# Refractory Hypokalemia While Weaning Off Bypass

**Abstract**

Hypokalemia is defined as serum potassium level less than 3.5 mEq/L. When the serum level of potassium is less than 3 mEq/L, intravenous potassium supplementation is warranted. A 23 yr old adult female with complaints of dyspnoea (NYHA II) since 6 yrs, dyspnoea (NYHA III) and paroxysmal nocturnal dyspnoea on and off since 2 months, diagnosed with severe mitral stenosis, was posted for mitral valve replacement. After the release of ACC, ECG revealed sine wave pattern, Transesophageal echocardiographic examination revealed global hypokinesia and ABG showed potassium of 2.3 mEq/L. Hypokalemia in cardiac patients can occur due to the effect of poor oral intake, increased renal loss by the secondary hyperaldosteronism in congestive heart failure, loss due to use of digoxin and diuretics like thiazide diuretics, loop diuretics etc. Hypokalemia should be avoided while weaning off cardiopulmonary support as it can lead to atrial and ventricular arrhythmias. Potassium ion is very important for the normal contractility of the heart. Hypokalemia if refractory to intravenous potassium supplementation, concomitant magnesium deficiency should be suspected and treated.

**Keywords:** *Cardiopulmonary bypass, hypokalemia, magnesium, refractory*

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## Introduction

Hypokalemia is defined as serum potassium level <3.5 mEq/L.[1] When the serum level of potassium is <3 mEq/L, intravenous (IV) potassium supplementation is warranted. The electrocardiograph (ECG) manifestation of hypokalemia includes T-wave flattening followed by T-wave inversion, ST depression, prominent U waves, prolongation of QTc interval, torsades de pointes, and asystole. It can even lead to sudden cardiac death. Here, we report a case of refractory hypokalemia while weaning the patient off cardiopulmonary bypass (CPB) leading to ECG changes and poor left ventricle function.

## Case Report

A 23-year-old female with complaints of dyspnea New York Heart Association (NYHA) Class II for 6 years and dyspnea (NYHA III) with on and off paroxysmal nocturnal dyspnea for 2 months was diagnosed with severe mitral stenosis. She was planned for mitral valve replacement and written informed consent was taken. She was on tablet atenolol 25 mg od and tablet torsemide 10 mg od preoperatively. ECG showed atrial fibrillation with controlled ventricular rate. Blood reports revealed hemoglobin of 12.9 g/dL, serum potassium of 3.1 mEq/L, total bilirubin was 1.5 mg/dL, and lactic dehydrogenase was 490 U/dL. All the other blood reports were within normal limits. On the morning of surgery, the patient was shifted to operation theater, intravenous access secured with 16G cannula, arterial line secured in left radial artery, and right internal jugular vein cannulated with 8 Fr 4 lumen catheter. Standard monitoring was done and the patient was induced with injection fentanyl, midazolam, etomidate, and pancuronium; intubated and maintained with oxygen, air, and isoflurane inhalation. After heparinization (200 mg), aortic and bicaval cannulation was done; CPB was initiated. After the aortic cross-clamping, sanguineous antegrade root cardioplegia was instituted and mitral valve was replaced with size 29 SJM valve. Arterial blood gas (ABG) revealed a potassium of 2.8 mEq/L. 10 mEq of potassium replaced on the pump.

After the release of aortic cross-clamp, ECG revealed sine-wave pattern and ST elevation with features of low cardiac output. Transesophageal echocardiographic examination revealed global hypokinesia and ABG showed potassium of 2.3 mEq/L.



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The patient was given potassium 5 mEq, then 10 mEq and followed by 20 mEq while monitoring the arterial potassium levels on ABG. The ECG changes persisted and magnesium infusion was then started 2 g given slowly over 2 h, suspecting concomitant hypomagnesemia. After starting magnesium infusion, the ECG changes began to normalize and gradually, contractility of the heart also improved. The patient was then weaned off CPB with noradrenaline at 0.1 μg/kg/min, dobutamine 8 μg/kg/min, and adrenaline 0.06 μg/kg/min infusion. Urine output was 1.5 ml/kg/h after weaning off bypass. Postoperatively, the output was 1 ml/kg/h so injection furosemide 20 mg iv bd was given. Postoperatively, the patient had persistent hypokalemia that responded to magnesium infusion that was continued on postoperative day 0 and 1. Serum magnesium evaluated on postoperative day 0 and 1 was 0.7 mmol/L and 0.9 mmol/L. The patient was weaned off the ventilator and extubated after 16 h and weaned off the inotropic support over 36 h. She was discharged on postoperative day 3 from ICU.

