units of blood, give 10 ml of 10°/o calcium gluconate.

Long term management

1. Treatment of underlying etiology.
2. Calcium supplementation : An asymptomatic hypocalcemic patient

needs oral elemental calcium 1 to 3 gram per day. Calcium is best absorbed when taken between the meals.

3 Vitamin D supplementation

Calcitriol (1,25 (OH) D) is the most potent of the vitamin D preparations and has the fastest ~n~et and the shortest duration of action. So there is no risk of vitamin D intoxication but disadvantage is the higher cost. 120 · CH .. 3 . . Fluid and Electrolyte Disorders

Vitamin D (ergocalciferol) requires several weeks to achieve full effect. Although cost is low, because of long half-life and storage in fat, it carries higher risk of vitamin D intoxication.

PHOSPHORUS

Ph6s.phorus is critical for bone formation and cellular energy metabolism. Phosphorus is chiefly intracellular and only 1°/o is in the ECF. The normal serum phosphate level is 3.0-4.5 mg/di. It is best measured in the fasting state, since there is diurnal variation (lower value in the morning and higher at night and post meals). Major regulatory factors includes PTH, 1,25(0H)2D3 and insulin. PTH lowers serum phosphorus by increasing renal excretion. 1,25(0H)2D3 increases serum phosphorus by enhancing intestinal phosphate absorption. Insulin lowers serum levels by shifting phosphate into cells.

Hypophosphatemia

Etiology ·

Important causes of hypOphosphatemia are summarized in Table No. 3.20

Clinical Features

Acute Hypophosphatemia can cause :

(a) Muscular abnormalities : Proximal muscle weakness,

rhabdomyolysis, impaired diaphragmatic function, respiratory

failure and congestive heart failure.

(b) Neurological abnormalities : Parasthesia, dysarthria, confusion,

seizures or coma.

(c) Haematological abnormalities :

i. Enhanced oxygen dissociation causes tissue hypoxia, and

haemolysis.

ii.

Impaired phagocytosis and opsonization leading to increased

susceptibility to bacterial and fungal infections.

Chronic hypophosphatemia causes mineralization defect leading

to rickets in children ahd osteomalacia in adults. CH. 3 : Fluid and Electrolyte Disorders 121

Table No. 3.20 : Causes of hypophosphatemia

A Decre~se~ intestinal phosphate absorption

(1) V1tamm D deficiency

(2) Vitamin D dependant rickets type I and II

(3) Malabsorption, Phosphate binding antacids

B

Increased renal phosphate excretion.

(1) Hyperparathyroidism

(2) Vitamin D - deficiency

(3) X linked hypophosphatemic rickets

C

Shift of phosphate into intracellular fluid .

(1) Respiratory alkalosis

(2) Dextrose infusion

(3) Diabetic ketoacidosis

(4) Sepsis

(5) Hungry bone syndrome

Treatment

(1) Mild hypophosphatemia needs only treatment of underlying etiology.

(2) If serum phosphate level is > 1 .0 mg/di and patient is asymptomatic, oral phosphorus replacement is sufficient. Milk and milk product is an excellent source of phosphorus. Milk contains 1 gram of inorganic phosphorus per litre.

To correct hypophosphatemia, solutions containing sodium phosphate, potassium phosphate and neutral sodium phosphate are used.

Depending upon severity, upto 3 grams can be given in four to six divided doses in 24 hours.

(3) Severe (<0.5 to 1.0 mg/di) symptomatic hypophosphatemia may require

f.V. phosphate therapy. 1.V. infusion should be stopped when serum phosphorus level is greater than 1.5 mg/di or when oral therapy is possible. Extreme care must taken to avoid hyperphosphatemia, which may cause hypocalcemia, ectopic soft tissue calcification, hypotension and death. Hypophosphatemic patients . are frequently hypokalemic and hypomagnesemic and need correction.

MAGNESIUM

Magnesium is the fourth most common cation of the body (after Na, K and Ca), second most common intracellular cation (after K) and is the commonest intracellular divalent cation. About 60°/o of body magnesium is in bones, only 1°/o in ECF and rest is within the cells. Since clinical effects of magnesium disorders are determined primarily by tissue magnesium content, serum magnesium levels have limited diagnostic value. The normal serum magnesium level is 1.4 to 2.2 mEq/L (1.8 to 3.0 mg/di)

Magnesium plays an important role in neuromuscular function and maintenance of cardiovascular tone.

Hypermagnesemia

Hypermagnesemia is defined as serum magnesium concentration above 2.5 mEq/L (3.0 mg/di). As a normal kidney c~n.effectivel~ e~crete magn~siu~

load, hypermagnesemia is rarely seen in clinical practice 1f renal function normal.

Etiology

Renal failure patients, receiving magnesium containing antacids,

laxative or 1.V. ·fluids is the most common cause. · 124

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· Treatment of pre-eclampsia with 1.V. magnesium sulphate.

ARF with acute rhabdomyolysis.

· Diabetic ketoacidosis without treatment.

Clinical features

Neuromuscular manifestations: It includes muscular weakness; lethargy,

loss of deep tendon jerks, muscular paresis leading to respiratory

depression and respiratory failure.

Cardiac manifestations : It includes hypotension due to peripheral

vasodilatation, bradyarrhythmia and, in severe cases, cardiac asystole.

ECG changes : Due to generalized depression of cardiac conduction.

ECG changes are prolonged PR intervals, increased QRS ·duration

and QT interval and lastly complete heart block.

Hypocalcemia may occur due to hypermagnesemia induced decreased

secretion of PTH and end organ resistance of PTH.

Treatment

1. Eliminate source : Stop magnesium containing antacids, laxative and discontinue magnesium containing l.V. fluids.

2. 10°/o calcium gluconate, 10-20 ml l.V. slowly over 10 minutes will effectively correct hypotension by lowering magnesium levels.

3. If renal function is normal I nj. Frusemide after rehydration with isotonic saline will enhance renal excretion.

4.

Haemodialysis can effectively correct hypermagnesemia and is the treatment of choice in patients with renal failure.

5. Supportive treatment : Artificial respiration may be necessary if respiratory failure occurs

Hypomagnesemia

Hypomagnesemia is defined as serum magnesium concentration less t~~n 1.5 mEq/L (1.8 mg/di). However in symptomatic hypomagnesemia: 1 .t 15 usually less than 1 mEq/L. Magnesium deficiency is a common clinical problem, seen in about 10°/o of patients admitted in city hospitals and up to 65% patients in ICU.

Etiology:

Common causes of hypomagnesemia are alcoholism loop diuretics diarrhoea and poor nutrition. Causes of hypomagnesemia are ~ummarized in' Table No.

1 · Loop diuretics, osmotic diuretics

2. Hypercalcemia

3.Acute Pancreatitis

Increased gastro-intestinal losses .

Chronic diarrhoea due to malabsorption .

Vomiting or nasogastric aspiration

Poor intake

Prolonged malnutrition or magnesium free l.V. fluids

Chronic alcoholism and alcohol withdrawal

Others

1. Primary aldosteronism

2. Hypoparathyroidism

3. Drugs : Aminoglycosides, Cisplatin, Amphotericin B,

Cyclosporine A etc.

Clinical features

Hypomagnesemia rarely occurs as a single deficiency. It often causes hypocalcemia and hypokalemia, which contributes to the clinical picture.

Neuromuscular manifestations

They are similar to hypocalcemia and include lethargy, confusion, tremor, fasciculations, ataxia, tetany and seizures.

Cardiovascular manifestations

ECG abnormalities include prolonged PR and QT intervals. Atrial and ventricular arrhythmia may occur, especially in patients treated with

digoxin. Digitalis toxicity may be precipitated or aggravated by hypomagnesemia. 126

Metabolic abnormalities

Hypocalcemia : Mg deficiency results in decrease in PTH secretion and end organ resistance to PTH leading to hypocalcemia. If patient With hypocalcemia does not respond to calcium or vitamin-D replacement, think of hypomagnesemia and correct it.

