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Diuretics and Hypokalemia *

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A number of studies have been reported in the last three decades regarding hypokalemic action of diuretics.

Hypokalemia is a common and potentially dangerous result of prologed use of diuretics. In most instances, symptoms of hypokalemia such as : nausea, vomiting muscle weakness, loss of apetite are misleading to a clear cut diagnosis of hypokalemia if E. C. G. and determination of potassium level in serum do not confirm the diagnosis. Therefore, it is recommended that serial E. C. G. and determination of serum potassium is mandetory whenever prolonged use of diuretics in a patient is anticipated. In 1948 Terail concluded that the electrocardiogram correlates poorly with the changes in serum potassium and balance studies by Schwartz in 1954 revealed no constant correlation between body potassium and electrocardiogram. However, because of the few patients studied by Schwartz with serum potassium of less than 3 mEq/L; no conclurion on extensive depletion can be made; .

On the contrary, in 1958 Bellet et el; studied 78 patients and concluded that the electrocardiogram was an important adjunct in the diagnosis of Hypokalemia. Surawitz et al in 1959 stated that there was a good correlation between plasma potassium and the electrocardiogram and that, unless tachycardia was a tracing of hypokalemia II would be exp-

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cted in the serum potassium was less that 2. 7 mEq/L. In 1 7 patients with serum potassium less than 2. 7mEq/L, they found that 78 per cent had electrocardiogram typical of hypokalemia. Wearer and Burchell in 1960, in their study of 130 selected patients, also found a definite correlation between the electrocardiogram and the serum potassium. Our present study was initiated because of the variable conclusion was drawn by investigators on hypokalemic action of various diuretics.

We, in our brief but fairly extensive study, tried to deliniate the hypokalemic action of : Mercurial, thiazides, and furosemide on a comparative evaluation of thse diuretics.

METHOD:

1. For a period of six months the reports of all serum electrolyte determination of 28 selected cases taking diuretics (15 cardiac, 6 hepatic and 7 nephritic) were checked.

2. E. C. G. was taken before, during and after therapy on a weekly basis in every individual.

3. Serum potassium, sodium and chloride determination was carried out once weekly.

4. Urea clearance was checked once weekly.

5. Twenty four hours urinary excretion of Na and K and Cl was determined once weekly.

6. All patients were kept on regular hospital diet but no determination of dietary Na, K, and Cl could be carried out at all because of lack of facilities.

7. Symptoms of hypokalemia was carefully looked for in daily examination of the patients.

RESULTS:

1. OSMOTIC DIURETICS : Among this group, Mannitol was extensively tried in our medical service in the past 3 years. Mannitol is a polysacharide with a low molecular weight, which is filtered by glomeruli exclusively and is neither absorbed nor excreted by tubules.

After its adminstration, a fairly good diuresis will be obtained from first to four hours if kidney function is not impaired. Excretion of

Na, K, and Cl is not remarkable nor is the amount of urea after this diuresis' therefore its use will neither cause hyponatremia nor hypokalemia. It shoud be reminded that mannitol is contrindicated in those patients who have moderate or severe kidney failure since its accumulation will cause pulmonary edema.

2. Thiazides. : Thiazides are the most potent oral diuretics we have ever had in our hand for the treatment of edema.

Because of its specific action on both proximal and distal tubules, excretion of chlorides along with Na, K, is always remarkable.

Hypokalemia is a common complication of prolonged use of thiazides. Hyperuricemia is an accidental finding in those thiazides were used over a long period of time. The cause of elevation of serum uric acid is not yet understood. It seems probable that thiazides either accelerate reabsorption of uric acid from proximal tubules or inhibits the excretion of uric acid from this segment of the tubules. In 411 patients taking thiazides in our medical service, we observed the elevation of uric acid in only% II of these patients. A comparative study was also carried out with different thiazides such as Benzithiazides (Pluryl, Dytide) Chlor thiazide (Diuril), Fluthiazide to determine the hypokalemic action of these compounds. We roughly, beleive that triamterene added to some thiazides (Dytide) can delay the occurence of hypokalemia in these who are taking thiazides. In our series, we also noticed that hypokalemia always preceeds hyponatremia and this is quite contrary to what we see in those who take mercurial diuretics in whom hyponatremia preceeds hypokalemia.

3. Furosemide : Furosemide has recently been used in our service and our opinion about its clinical use and diuretic action is summurized as follows :

1. Excretion of 24 hours urinary sodium is remarkable,

2. Excretion of sodium is similar to those of mercurial diuretics .

3. It can be used in edematous nephritic patients with no remarkable side effects. since we noticed no changes in P. S. P. excretion and urea creatinin clearance in those who were taking these compound as diuretics.

4. Mercurial Diuretics : Mercurial diuretics are the most potent diuretics we have ever known It was first introduced to clinicians in 1816. Its action on tubules is due to its inhibitory action on succinic dehydrogenase. Sodium and potassium thus fail to be reabsorbed and will be excret.d in urine. Unfortunately, when creatinine clearance is reduced to 20 ml/min mercurial diuretics fail to reveal any therapeutic action.

We also noticed that whenever urea clearance is low this drugs will show no beneficial action on edema of the patients.

Urinary excretion of sodium and potassium, is remarkable after the use of mercurial diuretics, but hyponatremia always preceeds hypokalemia,

Summary :

We have studied hypokalemic action of different diuretics in a cooperative study on 28 patients and our results are as follows:

1. 28 patients (18 male and 10 female) were the subject of our present study.

2. E. C. G. was taken systematically on all individuals.

3. Clinical signs and symptoms of hypokalemia was looked for in d ıily examination of the patients.

4. Dictary K, Na, and Cl could not be determined because of lack facilities .

5. Determination of urinary Na, K, Cl were systematically carried ed out in all patients.

We strongly believe that small amount of potassium added to diuretics has no beneficial effect on delaying hypokalemia caused by these diuretics.

7. A rich diet containing adequate amount of potassium can satisfactorily prevent hypokalemia.

8. Furosemide and mercurial diuretics similarly cause hyponatremia rather than hypokalemia.

9. Thiazides cause hypokalemia rather than hyponatremia.

10. Addition of triamterene can delay the occurance of hypokalemia in those patients taking thiazides.

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