SERUM ELECTROLYTES IN UREMIA

Pages with reference to book, From 262 To 265

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Abstract

Sera from seventy uremic patients selected randomly were analysed for their electrolytes (Na+, K + C1 and C02). Marked hyponatremia, hypokalemia and hypochioremia was associated with 51.4,40 and 45.7 percent cases respectively. Fourteen percent cases had serum CO2 concentration above normal showing contraction alkalosis. l.V. fluid used in majority of cases was dextrose in water and combined with the use of diuretic "Lasix" whick may be attributed to the higher percentage of hyponatremia and hypokalemia in this study (JPMA 37: 262, 1987).

INTRODUCTlON

Acid, base and electrolyte balance play a vital role in normal cellular function. Adequate functioning of the cells requires not only the delivery of nutrients but also the maintenance of a constant intra and extra cellular environment. The main role of the kidney is the excretion of the waste products of metabolism like urea, creatinine and uric acid and the regulation of the volume and concentration of the various constituents of the body fluids and hence the maintenance of acid, base and electrolyte balance.[1]

Clinical conditions like uremia which results from direct or indirect interference with kidney functions are accompanied by an imbalance of the acid, base and electrolyte concentrations of body fluid and it is, therefore, impor. tant to monitor and manage these carefully and adequately. In uremia especially gross imbalance occurs because of the wide spread use of plain dextrose for intravenous therapy accompanied by the administration of heavy doses of diuretics like lasix. In our laboratory we have observed sodium levels as low as 101 m.moles/ litre and chloride levels of 32 m.moles/litre. These extremely abnormal results prompted us to investigate in detail the current management of cases of uremia in various medical and surgical units of the two teaching hospitals. Any deficiency in the management reported in this paper are primarily owing to a lack of adequate laboratory facilities round the clock and consequent incapability on the part of the clinicians to monitor the electrolyte levels.

MATERIAL AND METhOD

A total of 70 cases, 52 (743%) males and 18 (25.7%) females, having blood urea more than 80 mg/lOOml with various types of renal failures referred from medical and surgical units of both Khyber and Lady Reading Hospitals, Peshawar were included in this study.

Information about primary diagnosis, duration of illness, type and volume of intravenous fluid administered, type and dose of diuretic therapy given and dietary histories about salts restriction were recorded on a standard proforma.

Four to five mls of plain blood samples were taken for serum electrolytes estimation.

Serum sodium and potassium were determined using emission flame photometry with Gallenkamp flame analyser.

Serum chloride was estimated titremetrically[2] and total CO2 with Harleco micro CO2 appaEatus 64987.

RESULTS

Uremia due to various types of renal failures is mostly prevalent in the age group 31-50 years, the frequency being 41.4%. The frequency in the age group 51-70 years is 24.1%; 28.6% in the age group 11-30 years and 8.6% in below 11 years of age (Table 1).

TABLE – I
Age Distribution.

Age Groups in years	No. of Cases	% age
Up to 10	6	8.6
11 –30	20	28.6
31 –50	29	41.4
51 –70	15	21.4

TABLE – II
Showing Volume Of I.V. Fluid administered/24 Hours.

Volume In MLS	No. of Cases (66)	%age
1000	16	24.3
1500	3	4.5
2000	28	42.5
3000	15	22.7
4000	4	6.0

It can be seen from Table II that 16 (24.3%) cases were administered 1000 mis of I.V. fluid/24 hours. 3(4.5%) 1500 mls/24 hours, 28(42.5%) were administered 2000 mis per 24 hours, 15 (22.7%) were given 3000 mls/24 hours while only 4 (6.0%) were administered 4000 mis of I.V. fluid/24 hours Diuretic therapy was given to 55 (78.6%) cases and the diuretic used was lasix in 45 (81.8%) cases. Forty Five (64.2%) cases were kept on sodium restricted diet with an average duration of 6 days which is also an average duration of illness.

TABLE – III
Distribution of Serum Sodium in M. Moles/L.

Serum Sodium	No.of Cases	%age
101–110	5	7.1
111–120	7	10.1
121–130	24	34.3
131–140	28	40.0
141–150	6	8.6

Table III shows that 12 (17.2%) cases were markedly hyponatremic with mean serum sodium level 110 m.moles/litre. 24(343%) were found to be mild hyponatremic with mean serum sodium level 125 m.moles/litre. 34 (48.6%) cases were in the generally accepted normal range with mean serum sodium 140 m.moles/Iitre.