Hypokalemia : Mg deficiency enhances renal excretion of potassium. Hypomagnesemia and hypokalemia often coexist. So when hypokalemia does not respond to K+ replacement, rule out associated Mg deficiency and correct it.

Treatment

Correct underlying etiology and-coexisting hypocalcemia and hypokalemia.

(2) Magnesium sulphate ($MgSO_4$)

$MgSO_4$ is available as powder as well as 10% or 50% solution. 5 ml of 10°/o or 1 ml of 50o/o $MgSO_4$ contains 4 mEq magnesium. The 50% solution must be diluted before use.

Conversion equations for Mg therapy are

1 gram $MgSO_4$ = 8.1 mEq of magnesium

1 mEq of Mg is provided by 123 mg of $MgSO_4$

1 mmol = 2 mEq = 24 mg elemental magnesium

Onset of action : l.V. immediate, l.M.<1 hour

Peak effect: l.V. few minutes, l.M. 1-3 hours

Duration of action: l.V. 30 min, l.M. 3-4 hours

Treatment of mild hypomagnesemia

Mild deficiency (serum Mg concentration 1.5 mEq/L or 1.8 mg/di) needs oral supplementation of 2 gram three times a day without producing diarrhoea. For dietary supplementation, Mg rich diet includes green vegetables, nuts and legumes, chocolate and fruits such as bananas, grapes and oranges.

(4) Treatment of severe Mg deficiency

Patients with severe hypomagnesemia (serum Mg concentration less than 1.0 mEq/L or 1.2 mg/di) need parenteral Mg supplementation.

FLUID THERAPY IN HYPOVOLEMIC SHOCK . ....

Fluid loss leading to hypovolemia, hypotension and shock are common clinical problems. Amount of fluid to be given is decided by clinical and other guidelines

1.

Poor expansion of intravascular volume : 1,000 ml of D-5°/o will

increase 330 ml of ECF volume and only 83 ml (1 /4 of ECF) stays in the intravascular space. As 1 litre of D-So/o will increase intravascular volume only by 83 ml, rise in blood pressure would not occur satisfactorily in the patient with shock.

2.

Increased urine output due to osmotic diuresis: Larger and faster infusion of D-5°/o will cause glucose load(> 25 gm/hour) and osmotic diuresis will occur. So even in presence of hypovolemia, there will be increased urine output, which delays correction of dehydration. Increased urine output will also create false impression that now there is no fluid deficit. In such a setting rate of fluid replacement may be slowed down despite hypovolemia, therefore hypotension may not improve. So 5°/o-dextrose should be avoided in the treatment of shock.

Fluid Therapy in Medical Disorders

The other reason to prefer isotonic saline as initial therapy is its safety in unknown glycemic status. Moreover isotonic saline is least expensive, readily available, and reaction free (as compared to colloids), so preferred for the initial treatment of shock.

Rate of infusion is decided on the clinical status and vital data. Depending upon the nature of loss, NaHC03 and KCI is added to the infusion.

1. Ringer's lactate is the most physiological fluid. Its composition is

almost identical to ECF, so large volume can be infused without fear of electrolyte imbalance.

2.

It corrects acidosis (lactate is converted into HC03) produced by lactic acid due to anaerobic metabolism during hypotension.

Q. Why Ringer's lactate is avoided in initial treatment of shock ?

A. Ringer's .lactate is not an ideal fluid for initial fluid therapy because.

1. Potassium in Ringer's lactate is unsafe till renal status is uncertain.

2.

In. shock state hepatic conversion of lactate to bicarbonate is unpredictable.

So use of RL for initial .treatment of shock may cause hyperkalemia or lactic acidosis and hence avoided. NaHC03 is preferred agent for initial treatment of acidosis with shock.

.1. More effective plasma volume expansion, since it remains in the vascular space (in contrast to saline, where 2/3 of it enters the interstitium).

2. Lesser risk of pulmonary oedema, since the increase in plasma oncotic pressure favours fluid movements out of the interstitium into the vascular space.

The primary indication of the use of albumin or other colloids is in hypovolemia with hypotension in protein losing states such as burns.

Although these solutions are also used in treatment of shock or severe hypovolemia, they appear to offer little or no advantage over the pure electrolyte solutions. Isotonic saline is equally effective in producing volume repletion, although volume required is 2.5 to 3 times greater than that of- colloids, because of its extravascular. distribution. Replacement of such large volume by colloids is not harmful. On the contrary it replaces the interstitial fluid deficit that is induced both by fluid loss and by fluid movement into the cells.

FLUID THERAPY IN GASTROINTESTINAL DISORDERS .

Pathophysiology of fluid, electrolytes and acid base disturbances and fluid therapy of the following important G.I. disorders are included here~

1. Enteric disorders :

Vomiting or nasogastric aspiration, diarrhoea and haemetemesis

2. Hepatic disorders :

Ascites in cirrhosis of liver and hepatic encephalopathy

3. Pancreatic disorders :

Acute pancreatitis CH : 4 : -Fluid Therapy in Medical Disorders . 135

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Fluid Therapy in Vomiting

Upper gastrointestinal losses (vomiting o'r nasogastric aspiration) is a commonly encountered. problem. For proper. selection of fluid, i,t is necessary to understand the basic physiology.

1. Hypovolemia : Due to loss of fluid there is dehydration-:

decreased ECF volume.

2.

tlypokalemia : Decreased ECF volume and loss of sodium in gastric juice will lead to increased secretion of aldosterone. Under aldosterone influence, there will be increase in reabsorption of sodium and increase in secretion of potassium leading to greater loss of potassium in urine (Table No. 4.3). So hypokalemia occurs partly due to loss of potassium in vomiting but chiefly due to its loss in urine (renal loss).

3.

Corrects Hypochloremia, Alkalosis and Dehydration

A. Besides the clinical criteria and urine output monitoring, urinary pH i~ very important to assess efficacy of fluid therapy. Acidic urine suggests need for more vigorous treatment and alkaline urine suggests response to therapy.

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.A. Isotonic normal saline is ideal for gastric irrigation . (especially in electrolyte disturbances, hypovolemia and haemodynamically unstable patients). Remember never to use plain water for irrigation. Plain water draws more gastric secretions iri to the stoma.ch in attempt to make the fluid isotonic for absorption. Removal of this electrolytes containing fluid can deplete fluid and electrolytes. ·

For the same reason, in patients with contiriious hasogastric

aspiration, it is important to restrict the amount of ice chips given by mouth. Nasogastric suctioning of ice chips : can cause fluid and electrolytes loss from the stomach. CH. 4

Fluid and electrolyte abnormality in diarrhoea is summarized below :

1. Hypovolemia : Loss of fluid in diarrhoea occurs due to

a. Abnorma.lly increased secretion of fluid into the small bowel (secretory diarrhoea due to G. I. infections e.g. E. coli, Vibrio cholerae or rota virus).

b.

Decreased absorption of fluid by intestine (osmotic diarrhoea due to purgatives like magnesium sulphate or malabsorption of glucose or lactate in children).

c. Additional loss of water can also occur due to associated vomiting or fever.

2. Sodium deficit : Diarrhoea causes loss of Na, resulting in Na deficit in all patients, but proportion of Na loss as compared to water loss will decide serum Na concentration and type of dehydration.

a.

b.

c.

In most frequent cases of diarrhoea loss of water and sodium are in the same proportion leading to isotonic dehydration hypovolemia.

In some infants with diarrl1oea net loss of water is in excess of Na (deficit of water is greater than deficit of Na), which ·1eads to hypertonic (hypernatremic) det1ydration.

If net loss of Na is greater than loss of water (deficit of Na is ' v 140 · usually adult patients with diarrhoea· who drink large amount of , water, hypotonic fluids or receive 1.V. infusion of 5°/0-dextrose may develop hyponatremia.

