International Journal of Medicine Research
ISSN: 2455-7404; Impact Factor: RJIF 5.42
Received: 22-11-2018; Accepted: 25-12-2018
www.medicinesjournal.com
Volume 4; Issue 1; January 2019; Page No. 41-42

Spurious Hyperkalemia can to be misdiagnosed and unnecessary treated

Dr. Priti Dave[1*], Dr. Deepak Bhosle[2], Dr. Anuj Darak[3], Dr. Kathan Doshi[4]
[1-4] Bharati Vidyapeeth Medical College Hospital and Research Centre, Bharati Vidyapeeth Deemed to be University, Pune, Maharashtra, India

Abstract
Pseudo hyperkalemia is an elevation of serum potassium without increase in plasma potassium. A 55 years male, post splenectomy status for splenic abscess was referred to us for persistent hyperkalemia. His renal function test were normal. ECG showed no signs of hyperkalemia. Inspite of repeated corrections by calcium gluconate, insulin dextrose drip and asthalin nebulization serum potassium remained elevated. On doing plasma potassium levels it was found that elevated potassium was Pseudo hyperkalemia. Awareness about this condition can prevent misdiagnosis and unnecessary treatment.

Keywords: pseudo hyperkalemia, postsplenectomy, splenic abscess

1. Introduction
Pseudo hyperkalemia is an elevation of serum potassium without increase in plasma potassium. Hartmann and Mellinkoff noticed this first in 1955 [1]. It should be thought of when serum potassium exceeds plasma potassium by 0.4 mmol/L. It is associated with thrombocytosis and erythrocytosis due to discharge of potassium from platelets and red blood cells during clotting Even increased white blood cells can release potassium and cause factitious hyperkalemia in cases of chronic myelogenous leukemia [1].

2. Case report
A 55 yrs male diabetic and hypertensive patient, postsplenectomy status for splenic abscess was referred to us for persistent hyperkalemia on 10th postoperative day. He was given pneumococcal, haemophilus influenzae, meningococcal quadrivalent conjugate vaccine six days after surgery. His blood sugars were fairly well controlled on regular insulin 18 units in 24 hrs divided before 3 meals. He was having no fever and wound appeared healthy. His complete blood count showed Haemoglobin of 14.5gm/dl, WBC count of 39300/cumm, Platelets of 25,24,000 lacs/cumm. His WBC count before surgery was 28000/cumm and platelet count was normal. His blood culture showed staphylococcus aureus for which Vancomycin (1 gm twice day) along with meropenem1 gm twice a day was given.
His serum potassium was 6.8 meq/L for which he was given calcium gluconate, dextrose insulin drip, asthalin nebulisation and potassium binding resins initially. There was repeated elevation of serum potassium in the range of 5.6 – 6.8 meq/L for next 3 days, in spite of the correction. His Blood urea and serum creatinine was normal. USG abdomen also showed normal kidneys and he was passing adequate urine output. He was not on any drugs which may elevate serum potassium. His WBC was high even after 10 days of Meropenem, However blood culture became negative. Thinking whether is it related to sepsis causing acute kidney injury, repeat CT abdomen was done which showed splenectomy status without any collection of pus. Repeat renal function tests were normal except serum potassium. Reconsidering the situation haematologist reference was taken, fresh serum and plasma potassium (heparin bottle) were repeated taking care of maintaining temperature and sending sample immediately to lab for analysis. Serum potassium came out to be 5.9 meq / l and plasma potassium was normal. All corrections were stopped. Our patient was suffering with pseudohyperkalemia. He was discharged on tablet Aspirin 75 mg with advise to follow up after every 15 days. His Platelets and WBC counts gradually reduced and became normal in 4 weeks His potassium also followed the same course.