TABLE – IV
Distribution of Serum Potassium in M. Moles/L.

Serum Potassium	No.of Cases	%age
1.6 – 2.5	06	8.6
2.6 – 3.5	22	31.4
3.6 – 4.5	21	30.0
4.6 – 5.5	13	18.6
5.6 – 6.5	06	8.6
6.6 – 7.5	02	2.8

Table IV reveals that 6 (8.6%) cases were markedly hypokalemic with serum potassium mean level being 2.0 cases were m.moles/litre. 22(31.4%) hypokalemic with mean serum potassium 3.0 m.moles/litre.
Thirty four (48.8%) cases were in the generally accepted normal range with mean serum potassium 4.5 m.moles/Iitre; while 8 (11.4%) cases were moderately hyperkalemic with mean serum potassium level of 6.5 m.rnoles/litre.

TABLE – V
Distribution of Serum Chloride in M. Moles/L.

Serum Chloride	No.of Cases	%age
51 – 60	03	04.3
61 – 70	05	07.1
71 – 80	08	11.4
81 – 90	16	22.8
91 – 100	24	34.3
101 – 110	09	13.0
111 – 120	05	07.1

Table V shows that 8 (11.4%) cases were having severe hypochioremia with mean serum chloride of 60 m.moles/litre. 24 (34.2%) cases were moderately hypochioremic with mean serum chloride level being 80 m.moles/litre. 33 (47.3%) cases were in the generally accepted normal range with mean level of 100 m.moles/litre; while only 5 (7.1%) cases were hyperchloremic with mean serum chloride level of 115 m.moles/litre.
The distribution of serum CO2 is given in Table VI;

TABLE – VI
Distribution of Serum CO_2 in M. Moles/L.

Serum CO_2	No. of Cases	%age
11 – 15	07	10.0
16 – 20	08	11.4
21 – 25	25	35.7
26 – 30	20	28.7
31 – 35	05	07.1
36 – 40	05	07.1

which shows that 7(10%) cases were low in serum CO2 content with mean level of 13.0 m.moles/litre indicating marked acidosis. 8(11.4%) cases were found to be mild acidosis with mean serum carbon dioxide 18 m.moles/litre; while 50 (71.4%) cases were in the usually accepted normal range with mean serum carbon dioxide 30 millimoles/L; and only 5(7.1%) cases were suffering from alkalosis with mean serum carbon dioxide 38 m. moles/litre.

DISCUSSION

The high percentage of hyponatremia and hypokalemia may be due to the common practice of administering I.V. dextrose in water which although is isotonic but leaves water only when glucose is metabolised. When very much water is retained, plasma (Na+) falls because:

Variable degree of salt wasting due to reduced sodium chloride reabsorption are present in a variety of renal diseases. Most patients with renal insufficiency are unable to maximally conserve sodium if placed on a sodium restricted diet, as are in this study (64.2%); in contrast, normal subjects can lower Nat excretion to less than 5 m.moles/day. A patient with renal disease may have an obligatory Na+ loss of 10-40 m.moles/ day.[3]

The decrease in Nat reabsorption appears also to be due to in part to the osmotic diuresis as a result of increased urea excretion in the remaining functioning nephron[4]

This degree of Nat wasting usually is not clinically important since normal sodium balance is maintained as long as the patient is on a regular diet.

However a low sodium diet is potentially hazardous and should be prescribed only with careful monitoring of the patient.

In rare cases, the patient showed a more severe degree of Nat wasting and hypovolemia will ensue

unless the patient maintains a high sodium intake. This occurs primarily in tubular and interstitial disease such as medulary cystic disease, chronic pyelonephritis, polycystic renal disease, the diuretic phase of acute tubular necrosis and after the release of urinary tract obstruction[5-8]

In these conditions the tubular epithelium is damaged resulting in a fall in Na+ reabsorption and obligatory urinary losses that may be greater than 100 m.moles/day. It is therefore suggested that therapy must be directed towards finding the level of Na+ balance and enough Nat must be given to replace obligatory urine losses.