Hypokalemia : Hypokalemia occurs because. fluid. lost in diarrhoea *!s* rich in potassium. (Normally 8-15 mEq potassium ions are excre~ed rn faeces daily. Much greater loss occurs with diarrhoea). Moreover in the process to retain sodium under the influence of aldosterone, potassium is lost in urine and secretion of potassium *in* intestine also increases, which aggravates hypokalemia.

In patiehts with hypokalemia due to diarrhoea, the actual serum potassium concentration may be misleading due to acidosis, water and sodium loss. Therefore, serum potassium concentration may be normal (or high) inspite of loss of total exchangeable body potassium and a low potassium concentration in the cell.

Hyperchloremia : The ileal and colonic mucosa possesses a luminal ci-/HC03 - exchanger that is capable of reabsorbing chloride in exchange of bicarbonate. secreted, more chloride is absorbed from intestine causing hyperchloremia. In attempt to increase sodium absorption (to maintain ECF volume) from intestine and kidney, simultaneous chloride absorption by NaCl

transporter will also aggravate hyperchloremia. So during diarrhoeahyperchloremia occurs.

Metabolic acidosis : Fluid secreted distal to pylorus is rich in bicarbonate. Diarrhoea leads to large amount of HCo3 - secretion (30-45 mEq/L) in the gut which is excreted, and leads to metabolic acidosis. If diarrhoea causes severe hypovolemia or renal failure, renal compensation to loss of bicarbonate is lost and severe metabolic acidosis may develop rapidly. Acidosis may also result from excessive production of lactic acid when patient has hypovolemic shock.

So hyperc~loremic, **hypokalemi.c, metabolic acidosis** occurs in patients **with diar'rhoea.**

1. Oral rehydration therapy (ORT) : As oral rehydration therapy is easily available, simple to use and safe, it is a preferred method of fluid replacement. Losses due to diarrhoea can be effectively corrected by oral rehydration solutions (ORS). Readily available ORS provides Na+, K+, Cl- and bicarbonate along with glucose, which effectively corrects fluid and electrolyte abnormalities, and also provide calories.

Oral rehydration therapy is based on the principle that the intestine actively absorbs glucose and Na is carried with it. Glucose enhances Na and secondary water transport across the mucosa of the upper intestine, even in presence of infective d,iarrhoea.

Avoid correction of losses due to diarrhoea, totally with electrolyte

free solutions (i.e. water, glucose water, tea, soft drinks or commercially available fruit drinks). As it provides only fluid, but lacks electrolytes, it can cause hyponatremia and is not effective in correction of hypovolemia. For detailed discussion on ORT, please refer to Chapter No. 8 ("Fluid therapy in children").

2. Intravenous fluid therapy : I.V. fluid therapy is indicated when rapid correction of blood volume is required for severe dehydration and shock,. inability. of patient to take ORS due to pe,rsistent vomiting or ORT fails to correct volume depletion due to greater losses.

The preferred LV. fluids to correct losses due to diarrhoea are Ringer's lactate and isotonic saline~ But no l.V. fluid is ideal, because they all are deficient in atleast some of the electrolytes required to correct the deficiency found in patients dehydrated :by acute diarrhoea.

To ensure adequate electrolyte replacement, patient needs supplementation of potassium and *I* or .. bicarbonate to l.V .. fluiqs, or ORS should be given as soon as the patient on I. V. fluid is able to drink.

a. Ringer's lactate solution : It is the best commercially available solution. It is the preferred solution because it not only provides an adequate concentration of sodium but also provides bicarbonate (by hepatic conversion of lactate) for the correction of metabolic acidosis. How~ver its potassium

concentration is low (4 mEq/L) and solution provides no glucose to prevent hypoglycemia. So the patient with diarrhoea may require additional potassium, glucose and at times bicarbonate supplementation (1.V. or oral).

b. Isotonic saline : It effectively corrects hypovolemia and provides Na along with water. Isotonic saline does not contain potassium to replace potassium deficit or base to correct metabolic acidosis. So patient may require additio~C:ll supplementation of potassium (10-20 mEq/L) and NaHC03 (20-30 mEq/L) to correct existing hypokalemia and metabolic c. acidosis.

Although isotonic saline lacks K+, adequate supply of sodium and water will prevent urinary loss of K+ by suppressing aldosterone.

Similarly isotonic saline does not correct metabolic acidosis directly, but adequate correction of hypovolemia will improve renal perfusion, which will permit renal correction of f!letabolic acidosis.

5o/o dextrose : It is not an acceptable I. V. fluid because it does not correct acidosis, hypokalemia; and sodium 9eficit.

50;0 de.xtrose :is not effective in correction of hyopovolemia. Rapid infusion of large volume of 5°/o dextrose also carries the risk of hyponatremia and hyperglycemia leading to osmotic diuresis. However D-5°/o with 45 mEq NaHC03 (2 ·amp - :50 ml of 7.5°/o NaHC03) and 20-30 mEq of potassium ·chloride · ·is effective.

FLUID THERAPY IN DIABETIC KETOACIDOSIS

Diabetic ketoacidosis (OKA) is a medical emergency condition that can be life threatening if not treated promptly. OKA is one of the common

complications of type-I diabetes mellitus (IDDM) associated with significant fluid and electrolyte imbalance.

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Definition

Diabetic ketoacidosis is characterized by the triad of hyperglycemia (blood glucose> 250 mg/di), acidosis (HC03 < 15 mEq/L, pH< 7.3) and ketonemia.

Pathophysiology

A. OKA o_ccurs due to reduction in effective concentration of the circulating insulin (decreased insulin secretion or utilization) and concomitant effect of counter-regulatory hormones (catecholamines, glucagon, growth hormone and cortisol).

This hormonal alteration brings about three major metabolic events :

1. Hyperglycemia resulting from accelerated gluconeogenesis (increased hepatic glucose production) and decreased peripheral glucose utilization.

2. Increased proteolysis and decreased protein synthesis.

3. Increased lipolysis and ketone production. Insulin deficiency leads to increased lipolysis, leading to increased plasma and subsequently hepatic free fatty acids (FFA), which leads to increased production of ketone bodies. Glucagon is the primary hormone responsible for inducing hepatic ketogenesis. Common precipitating factors for OKA are inadequate or discontinuation of insulin, irregular diet, infectio n, trauma, surgery, emotional stress, myocardial ischemia etc.

1. Fluid and Electrolyte loss

The renal threshold for glucose is approximately 170 mg/di. Hyperglycemia in OKA leads to glycosuria. This glycosuria leads to osmotic diuresis. So large amount of water and electrolytes are lost. Hyperglycemia indu.ced hyperosmolality causes the movement of water out of the cell with subsequent intracellular dehydration, extracellular fluid expansion and diuresis. Urinary loss then leads to progressive dehydration and volume depletion which causes diminished urine flow and greater retention of glucose in plasma. Vomiting due to OKA also leads to loss of water and electrolytes aggravating hypovolemia.

2. Metabolic Acidosis

In OKA fat is mobilized at an excessive rate to supply energy. As a result, large amount of ketone bodies (B- hydroxy butyric -acid and acetoacetic acid) are formed. These ketoacids are neutralized by bicarbonate buffer, thus serum bicarbonate level decreases leading to metabolic acidosis. Due to large amount of ketones, the anion gap is high.

Severe dehydration induced hypoperfusion leads to impaired renal function, which also contributes to the metabolic acidosis.

3. Potassium Imbalance

Hyperkalemia may occur because of metabolic acidosis and lack of insulin (so potassium will shift from intracellular to extracellular

compartment leading to hyperkalemia).

continuous loss of K• in urine. Potassium is also lost due to vomiting. This Ki- loss leads to deficit of total body potassium. So in OKA, serum K+ may be high, normal or even low but there will be deficit of total body potassium.

~ Other losses

In addition to loss of water, sodium and potassium there is loss of magnesium and phosphorous.