3. Discussion
Serum potassium is said to be raised when above 5.5 meq/l. Hyperkalemia is considered severe when serum potassium levels are more then 6 –6.5 meq/L, although variably defined. We all know that it is a clinical emergency needing medical treatment earliest in view of risk of cardiac arrythmias progressing to ventricular fibrillation and cardiac asystole and no physician will be comfortable with the same. Common conditions causing Hyperkalaemia are chronic kidney disease, acute kidney disease and some of the commonly used drugs in hypertension. Diabetes mellitus and cardiovascular disease can indirectly be linked with hyperkalemia[2]. Our patient had normal renal functions, cardiac status was normal and was not on any drugs which might have caused hyperkalaemia. There were no ECG changes like tall T wave, short QT interval and no ST segment depression suggestive of Hyperkalemia.
Serum and plasma Potassium was repeated after proper techniques by phlebotomist by avoiding tourniquet. fist formation providing required temperature and immediately sending the sample to lab to avoid any delay. Serum potassium was reported as 5.9meq/L which was lesser then previous values This time no correction was given. Gamal E H A el Shallaly studied blood changes after Splenectomy in portal hypertension, The Amna Model and found that reactive thrombocytosis (RT >500x10^9/l) occurred after the 7th post-splenectomy day in their both patients and continued from day 8 to week 10. Extreme thrombocytosis (> 1000x 10^9 /L) occurred from day 11 to day 21 postoperatively. The count was back after 17 weeks. Reactive leucocytosis occurred

immediately after splenic artery ligation and returned to normal levels during the 2nd postoperative week [3].

Platelets are sequestrated in spleen due to which reactive thrombocytosis develop in post splenectomy cases, incidence being reported as75% to 82%.It peaks at 1 to 3 weeks post splenectomy and returns to normal in weeks, months to years. This may cause venous thrombosis like mesentric, portal, splenic vein as well sometimes arterial thrombosis. We should be aware to start these patients on aspirin to prevent platelet aggregation or hydroxyurea or plasmapheresis in severe cases [4].

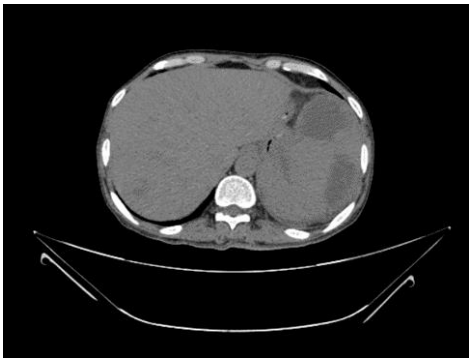

Fig 1: CT scan of abdomen showing splenic abscess for which patient was subjected to splenectomy.

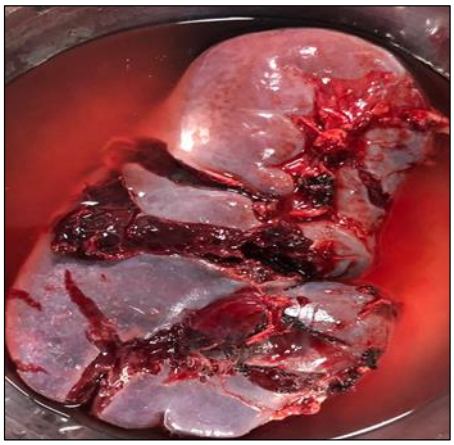

Fig 2: Image showing spleen after surgery.

4. Conclusion

When Hyperkalemia is resistant to correction associated with thrombocytosis or erythrocytosis with no clinical or ECG evidence of elevated serum potassium along with nothing suggestive of CKD or any other etiology one should think of Pseudohyperkalemia. To confirm this diagnosis samples should be sent in standard biochemistry tube and Lithium /Sodium heparin bottle to get both serum and plasma values of potassium. It should be sent at room temperature and processed within one hour of collection. This will avoid unnecessary correction of serum potassium and will prevent development of Hypokalemia.

5. References

1. Sevastos N, Theodossiades G, Archimandritis AJ. Pseudohyperkalemia in Serum: A new insight into an Old Phenomenon. Clin Med Res.2008; 6(1):30-32.
2. Kovesdy CP. Updates in hyperkalemia: Outcomes and therapeutic strategies. Rev Endocr Metb Disor. 2017; 18(1):41-47.
3. ElShallaly G *et al*. Blood Changes after Splenectomy in Portal Hypertension. The ‘Amna Model’. Sudan Journal of Medical Sciences. 2013; 8(1):47-56.
4. Khan PN, Nair RJ, Olivares J, *et al*. Postsplenectomy reactive thrombocytosis. Proc (Bay lUniv Med Cent). 2009; 22(1):9-12.
5. Ferraioli G, Brunetti E, Filice C, *et al*. Management of splenic abscess: report on 16 cases from a single center. International Journal of Infectious Diseases. 2009; 13(4):524-530.