

## Pseudohyperphosphatemia due to contamination with heparin: A case for caution

Sir,

With reference to an informative letter by Amalnath and Dubashi,<sup>[1]</sup> we would like to share our experience of preanalytical error resulting in pseudohyperphosphatemia due to contamination of blood sample with heparin obtained from on central venous catheter.

A 26-year-old software engineer was brought to the emergency department after an episode of syncopal attack. He recovered spontaneously, but was dyspneic and diaphoretic. There were no evidences of seizure activity or incontinence. He denied history of chest pain, palpitation, cough, bleeding manifestations, aura, herbal supplements, illicit drugs, or any regular medication. On examination, he was found to have tachycardia, tachypnea, hypotension, and hypoxia in room air.

His blood chemistry revealed normal serum electrolytes, glucose, blood urea, and creatinine. His coagulation profile and complete blood count were unremarkable. His computed tomography (CT) of the brain was negative for bleeding, aneurysm or an embolic event. Further evaluation revealed deep vein thrombosis involving the origin of the right common iliac vein down to the level of the distal right femoral vein and proximal long saphenous vein. CT pulmonary angiogram showed bilateral pulmonary emboli in the segmental branches and he was given standard anticoagulation treatment with unfractionated heparin.

We were puzzled with his very high and fluctuating levels of phosphorus levels, even though he did not receive any phosphate containing drugs nor alterations in his dietary pattern. There was no evidence of bleeding, hemolysis or rhabdomyolysis. His liver function, lipid profile, plasma lactate, and parathyroid hormone levels were normal. Analytical error was suspected, as the clinical and other laboratory values could not explain hyperphosphatemia. On further interrogation, we noticed his blood samples were drawn from the central line, the patency of which was maintained by heparinized saline. Repeat blood specimens drawn simultaneously from other peripheral vein revealed normal serum phosphorus level (4.6 mg/dl).

Unexplained hyperphosphatemia in chronic kidney disease patients are often blamed to nonadherence of

dietary restrictions or phosphate binders. However, if it is noticed in a patient with a central line, the differential diagnosis of hyperphosphatemia shall include the use of heparin<sup>[2]</sup> or tissue plasminogen activator,<sup>[3]</sup> which may erroneously increase serum phosphate levels due to improper sampling. According to the manufacturer's package insert, heparinized saline is buffered with phosphate solution, which contains approximately 105 mg/dl of phosphate. Based on our case coupled with previous reports,<sup>[2,4]</sup> we attribute the source for elevated phosphate concentration to blood samples contaminated with heparinized solution. Ball *et al.*<sup>[4]</sup> have concluded that even minute amount of heparin contamination contributes to significant elevation of serum phosphate levels. Schiller *et al.*<sup>[5]</sup> suggested adherence to strict blood drawing procedures from indwelling catheters so as to avoid contaminated samples resulting in factitious laboratory values/reports.

The concept of pseudohyperphosphatemia secondary to heparin flush contributing to preanalytical error does not receive due attention. Therefore, it is worth considering spurious hyperphosphatemia in the differential diagnosis of unexplained elevated phosphorus level with normal serum calcium.

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<b>Quick Response Code:</b>	<b>Website:</b> <a href="http://www.indianjnephrol.org">www.indianjnephrol.org</a>
	<b>DOI:</b> 10.4103/0971-4065.133040