So in OKA, the patient has salt and water loss, hyperkalemia (besides deficit of total body potassium) and metabolic acidosis (with high anion gap). Pathophysiology of loss of fluid and electrolytes is summarized in Fig. No. 5.1.

Q. Roughly how much loss of fluid and electrolytes is expected in OKA ?

A. In diabetic ketoacidosis, usual loss is :

5-8 litre or

100 ml/kg body weight

Water

Sodium

400-700 mEq or 7-10 mEq/kg body weight

Potassium 250-700 mEq or

3-5 mEq/kg body weight

This loss represents loss of water in excess of loss of sodium and therefore, fluid lost in OKA more closely resembles hypotonic saline

solution rather than isotonic solution.

TREATMENT

A. Treatment of OKA can be discussed as follows :

1. Insulin therapy

2. Replacement of fluid and electrolytes

3. Treatment of precipitating factors

4. Avoidance of complications of therapy

5. To monitor treatment

1. Insulin therapy

Selection of insulin : Insulin preferred in treatment of OKA is purified, regular or short acting insulin. As hal~-life of l.V. insulin is not more than 5 minutes, insulin therapy with large loading dose followed by bolus at large intervals is discarded.

Insulin therapy preferred is loading dose followed by frequent small bolus or preferably insulin infusion drip . . With continuous low dose . infusion method lowering of blood sugar is smoother, hypokalemia is less severe and th.ere ar~ lesser chances of hypoglycemia and cerebral oedema.

Loading dose: 0.4 unit/kg body weight (half l.V. and half l.M.)

Subsequent therapy : Loading dose is followed by 5-10 units (or 0~1 unit/kg) of insulin hourly. Insulin infusion is preferred if patient is in ICCU, otherwise insulin is given IM. This bolus is continued till blood glucose reaches 250 mg/di.

. Q. How to prepare and infuse the insulin drip ?

A. In one litre fluid 100 units of insulin is added. After connecting l.V. set, initial 50 .ml fluid is rapidly run through and discarded as insulin particle.s stick to the wall of the tubings. If 1 ml/min (15 macrodrops or 60 microdrops/min) is the rate of this infusion, it will deliver 6 units insulin/hour.

Q. When to increase dose of insulin bolus ?

A. If blood glucose level does not decrease by at least 10% or 50-70 mg/di from the initial value, loading dose is repeated, or the drip rate doubled until blood glucose starts declining satisfactorily. Conversely, if the blood glucose has fallen by greater than 150 mg/di per- hour, the infusion rate should be halved. With insulin therapy control of hyperglycemia is faster than that of ketoacidosis. So once blood glucose falls below 250 mg/di, D-5°/o infusion is started. *D-5oi*

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infusion prevents

hypoglycemia and helps in early · resolution of ketoacidosis. CH. 5 : Fluid Therapy .in Diabetes Mellitus

2. Replacement of fluid and electrolytes . . ..

A. Fluld therapy

As large amount of fluid is lost in OKA, initial treatment priority is

restoration of fluid deficit with proper fluid.

Q. Which fluid to be infused ?

A. Isotonic sali'ne, Ringer's lactate and 5°/o-dextrose are the available and commonly used fluids. For initial treatment, only glucose free fluids (isotonic saline and Ringer's lactate) are given. In shock or hypotensive patient isotonic saline is preferred. Ringer's lactate is given in absence of shock, hypotension and in patient with good urine output. After initial fluid replacement, depending upon the serum sodium, the fluid should be changed to 0.45°/o saline. This allows more free water to be delivered. In OKA there is a greater deficit of free water in comparision to sodium. Ata later stage (once blood glucose falls below 250 mg/di), D-5°/o is i~fused.

. Most of the patients require about 5-8 litres of fluid to correct volume depletion. Fluid resuscitation should be started early and continued until the resolution of the ketoacidosis.

The optimum rate of infusion of isotonic saline. depends on the clinical status of the patient.

Severe hypovolemia and shock: In such patients fluid should be infused as quick as possible. Patients with normal cardiac function should receive the first litre of fluid within the first hour. Fluid replacement should be conti~ued at the rate of one litre per hour (or more) till the correction of intravascular

deficit and haemodynamic stability. Such rapid fluid infusion needs proper, close monitoring.

Maintanence requirement : After rapid fluid replac~ment ~~d in those patients who do not have significant fluid def1c1t,

Fluid Therapy in Diabetes Mellitus

l.V. fluid should be administered slowly. Initially fluid is infused at the rate of 500 ml for the first 4 hours followed by 250 ml/h~ur.

Q. When and why dextrose solution is given ?

A. In OKA D-5o/o is started once blood glucose falls below 250 m.g/dl. N.eed of 5°/o-dextrose at .this stage is :.

1. For b.etter intracellular distribution of free water . (corrects intracellular dehydration).

2. To p revent hypoglycemia and to help in resolution of the ketone bodies.

3. As a prophylactic measureto prevent the late cerebral oedema syndrome.

Q. How to asses s fluid status during therapy ?

A. Norm.ally good urine output reflects proper fluid therapy and reasonable hydration. But in DKA, only urine output will not be a dependable criterion for it. In DKA due to hyperglycemia induced osmoti~ diuresis, urine output will be high even in presence of yolu_me deficit. ,So other parameters need to be

. . considered to assess hydration.

8. Potassium supplementation

Marked urinary and G.I. loss of potassium (about 250-700 ·

mEq or 3 to 10 m'Eq/kg) occurs in OKA. However, initially serum

potassium is usually normal or high due to insulin deficiency

and metabolic acidosis. Regardless of the serum potassium

leveL, total body .reserves are depleted. which can cause life threatening hypokalemia.

Potassium replacement is always necessary but the time of administration varies .. It is dangerous to give potassium in shock

or oliguric patient. After initiation of therapy when blood

pressure becomes normal, urine output is established, blood

sugar starts declining, level of serum potassium is < 4 mEq/L

and when.there is no clue of hyper~alemia on EQG, than only

potassium supplementation is started. Usually ·potassium is

replaced after few hours of treatment with insulin and reversal

of acidosis. However in patients with low or normal initial level

of potassium, replacement may be done earlier.

A. An initial rate of potassium supplementation is 1 O mEq/hour,

when there is no hyperkalemia in · ECG ,and urine output is

adequate.

Once the potassium concentration falls below 4.5 mEq/L

20-40 mEq of potassium is added to one litre of infusion.

If hypokalemia is severe and causes serious cardiac arrhythmias, potassium should be supplemented in doses upto 20-40 mEq/hour under close ECG monitoring. Many patients require oral potassium supplementation for several days after treatmenrof OKA to correct potassium deficit.'

C. Treatment of metabolic acidosis

· A.~ , Proper . fluid ; and . insulin therapy ccin e~fectively correct metabolic acidosis in most of the patients with DKA. Only few patients with metabolic ac::idosis are tree:tted with . bicarbonate therapy because : .

1. Bicarbonate therapy can paradoxically increas·e ketone · p·roductio.n. The basic cause ·of ·the acidosis· is the lack of insulin and the accumulation ·ofketoacids. Therefore insulin 180 · CH. 5 · Fluid Therapy in Diabetes Mellitus . ' therapy · and metabolism of ketone bodies will correct acidosis .

. 2. NaHC03 may.paradoxically increase spinal fluid acidosis and may precipitate coma due to cerebral oedema.

3. Bicarbonate therapy may worsen tissue oxygen delivery by depleting RBC phosphate (2-3 OPG) .

Indications of bicarbonate .therapy in OKA are :

1. Severe metabolic acidosis (HC03 < 9 mEq/L, pH<7.0-7.1) . .

2.

OKA associated with shock.

3. Severe hyperkalemia.

Provide NaHC03 supplementation cautiously. Bicarbonate therapy is stopped when pH reaches 7 .2 to minimize possible detrimental side effects and to prevent metabolic alkalosis as circulating ketones are metabolized to bicarbonate with reversal of ketoacidosis.

