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Case Report

Heparin-Induced Hyperkalemia Following Heart Surgery: A Case Report

Farank Kargar[1], MD; Ali Zahedmehr[2], MD; Zahra Shafii[1*], MD; Touraj Babaee[1], MD; Sepideh Banar[3], MD; Yousef Rezaei[3], MD; Saeid Hosseini[3], MD

ABSTRACT

Heparin is widely used in patients undergoing cardiac surgeries on cardiopulmonary bypass; however, it can cause some complications, mainly including bleeding and thrombocytopenia. Heparin-induced hyperkalemia is a rare and less recognized adverse effect of heparin therapy that can lead to serious morbidities. Herein, we describe a 69-year-old Caucasian woman with coronary artery disease who underwent coronary artery bypass graft surgery. The postoperative electrocardiogram was normal without any signs of ischemia or arrhythmia. After the patient was transferred to the intensive care unit, her potassium level was 5.8 mEq/L, which remained high. She had not perioperatively received any causative agent of hyperkalemia. All laboratory tests were within normal ranges. Despite adequate diuresis, a relatively steady state of renal function, and hyperkalemia treatment, including intravenous glucose and furosemide and insulin infusion, the patient's potassium level increased progressively. All etiologies of hyperkalemia were ruled out, and consequently heparin-induced hyperkalemia was suspected. ***(Iranian Heart Journal 2023; 24(2): 104-107)***

KEYWORDS: Heparin, Hyperkalemia, Cardiac surgery

[1] Rajaie Cardiovascular, Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran.
[2] Cardiovascular Intervention Research Center, Rajaie Cardiovascular, Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran.
[3] Heart Valve Disease Research Center, Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran.

*** Corresponding Author:** Zahra Shafii, MD; Rajaie Cardiovascular, Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran.
Email: shafii_zahra@yahoo.com **Tel:** +98212392306

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Heparin has been routinely administered for patients undergoing surgery on cardiopulmonary bypass (CPB) in our daily practice since its first introduction in 1936. [1] Nonetheless, it may be associated with such complications as bleeding and thrombocytopenia. Heparin-induced hyperkalemia (HiH) is a rare and less recognized adverse effect of heparin therapy that can beget serious complications. Herein, to elucidate the pathophysiology and consequences of this phenomenon, we report a case of HiH in a patient undergoing coronary artery bypass graft surgery (CABG) and discuss this clinical entity.

Case Report

A 69-year-old Caucasian woman with coronary artery disease was admitted to undergo CABG. The patient was diagnosed with triple-vessel coronary artery disease and had a positive family history of ischemic heart disease, diabetes mellitus, hypertension, dyslipidemia, diabetic

nephropathy, and atrial fibrillation. Preoperative transthoracic echocardiography revealed a moderate reduction in the left ventricular ejection fraction (40%) along with mild regurgitation in the mitral and aortic valves. The preoperative serum values of sodium, potassium, and creatinine were 137 mEq/L, 4.2 mEq/L, and 1.5 mg/dL, respectively. After heparinization and the attainment of appropriate activated clotting time, CPB was commenced, and the patient was cooled down to 32 °C. For myocardial protection, cardioplegic arrest was performed using the del-Nido solution. The patient underwent CABG, during which 4 grafts were anastomosed to the left anterior descending artery, the posterior descending artery, and 2 marginal arteries. She was weaned off CPB after 93 minutes. The serum level of potassium at the end of the operation was 4.2 mEq/L.

The patient was transferred to the intensive care unit (ICU), where heparin was started as an intravenous infusion along with its routine use to wash arterial and central lines. Her electrocardiogram was normal without any sign of myocardial infarction. At 6 hours post-ICU admission, another measurement of the potassium level showed an increase to 5.8 mEq/L. Other laboratory tests, including arterial blood gas and blood glucose, yielded results within normal ranges. She received no causative agent of hyperkalemia. Despite adequate diuresis, a relatively steady state of renal function, and hyperkalemia treatment, including intravenous glucose and furosemide and insulin infusion, the patient's potassium level increased progressively, and she expired on the night of the operation day due to hyperkalemia heart dysfunction unresponsive to medical treatment.

DISCUSSION

Hyperkalemia and severe hyperkalemia are defined as serum levels of potassium exceeding 5.5 mEq/L and 6.5 mEq/L, respectively, based on the guidelines of the European Resuscitation Council. [2] Hyperkalemia caused by the impairment of renal excretion can be attributed to reduced sodium delivery to the distal nephron, decreased mineralocorticoid activity, or dysfunction in the cortical collecting duct. [3] The main cause of hyperkalemia in hospitalized patients is medication alone or in combination with conditions affecting renal function. [4,5]

Heparin may principally cause thrombocytopenia or skin necrosis; nevertheless, HiH is another adverse effect that can result in hyponatremia/natriuresis. [6] Heparin may also lead to increased potassium retention and sodium loss via hypoaldosteronism. HiH has been reported in patients undergoing CABG. [4,7] Heparin is a mandatory component of the treatment regimen during cardiac surgeries on CPB and afterward. The most probable etiologies for the development of HiH include a reduction in the number and affinity of angiotensin-II receptors in the adrenal glands, the inhibition of the final enzymatic steps of aldosterone formation, and adrenal hemorrhage leading to adrenal insufficiency. [6]

To the best of our knowledge, the existing literature contains only 2 cases of HiH after cardiac surgery. Day et al [4] described a 55-year-old woman with unstable angina who underwent CABG, during which HiH developed following heparin administration for CPB and intra-aortic balloon pump prophylaxis. Within 48 hours after heparin discontinuation, her hyperkalemia was resolved, and she was successfully extubated. Bhaskar et al [7] described a 68-year-old white man with unstable angina who underwent coronary artery revascularization, complicated by HiH in the ICU. After heparin discontinuation, he recovered and was discharged from the

hospital. Our patient also had a clinical presentation similar to the aforementioned cases; however, he did not survive unfortunately. We considered HiH to be the most likely cause of hyperkalemia in our patient for the following reasons:
1) The patient had no known previous history of hyperkalemia. 2) She did not take any medications causing hyperkalemia. 3) There was no significant decrease in her renal function or urine output. 4) Her urinary pH was normal. 5) Her hyperkalemia continued (or persisted) despite the implementation of recommended treatments until heparin discontinuation.
The management of HiH has been elaborated upon in the literature based on the abovementioned pathogenesis (ie, hypoaldosteronism). The mainstay of therapy for HiH is heparin discontinuation; still, it might be life-threatening, particularly in patients needing intravenous anticoagulation for the prophylaxis or treatment of thromboembolic events. Therefore, the multifactorial approach to the management of these cases can be of great significance since some contributors to HiH, such as renal dysfunction, potassium hemostasis, and preoperative potassium-elevating medication, can be modified before cardiac surgery. In addition, the intraoperative use of the priming solution and diuretics may also significantly contribute to HiH development, which should be considered in high-risk patients. Moreover, fludrocortisone acts through direct effects on the distal tubules, thereby promoting potassium excretion and helping with the treatment of HiH. [8]
In conclusion, HiH is a very rare post-cardiac surgery complication that can develop in high-risk patients possessing some of the contributors to this phenomenon, including renal dysfunction, diabetes mellitus, and preoperative potassium-elevating medication. Surgeons should keep in mind such a rare and life-threatening entity in their daily practice so that they can implement appropriate diagnostic and therapeutic modalities to improve patient outcome in the postoperative period.

Conflict of Interest
The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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