

The ECG Manifestation of Hyperkalemia in End Stage Renal Disease Patients on Maintenance Haemodialysis

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ABSTRACT:

BACKGROUND:

It is speculated that usual electrocardiographic manifestations of hyperkalemia are less pronounced in patients with end stage renal disease ((ESRD)) than in those with normal renal function, those patients usually have tolerance for hyperkalemia and the usual cardiac and neuromuscular sequelaes are less evident possibly due in part to fluctuation in serum calicium concentraton .

METHODS:

We studied 70 consecutivly selected stable haemodialysis patients to determine the prevalence of ECG changes of hyperkalemia, predialysis serum potassium concentration and pther electrolytes were measured and smltaneous 12- lead electrocardiogram obtained.

RESULTS:

The 70 study subjects ((35 males, 35 females)) age $45.5_{\pm}10.6$ ((range 24-60)) mean duration of ESRD was $22_{\pm}20$ months((range4-60)).

-Mean predialysis serum potassium concentration was $4.7_{\pm}0.9$ mEq/L(range 3.5-7.1).

- Mean precordil leads T wave amplitude for entire group was $5.2_{\pm}4.4$ mm.

- Mean T wave to R wave ratio was $0.4_{\pm}0.1$.

- Mean serum calcium concentration was $9.0_{\pm}0.61$ mq/dl(range 7.5-11.8).

-No study subject had evident arrhythmia.

-There was no significant difference in T wave amplitude ($p=0.11$)or T wave to R wave ($p=0.12$) between quartiles of serum potassium concentration.

-Total serum calcium concentration had inverse relation with T wave amplitude ($p=0.007$).

CONCLUSION:

Haemodialysis patients with hyperkalemia may not exhibit the electrocardiographic changes of hyperkalemia, thus the absence of ECG changes in hyperkalemic haemodialysis patients should interpreted with caution

KAY WORDS: ECG, ESRD, haemodilysis, hyperkalemia.

INTRODUCTION:

There are very few symptoms or signs of hyperkalemia, and these tend to occur only with very high levels. Symptoms generally do not become manifested until the plasma potassium concentration exceeds 7.0 mEq/L ⁽¹⁾, unless the rise in potassium concentration has been very rapid. Other symptoms related to the underlying cause may be present, such as polyurea and polydipsia with uncontrolled diabetes.

Severe muscle weakness or paralysis — Muscle weakness usually begins in the lower extremities and progresses to the trunk and upper extremities. If severe, it can progress to flaccid paralysis ^(2, 3). It is rare to have respiratory muscle weakness, and patients usually have intact sphincter tone and normal cranial nerve exam. Weakness resolves with correction of the hyperkalemia.

Cardiac conduction abnormality — a tall peaked T wave with shortened QT interval is the first change seen on the ECG in a patient with hyperkalemia . This is followed by progressive lengthening of the PR interval and QRS duration. The P wave may disappear, and ultimately the QRS widens further to a

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"sine wave". Ventricular standstill with a flat line on the ECG ensues with complete absence of electrical activity. Ventricular fibrillation or standstills are the most severe consequences.

A variety of other conduction disturbances, including right bundle branch block, left bundle branch block, bifascicular block, and advanced atrioventricular block may also be seen⁽⁴⁾.

There is large inter patient variability in the actual potassium level leading to progression of ECG changes with worsening hyperkalemia, in part related to the presence or absence of concomitant hypocalcaemia, acidemia, or hyponatremia⁽¹⁾. Thus, monitoring of the ECG is essential⁽¹⁾.

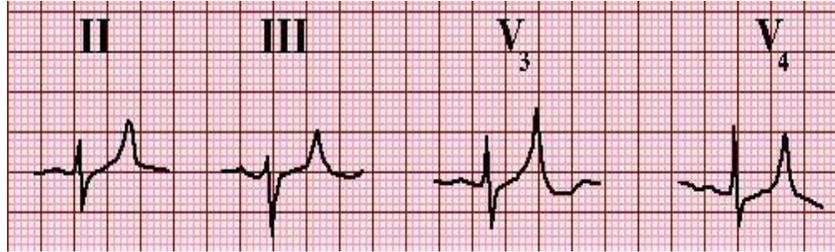


Figure 1: Peaked tented T wave in the ECG of patient with hyperkalemia

PATIENTS AND METHODS:

A prospective study of 70 selected stable patients with end stage renal disease receiving haemodialysis in AL Yarmook dialysis unit and AL Kindey dialysis unit.

Inclusion criteria were:

1. End stage renal disease on maintenance haemodialysis twice weekly for at least 4 month
2. The age 18 year and older
3. Patient acceptance.

Exclusion criteria were:

1. The age less than 18 year
2. Patient refusal.
3. Treatment with drug which affect cardiac conduction

The following information were obtained from each patient:

1. Age
2. Sex
3. Etiology of end stage renal disease (if known)
4. Duration of end stage renal disease

Investigations:

1. Serum potassium concentration
2. Total serum calcium concentration
3. Serum sodium concentration

The above investigations were measured in a blood sample obtained immediately before a regular dialysis treatment.

Laboratory measurement of serum electrolytes were

performed in the central hospital laboratory of the above mentioned hospitals within 1 hour after sampling.

A simultaneous 12 -lead ECG was obtained in all subjects.

The ECG was read without knowledge of the serum K level

The amplitude of the precordial leads T wave was measured and the ratio of the T wave to R wave measured for each patient.