A. Bolus infusion of bicarbonate should be avoided except as an emergency resuscitation measure. A solution of NaHC03 44-88 mEq (2-4 Amp of 25 ml 7.5°/o NaHC03) per litre of 0.45°/o saline can be infused as a substitute for isotonic saline, as per the requirement.

D. Correction **of hypophosphatemia**

Decreased oral intake and increased urinary loss of phosphate is known to cause phosphate depletion. Hypophosphatemia frequently occurs during treatment of OKA (in similar way as hypokalemiashown improvement in morbidity. In addition to lack of efficiency, phosphate. Administration . is not without ·risk, since _hyperphosphatemia and hypocalcemia may occur. So reserve , phos.phate administration for the occasional patient who develops .... a severe, symptom aticreduction In the plasma phosphate concentration. Early Initiation of oral intake containing milk can effectively and rapidly correct hypophosphatemfa in most *o1* the

patients .

3. Treatment of precipitating factors

80°/o of OKA occurs due to lack of insulin or due to presence *of* infection. By careful history and evaluation, precipitating factor for OKA is diagnosed and treated meticulously.

4. Avoidance of therapy related complications

Care should be taken to avoid treatment-related complications.

Hyperchloremic acidosis

Possibility of cerebral oedema during treatment of OKA is due 'to too rapid administration of fluid and correction of hyperglycemia leading to imbalance between the brain and ECF. Cerebral oedema can also occur due to excess use of bicarbonate, development of hyponatremia or allowing blood glucose to fall rapidly below 200 mg/di.

For proper monitoring, in all patients with OKA a therapeutic fl v sheet should be maintained, where time and amount of in ufin .. fluid and electrolytes administered, vital data. urin ut .. ut. laboratory parameters are recorded. Chief par m t r · u · d f r monitoring of treatment are :

a.. Blood glucose level

b. Plasma ketone *I* keton urla

c. pH - anion gap 182

CH .-s-

Fluid Therapy in Diabetes Mellitus

a Blood Glucose

· · · · · Effective therapy reduces the blood glucose level. But as hyperglycemia responds faster than ketoacidosis, once blood glucose falls below 250 mg/di, insulin is continued along with glucose infusion till ketosis disappears.

b. , Plasma ketone *I* ketonuria

c. Presence of ketosis is important .tor diagnosis and to decide how long to · treat s·uch patients aggressively. But in DKA, therapeutic . response and extent .of recovery in ketosis is not parallel, so this test is not very useful in monitoring the treatment. Nitroprusside reacts chiefly with acetoacetate, to a lesser extent with acetone and not at all with B-hydroxybutyrate. Usually ratio of B-hydroxybutyrate to acetoacetate is 3: 1 and may reach up to 7: 1 in severe hypoxia or shock.

With proper treatment in OKA patients with shock-hypoxia, . circulation is reestablished and tissue oxygenation is restarted. This leads to conversion of large amount of the accumulated B-hydroxybutyrate to acetoacetate with clinical recovery of the patient. But as the test picks up only acetoacetate and not at all with B-hydroxybutyrate, although total ketone concentration is . falling, paradoxicall"y ketosis may seem to WOr?en.

pH and anion gap

Therapeutic progress can be more accurately. monitored with se~um pH and a~ion gap measurement Rise in the pH and normalization · of increased anion gap suggests response to . therapy. If the anion remains elevated ahd pH is persistently low, this indicates insulin resistance and requires an aggressive increase in the amount of insulin needed. ·

6. Supportive treatment

Supportive treatment is discussed later, along with treatment of hyperglycemic, hyperosmolar nonketotic coma. CH. 5

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Hyperosmolar Nonketotic Coma (HO~K) ; .. .

Hyperglycemic, hyperosmolar rionketotic coma (nonketotic hyperdsmolar syndrome) is usually a complication of NIDDM· in the elderly, in whom severe dehydration and severe hyperglycemia occurs without the development of ketoaCidosis.

This syndrome occurs over a longer interval than. OKA, usually in the patient unable to .drink sufficient amount of water to keep up with the t.frinary fluid loss. A full blown picture does not occur until volume depletion has b~c ome

severe en9ugh to decrease urine output. The severe . glycemia is p·artly caused by decreased renal glucose excretion due to either intri'nsic

underlying renal disease or due to decreased glomerular filtration. and prerenal azotemia secondary to marked dehydration.

When ketoacidosis occurs nausea, vomiting and air hunger brings the patient to the physician before extreme dehydration occurs. As ketoacidosis is absent, clinical symptoms are mild at the early stage and so dicl'griosis is delayed. Usual precipitating factors are drugs (steroids, mannitol, diuretics, and phenytoin) infections or cerebrovascular accident.

..

.-**Diagnosis**

1. Variable CNS symptoms (disorientation to coma).
2. Severe dehydration - hypovolemia.
3. Hyperglycemia, blood sugar > 600 mg/di
4. Absence of ketonemia and acidosis.
5. Plasma osmolality > 320 mOsm/kg
6. Severe azotemia.

Treatment

Therapy of hyperosmolar nonketotic coma should include correction of fluid and . electrolyte deficit, correction of hyperglycemia and hyperosmolarity. Even with the best possible treatment mortality is very high (75°/

0). 184

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Fluid Therapy in Diabetes Mellitus

a Fluid therapy

. The most important measure is rapid administrati~n of large amount of intravenous fluid to reestablish the circulation and unne flow. The.aver.age fluid deficit is 10-11 litres. Initially 0.9°/o isotonic salin.e without additive ' is pr.eferred to correct fluid deficit and 2-3 litres is given in the first ? hours. Subsequently, half strength (0.45°/o) saline can be used to correct relative free water deficit. As hyperglycemia i.s controlled and blood glucose reaches to 300 mg/di, 5°/o-dextrose is s~arted, especially to ·correct intracellular dehydration (to supply tree water). Dehydration in hyperosmolar nonketotic coma is usually more severe than in OKA and 'approximately 12°/o of total body weight fluid ' replacement is required in first 12-24 hours.

b. Insulin treatment

To control severe hyperglycemia, insulin therapy preferably by infusion is given in similar way to OKA. Larger dose of insulin may be necessary . especially in obese patient.

c. Electrolyte management

Potassium salts are usually required earlier in the treatment . of hyperosmolar nonketotic coma than in OKA because the intracellular shift of plasma potassium during therapy is accelerated in the absence of acidosis. If lactic acidosis is present, sodium bicarbonate should be given until tissue perfusion is reestablished. Method and need of administering electrolytes and bicarbonates is similar to OKA.

d. Monitoring of therapy

Blood sugar sh~uld be monitored half or one hourly and electrolytes frequently. Detarls of therapy, vital data, urine output and laboratory parameters are recorded in continuous flow sheet.

e. Supportive treatment

Oth~r additional supportive measur~s to be kept in mind are :

1. Nasogastric tube in unconscious patient.

2. CVP line in patient with cardiovascular disease.

3. Plasma or plasma expander in persistent hypotension.

4. Low dose heparin in comatose obese patient to prevent deep vein thrombosis.

5. Recognition and treatment of precipitating factors i.e. drugs (steroids, mannitol, diuretics'. and phenytoin), infections or cerebrovascular accident.

6. Antibiotics, if infection i~ detected or suspected~

7. ECG : Cardiac monito"ring as a guide to potassium therapy.

a. Bladder catheterization if sensorium is altered.

9. Frequent monitoring of pulse, BP, respiralion and level of consciousness.

FLUID THERAPY IN CHILDREN

Fluid therapy ln children needs better understanding and more careful planning as compared to dults because in children fluid requirement is higher and they are more prone to dehydration.

When to *give I.* V. fluid ?

A. *Oral fluid* replacement is *always* a safe and preferred mode. The indications of I. V. *fluid* therapy are shock, severe dehydration, *uncontrolled* vomiting or diarrhoea, inability to drink, paralytic ileus abdominal distension, impaired sensorium and serious complications.