## Discussion

Hypokalemia in cardiac patients can occur due to the effect of poor oral intake, increased renal loss by the secondary hyperaldosteronism in congestive heart failure, and loss due to use of digoxin and diuretics such as thiazide diuretics and loop diuretics.[1] Hypokalemia can lead to nonspecific effects such as muscle cramps, generalized weakness, fatigue, and hypertension, which are often implicated as features of cardiac illness; hence goes undetected.[1,2] If serum potassium drops below 3 mEq/dL, IV potassium infusion diluted in normal saline while monitoring clinical status and serum potassium is recommended.[3,4] Glucose in the diluent can cause a further intracellular shift of potassium and reduction in serum potassium levels; hence to be avoided.[3,5]

Loop diuretics and digoxin cause concomitant hypomagnesemia.[6] Intracellular $Mg^{2+}$ is a critical determinant of renal outer medullary potassium (ROMK)-mediated $K^+$ secretion in the distal nephron. $K^+$ is taken up into cells across the basolateral membrane through Na-K-ATPases and secreted into luminal fluid through apical ROMK channels. Intracellular $Mg^{2+}$ binds and blocks the pore of the ROMK channel from the inside, thereby limiting outward $K^+$ flux.[7] Mg deficiency causes impaired functioning of Na-K pump and intracellular K depletion.[8] Rapid exchange of Mg occurs between myocardium and plasma thereby leading to profound effects on the myocardium.[9]

Hypokalemia should be avoided while weaning off cardiopulmonary support as it can lead to atrial and ventricular arrhythmias.[10] Hence, hypomagnesemia along with hypokalemia can present with features of difficult weaning from CPB support.

## Conclusion

Potassium ion is very important for the normal contractility of the heart. Hypokalemia if refractory to IV potassium supplementation, concomitant magnesium deficiency should be suspected and treated.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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### Conflicts of interest

There are no conflicts of interest.

## References

1. Weiner ID, Wingo CS. Hypokalemia – Consequences, causes, and correction. J Am Soc Nephrol 1997;8:1179-88.
2. Krishna GG. Effect of potassium intake on blood pressure. J Am Soc Nephrol 1990;1:43-52.
3. Ahlawat SK, Sachdev A. Hypokalaemic paralysis. Postgrad Med J 1999;75:193-7.
4. Kruse JA, Carlson RW. Rapid correction of hypokalemia using concentrated intravenous potassium chloride infusions. Arch Intern Med 1990;150:613-7.
5. Griggs RC, Resnick J, Engel WK. Intravenous treatment of hypokalemic periodic paralysis. Arch Neurol 1983;40:539-40.
6. Cohn JN, Kowey PR, Whelton PK, Prisant LM. New guidelines for potassium replacement in clinical practice: A contemporary review by the national council on potassium in clinical practice. Arch Intern Med 2000;160:2429-36.
7. Huang CL, Kuo E. Mechanism of hypokalemia in magnesium deficiency. J Am Soc Nephrol 2007;18:2649-52.
8. Agus ZS. Hypomagnesemia. J Am Soc Nephrol 1999;10:1616-22.
9. Chakraborti S, Chakraborti T, Mandal M, Mandal A, Das S, Ghosh S, *et al.* Protective role of magnesium in cardiovascular diseases: A review. Mol Cell Biochem 2002;238:163-79.
10. Licker M, Diaper J, Cartier V, Ellenberger C, Cikirikcioglu M, Kalangos A, *et al.* Clinical review: Management of weaning from cardiopulmonary bypass after cardiac surgery. Ann Card Anaesth 2012;15:206-23.