The study group was divided into 4 quartile according to the level of serum potassium

RESULTS:

The 70 study subjects (35 male, 35 females), age 45.5 ± 10.6 (range 24-60), mean duration of ESRD was 22 ± 20 months (range 4-60)

Seventeen (24.2%) of the study group had diabetes mellitus.

Mean predialysis serum potassium concentration was 4.7 ± 0.9 mEq/l (range 3.5-7.1)

Mean pre-cordial lead T wave amplitude for the entire group was 5.2 ± 4.4 mm

Mean T wave to R wave ratio was 0.4 ± 0.1

Mean serum total calcium concentration was 9.0 ± 0.61 mg/dl (range 7.5-11.8)

Mean serum sodium concentration was 136 ± 3.9 meq/l (range 131-145)

No study subject had arrhythmia.

There was no significant difference in "T" wave

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amplitude ($p=0.11$) or "T" wave to "R" wave ($p=0.12$) between quartiles of serum potassium concentration (Table 1)

"T" wave amplitude was equivalent in patient with

serum K concentration >5.5 (7.1 ± 4.1) or <5.5 mEq/l (5.2 ± 3.5 mm) ($p=0.13$)

"T" wave to "R" wave ratio was equivalent in patient with serum K concentration >5.5 (2.8 ± 3) or <5.5 mEq/l (1.9 ± 2.7 mm)

Total serum calcium concentration had inverse relation with "T" wave amplitude ($p=0.002$)

Table 1: Comparison of t wave amplitude and t wave to r wave ratio by quartile of serum k concentration

	First quartile ($k < 4.4$ mEq/l) (15 patients)	second quartile ($k < 4.4 - 4.9$ mEq/l) (25 patients)	third quartile ($k > 4.9 - 5.2$ mEq/l) (27 patients)	fourth quartile ($k > 5.2$ mEq/l) (13 patients)	p
T wave amplitude mm	5.0 ± 2.7	4.8 ± 2.9	5.7 ± 3.8	7.2 ± 5.0	0.11
T wave to r wave ratio	1.7 ± 0.9	1.5 ± 1.4	1.7 ± 1.9	3.4 ± 3.9	0.12

DISCUSSION:

In our study we found that haemodialysis patients with a high predialysis serum potassium concentration (>5.5 meq/l) did not manifest typical ECG changes associated with hyperkalemia. Also there was no difference in T wave amplitude or T wave to R wave ratio between those with low or high predialysis serum K concentration.

Our result confirm the case report by *Szerlip et al* ⁽⁵⁾, of two renal failure patients with a serum K concentration > 9 meq/l who did not have ECG manifestation.

Hyperkalemia reduce the resting membrane potential, bringing it closer to threshold. It slows conduction velocity and increases the rate of repolarization due to an increase in membrane permeability for potassium ⁽⁶⁾.

The early changes in repolarization that may manifest initially as tall peaked T waves in the pre-cordial leads of the ECG are early signs of hyperkalemia ⁽⁶⁾. Tall peaked T waves may be followed by decrease amplitude of R wave, widened QRS complex, prolonged P-R interval, and then decrease amplitude of the P wave ⁽⁶⁾. Finally the QRS blends into the T wave, forming the classic sine wave ⁽⁵⁾.

Cardiac arrest or ventricular arrhythmia may occur at any point in this progression.

There is no clear explanation for the absence of usual ECG findings of hyperkalemia in ESRD, however the finding of inverse relation between total serum

calcium concentration and T wave amplitude suggest that elevated serum calcium concentration in haemodialysis patient may blunt the cardio toxic effects of hyperkalemia ⁽⁷⁾.

Chronic dialysis patients often have an increased tolerance for hyperkalemia as electrocardiographic changes are frequently not seen until the serum potassium concentration exceeds 6.0 to 6.5 mEq/L ⁽⁸⁾. Although secondary hyperparathyroidism is a common feature of ESRD which usually associated with hypocalcaemia, total serum calcium levels in many haemodialysis patients is often above the average due to a regimen designed to increase serum calcium concentration and combat secondary hyperparathyroidism ⁽⁹⁾.

(oral calcium therapy, supplemental vitamin D, and treatment with a dialysate that often has as much as 3.5 mEq/l of ionized calcium).

The total body potassium is decrease in haemodialysis patients ^(10, 11). The elevation in serum potassium concentration tells little about the ratio between intracellular and extracellular potassium, which is the critical factor in membrane depolarization.

The cell membrane changes in ESRD, such as deposition of phospholipids may counteract any of the cardiac effect of hyperkalemia.

In addition, it is suggested that the rate of rise in serum potassium, which is slow in ESRD, may be more relevant than the actual level of serum

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potassium attained. Thus, during a slow rise in serum potassium concentration, concurrent compensatory changes occur to counteract the effect of hyperkalemia on membrane depolarization⁽¹³⁾.

Dialysis patients often have elevations in total body and intracellular potassium; as a result, the transcellular gradient may not be altered with moderate hyperkalemia, resulting in the absence of hyperkalemic changes on the electrocardiogram⁽¹²⁾.

One should be aware of the commonly used medications in ESRD which may cause hyperkalemia like Digoxine, Angiotensin converting enzyme inhibitors, Angiotensin receptor blockers, NSAID, Spironolactone, Amiloride, Heparin, Cyclosporine and Salt substitutes⁽¹⁴⁾.

CONCLUSION:

Haemodialysis patients with hyperkalemia may not exhibit the electrocardiographic changes of hyperkalemia, thus the absence of ECG changes in hyperkalemic haemodialysis patients should be interpreted with caution.

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