However it should not be assumed that a patient with salt wasting has a normal ability to excrete a Na+ load because some patients with renal failure develop volume depletion with Na+ restriction, may develop Nat retention, odema and hypertension if placed on a diet rich in Nat. In such patients the range of Na+ balance is relatively narrow and must be determined empirically.

Salt wasting nephropathies may also be complicated by hypoka.lemia and hyperkalemia as 39 and 11.4% of cases in the present study has been found (Table IV).

If the impairment in tubular reabsorption occurs in the proximal tubule or loop of Henle, there will be an increase in K+ and water delivery to K+ excretory site in the distal tubule resulting in enhanced K secretion and possibly K+deple tion and hypokalemia. In severe cases daily K+ losses in the urine can exceed 200 L^9 m.moles . However if volume depletion develops because of lack of replacement of urinary Na+ losses, there may be a decrease in renal perfuion thereby reducing K+ excretion and predisposing towards hyperkalemia.

Table V shows that 45% of cases are hypochioremic out of which almost 50% have severe hypochioremia which is a common condition after persistent vomiting, severe burns, diarrhoea, “Salt loss kidney”, uremia and Addison’s Disease.

Chloride concentration is markedly affected by changes in Na+ concentration . In hyponatremia the chloride figure is usually reduced by a similar amount unless ther are also disturbances of acid base balance which affects the HCO3 concentration. Very low figures for chloride down to 50 m.moles/litre have been reported to be associated with marked hyponatremia[1] and in this study 4.3% cases have been noted with serum chloride concentration below 60 m.moles/litre.

When the body is depleted of chloride serum HCO3 concentration increases to maintain an ion concentration.

If table V and VI are compared it can be seen that the 11.4% of markedly hypochioremic cases are counter balanced by 14.2% cases having serum CO2 level significantly elevated, leading to Alkalosis. In such cases, beside the removal of hypovolemia, check should also be kept on electrolytes level and especially when only dextrose in water is administered which, after the glucose being metabolized, leaves only water, which if retained lowers the plasma Na and K+ level considerably. Also, when diuretics are used with dextrose in water I.V. therapy, the check on electrolytes level becomes very much essential.

Lasix which is a more active of diuretics and commonly used (in 81.7% cases in the study under report), acts on the ascending limb of the loot of Henle. It causes excessive diuresis and Na and K+ loss.

ACKNOWLEDGEMENT

Thanks are due to Consultant Surgeons and Physicians of all units of both Khyber and Lady Reading Hospitals Peshawar especially to Prof. Tariq Nishtar for referring their cases. Dr Tasleem Akhtar is greatly acknowledged for her valuable suggestions and guidance and Mr. Mohammad Zaid and Wisal Klian Laboratory Assistants and Mr. Mohammad Hayat for their assistance and typing the manuscript.

REFERENCES

1. Varley, H., Gowenlock, A.H. and Bell, M. Practical clinical bio-chemistry. 5th ed. London, William Heinemann, 1980, V.1.
2. Schales, and Schales, S.S. J. Biol. Chem., 1941; 140:879.
3. Coleman, A.J., Arias, M., Caiter, N.W., Rector, F.C. Jr. and Seldin, D.W. The mechanism of salt wasting in chronic renal disease. J. Clin. Invest., 1966; 45:1116.
4. Yeh, B.P.Y., Tomko, D.J., Stacy, W.K., Bear, E.S., Haden, H.T. and Falls, W.F. Jr. Factors influencing sodium and water excretion in uremic man. Kidney Int 1975; 7:103.
5. Thorn, G.W., Koepf, G.F. and Clinton, MJr. Renal failure Simulating adrenocortical insufficiency. N. Engi. J. Med., 1944; 231 :76.
6. Enticknap, J.B. The condition of the kidney in salt-losing nephritis. Lancet, 1952; 2:458.
7. Strauss, M.B. Clinical and pathological aspect of cystic disease of the renal medula; analysis of 18 cases. Ann. Intern. Med., 1962; 57:373.
8. Buerkert, J.E., Alexander, E., Purkerson, M.L. and Khahr, S. On the site of decreased fluid reabsorption after release of urethral obstruction in rat.J.Lab.Clin.Med., 1976;87: 397.
9. Bricker, N.S., Shwayri, E.I. Reardon, J.B., Keffog, D., Merril, J.P. and Holmes, J.H. Abnormality in renal function resulting from urinary tract obstruction. Am. J. Med., 1957;23:554.