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Factor	**Mild Dehydration**	**Moderate Dehydration**	**Severe Dehydration**
*Consciousness level	Fully Conscious	Restless, irritable	Lethargic, unconscious
*Ability to drink	Drinks normally	Eager to drink	Unable to drink
*Skin turgor	Instant recoil	Delayed 2-4 sec	Markedly delayed > 4 sec
Capillary refill	Normal < 2 sec	Delayed 2-4 sec	Markedly delayed > 4 sec
Note: Blood pressure mostly remains to be normal even in severe dehydration unless patient develops shock when it drops or may be undetected.			
* Indicates major criteria, there are also minor criteria: depressed fontanel if still open, sunken eyes, absent tears, dry tongue and mucus membrane, tachycardia, decreased or absent urine output.			

SIGNS	CLASSIFY AS	IDENTIFY TREATMENT (Urgent pre-referral treatments are in bold print.)
Two of the following signs: • Lethargic or unconscious • Sunken eyes • Not able to drink or drinking poorly • Skin pinch goes back very slowly	**SEVERE DEHYDRATION**	➤ If child has no other severe classification: — Give fluid for severe dehydration (Plan C). OR **If child also has another severe classification:** **— Refer URGENTLY to hospital with mother giving frequent sips of ORS on the way.** **Advise the mother to continue breastfeeding** ➤ **If child is 2 years or older and there is cholera in your area, give antibiotic for cholera.**
Two of the following signs: • Restless, irritable • Sunken eyes • Drinks eagerly, thirsty • Skin pinch goes back slowly	**SOME DEHYDRATION**	➤ Give fluid and food for some dehydration (Plan B). ➤ **If child also has a severe classification:** **— Refer URGENTLY to hospital with mother giving frequent sips of ORS on the way.** **Advise the mother to continue breastfeeding** ➤ Advise mother when to return immediately. ➤ Follow-up in 5 days if not improving.
Not enough signs to classify as some or severe dehydration.	**NO DEHYDRATION**	➤ Give fluid and food to treat diarrhoea at home (Plan A). ➤ Advise mother when to return immediately. ➤ Follow-up in 5 days if not improving.

A. *Aims* of parenteral fluid therapy are :

1. To correct *fluid* and *electrolyte* deficit

2. To provide maintenance requirements

3. To *replace* ongoing losses

Maintenance fluid and electrolyte requirements

b.

From body weight:

Maintenance requirements calculated as per the body weight is easy to remember and convenient to calculate.

1. Water requirement :

For 1st 1 O kg body weight = 100 ml/kg/day

For 11-20 kg body weight = 1000 ml + 50 ml/kg/day

Above 20 kg body weight = 1.500 ml + 20 ml/kg/day

Example : A 25-kg child will require

2.

Sodium:

10 kg. x 100 + next 10 kg x 50 + rest 5 kg x20

= 1 ,000 + 500 + 100

= 1,600 ml of total fluid

3 mEq/kg or 2-4 m'Eq/100 nil of fluid (as 0.45°/o, 0.33% or 0.2°/o NaCl, which contains 7.5 mEq, 5 mEq and or 2.5 mEq sodium/100 ml of fluid respectively) .

. 3. .Potassium :

2 mEq/kg or 2-4 mEq/100 ml of fluid.

4. Calorie requirement :

100 Kcal for first 1 O kg.

50 Kcal for next 1 O kg.

20 Kcal for remaining weight (above 20 kg.)

A. Modification in normal requirements are :

Decreased requirement : With oligo-anuria, meningitis due to

excessive ADH release and high humidity atmosphere. CH. 8 : Fluid Therapy in Children

3. It contains 28 mEq/L of acetate which will get converted into

bicarbonate by liver and will co'rrect' associated metabolic

acidosis.

4. It will also supply magnesium and phosphate.

5. It contains ·so g/L of glucose which provides caloric requirement

of the child . .

Conclusions :

1. For a child upto 12-13 kg. weight lsolyte-P is ideal.

2. ~or a child with 15-20 kg. or more weight needs lsolyte-P +

additional Na supplementation or I. V. fluid with higher Na

concentration.

A. a. 5°/o-dextrose : As it provides only water and glucose but no

electrolytes, child is at risk of developing hyponatremia and hypokalemia.

b. Isotonic saline or DNS : Normal requirement of sodium in children is roughly 30 to 50 mEq/L (30 mEq/L in young children with lesser weight i.e. 1 O kg, while 45 mEq/L in older children with greater weight i.e. 30 kg), sodium concentration of isotonic saline or DNS is 154 mEq/L (which is 3 to 5 times greater than normal requirement). So maintenance fluid therapy exclusively with it will lead to hypernatremia. Moreover it does not contain any potassium, therefore hypokalemia can occur. So it is not a preferred fluid for maintenance fluid therapy in children.

c.- Ringer's lactate : Ringer's lactate contains 130 mEq/L of sodium, 4 mEq of potassium and does not contain any glucose. As sodiU concentration of RL is 3-4 times greater than the requirement children, ·so RL as maintenance fluid will lead to hypernatre Moreover potassium concentration is very Jess (4 mEq/L Vs t required concentration of 20 mEq/L), so the child will be prone to develop hypokalemia. As RL is free of dextrose chi)d is prone to develop hypoglycemia and starvation ketosis. So RL is not a

suitable agent for maintenance fluid therapy in children.

10°/o dextrose with calcium gluconate (1-2 ml/kg/dose 6 houriy or 100-200 mg for every 100 ml) is the fluid of choice. (10°/o calcium gluconate, 10 ml ampoule contain 4 mEq = 224 mg calcium) The amount of 10°/o dextrose administered is 60-80 ml/kg on the first day with 10°/o increment everyday till it reaches to 160-180 m\/kg/day. Infant receiving phototherapy requires 25°/o increment in daily fluid requirement. No electrolytes are required for the first two days. Later on Na 3 mEq/kg/day and potassium 2 mEq/kg/day are added. So at this stage 1 /5 (0.2°/o) isotonic saline with added potassium chloride or lsolyte-P are suitable 1.V. fluids.

Initial Treatment

A. Aims of initial fluid therapy are :

1. To treat shock

2. To achieve haemodynamic stability

3. To improve tissue perfusion

A. Guidelines for initial fluid therapy are :

1. In shock stage, initial treatment remains the same in iso/ hypo/hypernatremic dehydration. Subsequent treatment differs as per the clinical and biochemic~I status.

2. If shock is present du(ing first hour 20-30 ml/kg isotonic saline or Ringer's lactate is infused. Same dose is repeated if

nec~ssary till pulse is well palpated.

3. After one hour if shock does not improve 1 O ml/kg, 5°/

4. If patient is asymptomatic and/or dehydration is isotonic or hyponatremic, one half of the calculated fluid for 24 hours should be administered in the first 8 hours. The remaining half should be administered in the remaining 16 hours.

Total fluid required= fluid deficfr +maintenance fluid.

5.

Ongoing losses should be assessed frequently and should be added to replacement of deficit and maintenance fluid and electrolytes. In febrile child there is additional 12°/o need of maintenance fluid which should also be calculated and added.

6.

Establishment of normal urine output (2 ml/kg/hour or 50 ml/kg/24 hours) suggests adequate tissue perfusion and reflects adequate fluid replacement. An urine output less than 0.5 ml/kg/hour suggest pathological oliguria and requires assessment to rule out inadequate hydration, development of renal failure or other causes.

7. Along with fluid therapy, specific treatment for the underlying etiology should also be started as early as possible.

Selection of fluid

To correct shock and to increase tissue perfusion the fluid which

rapidly expands volume of extracellular fluid, especially plasma, will be most preferred (see Chapter No. 4). Till urine output is established isotonic saline is the fluid of choice. On failure, 5°/o albumin may be required to treat shock.

In patient with shock, once urine output is established Ringer's lactate is the preferred I. V. fluid because :

1. It's composition is most physiological so avoids fluid electrolyte distur~ances even with rapid infusion.

2. Lactate in RL gets converted into bicarbonate so corrects associated metabolic acidosis, especially in cases of diar~hoea.

