Refractory hyperkalemia related to heparin abuse

Sir,

We read with interest the case report of refractory hyperkalemia due to heparin abuse.[1] An effect of heparin is hypoaldosteronism with hyperkalemia caused by direct inhibition of aldosterone biosynthesis

and by inhibition of angiotensin-II with secondary hypoaldosteronism.[2,3] In present case hyperkalemia persisted irrespective of calcium gluconate, dextrose insulin, fludrocortisone, salbutamol nebulizers and slow low efficiency renal dialysis.[1] Patient had acidosis, hypotension, septicemia and died of cardiac arrest.[1]

The potassium-adenosine triphosphate (K-ATP) is a poor inwardly rectifying channel consisting of pore-forming and sulfonylurea – receptor subunit. The pores confer ATP inhibition while the sulfonylurea receptor is the primary target for sulfonylureas, K-ATP channel openers, and nucleoside diaphosphates.[2] Hypoxia, metabolic acidosis and hypercapnia activates the K-ATP channels, resulting in vasodilatation of coronary, mesenteric, renal and smooth muscle bed and increases potassium efflux and modulates many of the kidney transport functions and maintains external potassium balance.[3] The potency of sulfonylurea drugs in antagonizing vasorelaxant action of K-ATP channel stimulation after the sepsis or endotoxin is well recognized in the laboratory model.[2] Reversal of the life threatening complications of hyperkalemia, vasodilator shock and severe bradycardia by the sulfonylurea inhibitor glibenclamide is a novel approach to the treatment of refractory hyperkalemia.[4] In present case refractory hyperkalemia would have been rectified by glibenclamide.[1,4]

The authors working at critical care at tertiary care institute thus could have tried glibenclamide in their case for better outcome.[1,4]

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