Intraoperative hyperkalemic cardiac arrest during revascularization surgery

Mamatha Kadiyala MD
Baystate Health, mamatha.kadiyala@baystatehealth.org

Stanlies D’Souza MD
Baystate Health, dsouzastan@yahoo.com

Follow this and additional works at: https://scholarlycommons.libraryinfo.bhs.org/all_works

Part of the Medicine and Health Sciences Commons

Recommended Citation
INTRODUCTION
Reperfusion following acute limb ischemia results in systemic inflammatory response which depends on duration and severity of ischemia. We report a case of fatal hyperkalemic cardiac arrest following reperfusion from our institution.

CASE DESCRIPTION
An 88 year old female with longstanding vascular disease, previous right axillo-femoral bypass, recent DVT in left lower extremity treated with lovenox, complicated by GI bleed after which anticoagulation was discontinued, presented with cold and numb right lower extremity, new onset of atrial fibrillation and elevated cardiac enzymes. Patient and her family refused amputation and decided on no perioperative resuscitation. She was taken to the OR emergently with plans to redo right axillo-femoral bypass. On reperfusion patient suddenly developed 4-5 beats of wide complex QRS followed by asystole. Patient did not respond to a bolus of calcium chloride. Subsequent blood sample was obtained resulting in potassium above 9 mmol/L.

REFERENCES
1. Cardiovascular Surgery 2002;620–30
2. Tex Heart Inst J 2006;33:40-7
3. Thorac Cardiovasc Surg 2000;48:274-78
4. Anesthesiology 2001;94:1133–8

DISCUSSION
Irreversible muscle cell damage starts after 3 h of ischemia and is nearly complete at 6 h. Reperfusion injury after extremity ischemia consists of two components: local response, which manifests as limb swelling with its potential for aggravating tissue injury and the systemic response due to release of inflammatory mediators such as histamine, complement, thromboxane, and bradykinin leading to multiple organ failure and death. Consequences of influx of large amounts of muscular waste products are systemic hyperkalemia, acidosis and potentially fatal arrhythmias. EKG manifestations of hyperkalemia include tall T waves, widening of QRS complexes, small P waves, prolonged PR interval and multifocal ventricular complexes leading to ventricular tachycardia and cardiac arrest. Management includes a combination of calcium gluconate, (membrane stabilizing effect), sodium bicarbonate and furosemide (to enhance renal excretion of potassium). The effects of reperfusion depend on the duration of ischemia. Obtaining a good history and discussion with surgeon about possible complications and surgical interventions is crucial. Some studies have shown that ischemic tolerance times can be increased with systemic anticoagulation and cooling. Nonetheless early revascularization of acute limb ischemia is a key to minimize reperfusion injury and its consequences. Modifying conditions of reperfusion like controlled reperfusion with appropriate clamp reapplying technique, changing the composition of the reperfusate can minimize morbidity and mortality. Studies have proven that therapeutic strategies such as antioxidant, complement and neutrophil therapy significantly prevents or limits reperfusion injury.

CONCLUSION
Anticipating consequences of reperfusion and understanding pathophysiology, manifestations and treatment of hyperkalemic response after revascularization of the acute ischemic limb may be life saving.