However, AL is av~ided in ~evere shock because impaired hepatic conversion of lactate can lead to lactic acidosis, rather than correction of acidosis. RL is also avoided in vomiting 242 induced dehydration where associated metabolic alkalosis gets aggravated by conversion of lactate to bicarbonate.

A. No. Though lsolyte-P is freely available and the most widely used fluid in pediatric practice, lsolyte-P should never be used in the initial treatment of shock, as it has definitive disadvantages.

1. Sodium content of lsolyte-P is just 1 /6 of Isotonic saline (25 mEq/L Vs 154 mEq/L of Na in isotonic saline) hence it's ability to expand the compromised intravascular

~olume and shock will be very poor.

2. Due to low sodium concentration, lsolyte-P can lead to hyponatremia and may worsen the general condition.

3.

lsolyte-P being rich in potassium (20 mEq/L) increases risk of hyperkalemia specially when renal functions are compromised. So use of lsolyte-P can be dangerous with unknown renal status or till the urine output is established.

A. No. 5%-dextrose should not be used to treat the initial phase of shock because

1. 1 litre of D-5o/o increases just 83 ml of intravascular volume (which decides blood pressure), so it is useless to select 'this fluid in the initial treatment.

2. It does not contain any electrolytes, especially sodium, so it will lead to gross electrolyte disturbances. If *?-5°!* is selected as a sole agent for fluid replacement, it can be life threatening.

3. Rapid infusion of So/a-dextrose (> 0.5 gms/kg/hr) w!ll cause osmotic diuresis, which is undesirable and 15 detrimental. n severe dehydration large volume of fluid at rapid rate is required. Rapid infusion of dextrose con•aining fluids i.e. lsolyte P, 5o/o-dextrose etc.) can cause hyperglycemia and subsequent

osmotic diuresis vhich leads to fluid loss and impairs correction of dehydra ·ion. Ho vever dextrose In water can be given I. V. at the rate of 0.5 gm/Kg body\ eigh r without causing glycosuria. Exceptions o this rul e are infants and sic, and malnourished children .1ith less glucose storage. As they are more prone to de 1elop h . poglycemia even the iinit.ial fluid should contain glucose.

A. _ The *fluid* deficit in severe dehydration is about 1 Oo/o of body weight *i.e.* 100 ml/kg, which should be infused as follows :

Infants

rnfant should be given I. V. fl _uid at the rate of 30 ml/kg in first hour, followed by 70 ml/kg in the next 5 hours, thus providing a total of 100 ml/kg in 6 hours.

Older children

Older children should be given l.V. fluid at the rate of 30 ml/ kg within first 30 minutes, followed by 70 ml/kg in the next 2.5 hours, thus providing a total of 100 ml/kg in 3 hours.

After the first 30 ml/kg l.V. infusion, a strong radial pulse should be easily felt. If it is still very weak and rapid, a second infusion of 30 ml/kg should be given at the same rate.

~· After initial stabilisation with AL, most of the children with good oral acceptance are treated with oral rehydration solution (ORS). l.V. fluids are continued in

exceptional cases of nonacceptance of ORS. Type of I. V. fluid selected subsequently depends on the type of dehydration and it's magnitude. In absence of deficit lsolyte-P is preferred.

A. Fluid and . sodium defici_t is corrected slowly in the first 24 to 48 hours, while ~ota~sium deficit in 3 to 4 days. ~sually potassium sup pl e~entat1on ts started after first 24 hours. However, early potassium replacement is necessary with proven hypokalerriia o~ when severe hypokalemia is expected as in severe vomiting with hypochloremic alkalosis, prolonged diarrhoea or diabetic ketoacidosis.

Sodium bicarbonate should be given if serum bicarbonate is below 15 mEq/L. Amount of sodabicarb req~ired (in mEq/L) == 0.4 x body weight (kg) x (desired HCO - measured HCO). The fluid selected is on the basis of underlying etiology and the sodium status of the child. Ringer's lactate is preferred in patient with diarrhoea induced dehydration while isotonic saline is the suitable initial fluid to treat vomiting induced dehydration.

On the basis of sodium status of a child fluid deficit is classified as lsonatremic, Hyponatremic or Hypernatremic dehydration. Table No. 8.4 provides guidelines about amount of fluid and N~ deficit, which enables us to select the right fluid for replacement. Clinically, patients with hyponatremic dehydration are haen:'odynamically

most unstable, while those with hypernatremic dehydration are least unstable.

After proper calculation, sodium concentration in th e fluid to be infused is determined and accordingly fluid is selected. lmpro~er selection of fluid may cause either hypernatremia or hypona~remia. If NaHCO .. is given to treat acidosis, sodium replaced with _the same sho~ld be considered in the calculation. 100 ml of. ~ NaHCO contains 90 mEq of sodium.

VALUE ADDED COURSE

FLUID THERAPY

Annexure II

STUDENT ENROLLMENT LIST (JULY-DECEMBER 2019)

S.No.	University no	Name of the student	Year / CRRI	Signature
1.	U15MB330	NAVEEN ANUSH .R	IVth	Naveen Anush
2.	U15MB331	NAYANA.G.CHANDRAN	IVth	Nayana Chandran
3.	U15MB332	NIVEDHITHA .A.N	IVth	Nivedhitha
4.	U15MB333	NIVETHA. S	IVth	Nivetha
5.	U15MB334	NIVETHITHA. R.N.	IVth	Nivethitha
6.	U15MB335	PADMA SUNDARI.P	IVth	Padma Nila
7.	U15MB336	PIRAI NILA. M	IVth	Pirai Nila
8.	U15MB337	PRAJEETH BALAGE. B	IVth	Prajeeth Balage
9.	U15MB338	PRAKASH .M	IVth	Prakash
10.	U15MB339	PRATIBA SHRUTHY. M	IVth	Pratiba Shruthy
11.	U15MB340	PRAVEEN. R	IVth	Praveen
12.	U15MB341	PREETHIKA. R	IVth	Preethika
13.	U15MB342	PRIYADHARSHINI .R	IVth	Priyadharshini
14.	U15MB343	RAGHAVI .B.R	IVth	Raghavi
15.	U15MB344	RAJESH .K	IVth	Rajesh
16.	U15MB345	RAKESH.R	IVth	Rakesh
17.	U15MB346	RAM KUMAR. S	IVth	Ram Kumar
18.	U15MB347	RAMRAJ. D	IVth	Ramraj
19.	U15MB348	RATCHAKESH. R	IVth	Ratchakesh
20.	U15MB349	REVANTH. C	IVth	Revanth

DEPARTMENT OF ANAESTHESIOLOGY
SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCES
OSUDU, KUDAPAKKAM, PUDUCHERRY-605 502

RESOURCE PERSON

DR. CHANDRASEKAR

COORDINATOR

Dr M KALASREE

Head of Dept. Anaesthesiology
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry -605 502.

Annexure III

MCQS FLUID THERAPY

1. The quantity of glucose in 5% dextrose

a. 32g

b. 50g

c. 41g

d. 5g

2. Correction of hypoglycemia is done by

a.50% dextrose

b.75% dextrose

c.25% dextrose

d 100%dextrose

3. Which of the following is colloid

a. RL

b. DNS

c. Hetastarch

d. Platelet

4. ECG changes in hyperkalemia

a. Wide QRS complex

b. T wave inversion

c. Flattened T wave

d. Tall T wave

5. Features of modearte hypovolemia

a. Dizziness

b. Weaknesss

c. Oliguria < 400ml/day

d. All of the above

6. Assessment of dehydration

a. Dry tongue

b. Loss of skin turgor

c. Sunken eyes

d. All the above

7. Normal serum potassium level

a.1-2 meq/l

b.8-10 meq/l

c.3.5-5.5meq/l

d.None of the above

8. Total body sodium for normal adults

a. 6500 mg

b. 5000mg

c. 4000mg

d. 3000mg

9. Fluid therapy is measured using

a. SpO_2

b. $EtCO_2$

c. CVP

d. None

10. RL is unsafe in following disorder

a. Liver disorder

b. Respiratory disorder

c. Renal disorder

d. Gynaecological disorder

PADMA Sundari. P

Annexure III

MCQS FLUID THERAPY

8/10

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10. RL is unsafe in following disorder

a. Liver disorder

b. Respiratory disorder

c. Renal disorder

d. Gynaecological disorder

Ramraj. D

Annexure III

MCQS FLUID THERAPY

7/10

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Annexure V

Student Feedback Form

Course Name: **FLUID THERAPY**
Subject Code: **ANAES 11**

Name of Student: _____ Roll No.: _____

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear					
2	Course contents met with your expectations					
3	Lecturer sequence was well planned					
4	Lectures were clear and easy to understand					
5	Teaching aids were effective					
6	Instructors encourage interaction and were helpful					
7	The level of the course					
8	Overall rating of the course	1	2	3	4	5

*** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory**

Suggestions if any:

Annexure V

Student Feedback Form

Course Name: **FLUID THERAPY**
Subject Code: **ANAES 11**

Name of Student: Praveen. R Roll No.: U15MB340

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear				✓	
2	Course contents met with your expectations				✓	
3	Lecturer sequence was well planned					✓
4	Lectures were clear and easy to understand			✓		
5	Teaching aids were effective					✓
6	Instructors encourage interaction and were helpful				✓	
7	The level of the course			✓		
8	Overall rating of the course	1	2	3	4	5

*** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory**

Suggestions if any:

Good.

Annexure V

Student Feedback Form

Course Name: **FLUID THERAPY**
Subject Code: **ANAES 11**

Name of Student: Nivedhitha · A · N Roll No.: U1SMB332

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear			✓		
2	Course contents met with your expectations				✓	
3	Lecturer sequence was well planned					✓
4	Lectures were clear and easy to understand				✓	
5	Teaching aids were effective					✓
6	Instructors encourage interaction and were helpful				✓	
7	The level of the course					✓
8	Overall rating of the course	1	2	3	4	5

*** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory**

Suggestions if any:

Excellent.

Date:02.12.2019

From
Dr. M Kalasree
Head Of Department Incharge
Department of Anaesthesia
Sri Lakshmi Narayana Institute of Medical Sciences
Puducherry

To
The Dean,
Sri Lakshmi Narayana Institute of Medical Sciences
Puducherry

Sub: Completion of value-added course: Fluid Therapy

Dear Sir,

With reference to the subject mentioned above, the department has conducted the value-added course titled: Fluid Therapy in July- Dec 2019 for 20 students. We solicit your kind action to send certificates for all the participants, whose name list is attached with this letter. Also, I am attaching the photographs captured during the conduct of the course.

Kind Regards,

Dr. M Kalasree

Encl: Certificates

Photographs

Sri Lakshmi Narayana Institute of Medical Sciences

Affiliated to Bharath Institute of Higher Education & Research

(Deemed to be University under section 3 of the UGC Act 1956)

CERTIFICATE OF MERIT

This is to certify that ___NIVETHA S_____ has actively participated in the Value Added Course on Fluid Therapy held during July - December 2019 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

Dr.CHANDRASEKAR
RESOURCE PERSON

Dr. M KALASREE
COORDINATOR

Sri Lakshmi Narayana Institute of Medical Sciences

Affiliated to Bharath Institute of Higher Education & Research

(Deemed to be University under section 3 of the UGC Act 1956)

CERTIFICATE OF MERIT

This is to certify that ____RAJESH K_____ has actively participated in the Value Added Course on Fluid Therapy held during July - December 2019 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

Dr. CHANDRASEKAR
RESOURCE PERSON

DEPARTMENT OF ANAESTHESIOLOGY
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OSUDU, KUDAPAKKAM, PUDUCHERRY-605 502

Dr. KALASREE M
COORDINATOR

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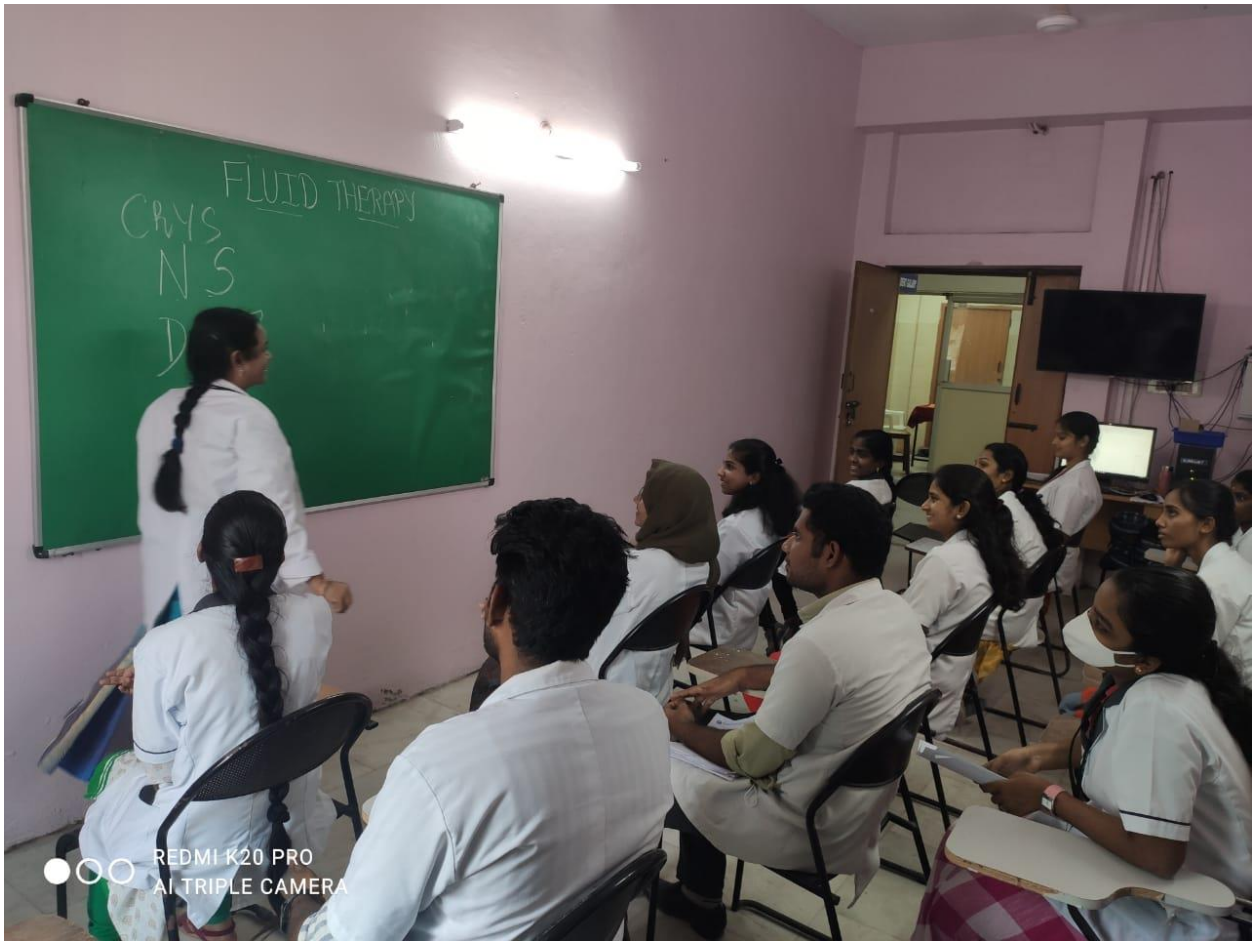
Osudu, Kudapakkam, Puducherry - 605 502.


FLUID THERAPY
CRYS
NS
REDMI K20 PRO
AI TRIPLE CAMERA