

Geisinger Acute Right Heart Failure Following Anesthetic Induction In a Previously Undiagnosed Pectus Excavatum Patient



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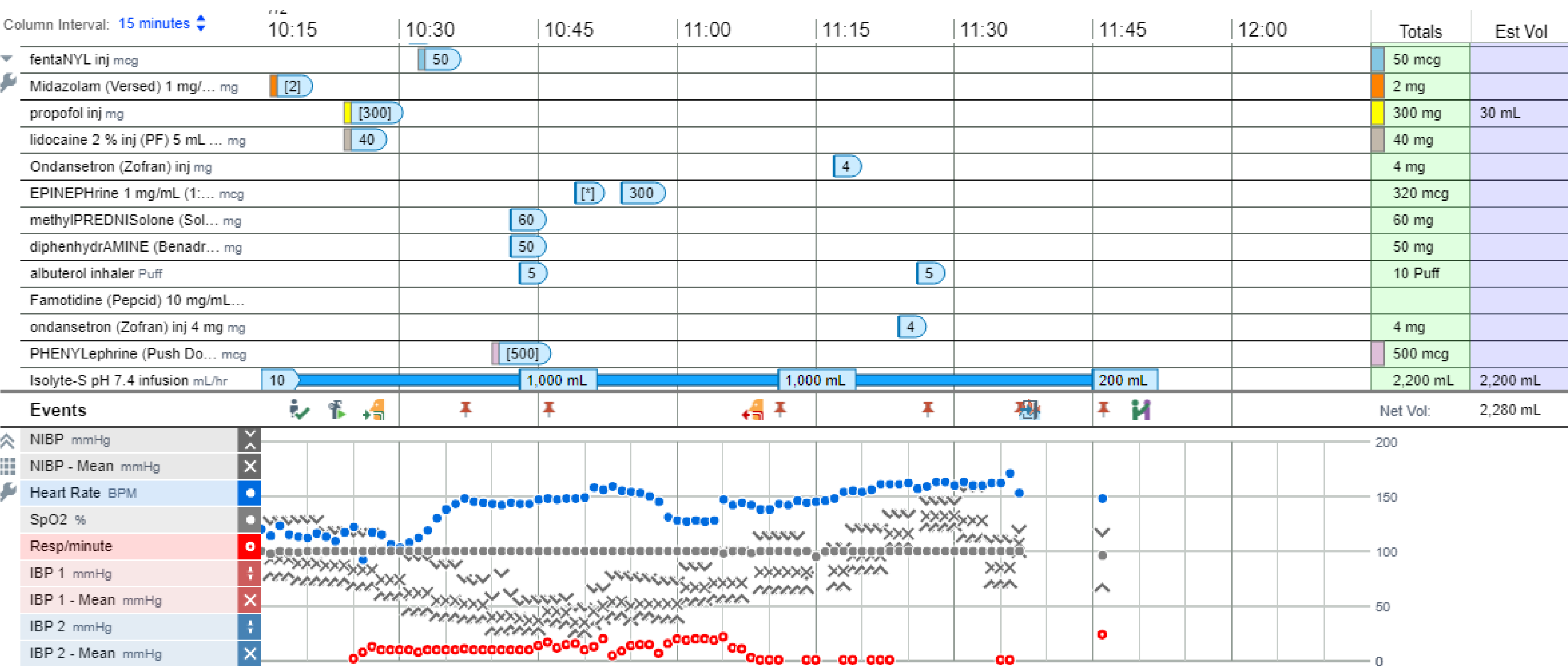
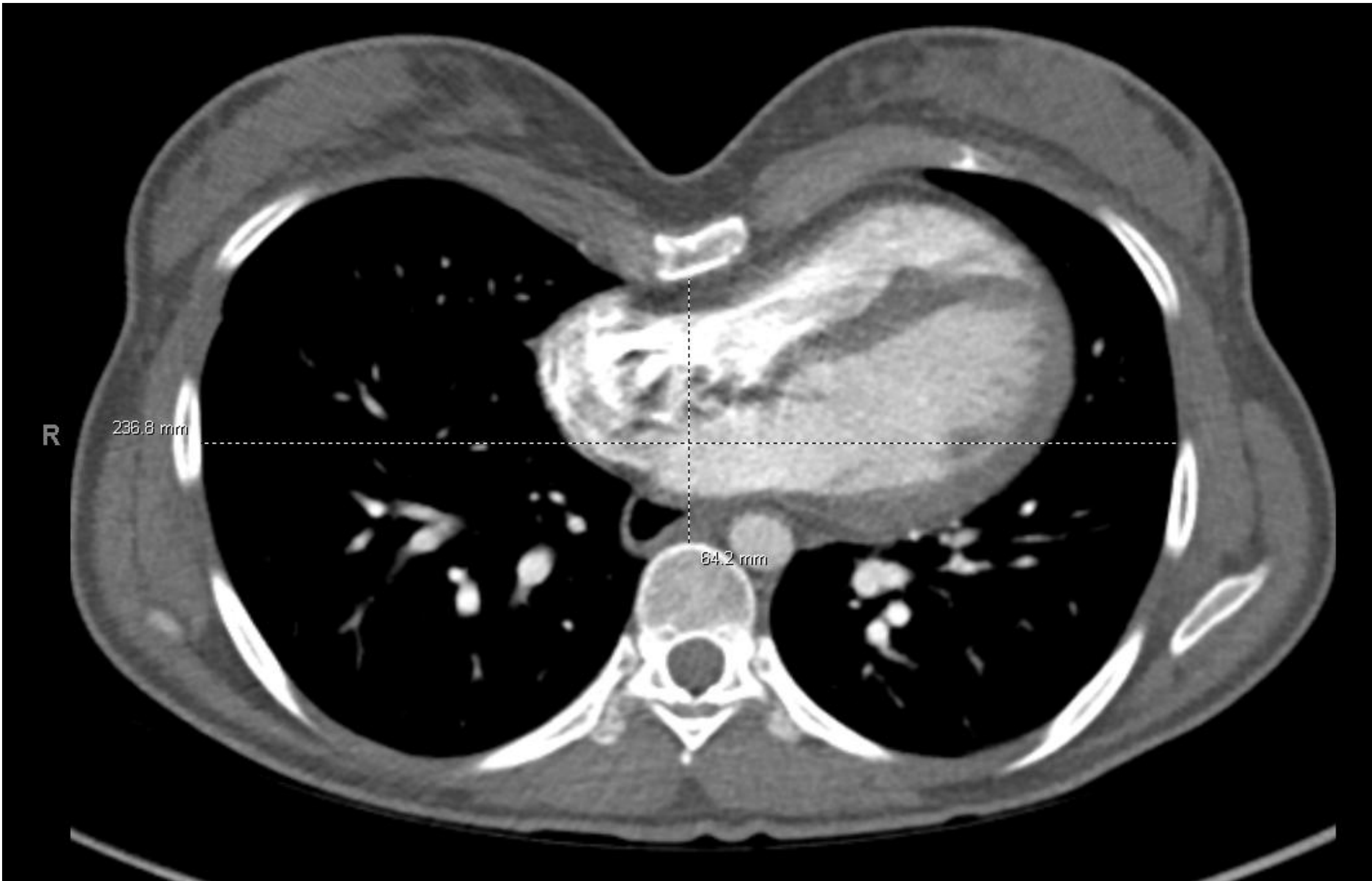
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Introduction

Anesthetic induction with propofol and positive pressure ventilation (PPV) are known to elicit several physiologic and hemodynamic changes. Vasodilation and myocardial suppression, secondary to the effects of propofol, and decreased right ventricular preload, due to increased intrathoracic pressure secondary to PPV, are of chief concern in patients with preexisting cardiac conditions. These factors may coalesce and lead to a reduction in stroke volume, resulting in decreased in end-organ perfusion.

Case Description

26-year-old female with no significant past medical history (ASA I) presented for elective podiatric surgery. After induction of anesthesia and LMA placement, she developed sudden tachycardia. Her heart rate increased from 118 to 140 bpm, with ST segment elevations on EKG. Anaphylaxis was initially suspected due to the rapid onset of symptoms, prompting abortion of the procedure, removal of the LMA, and collection of serum tryptase to assess for mast cell degranulation. Upon emergence, the patient was alert but complained of chest pounding and shortness of breath. She was treated with albuterol, which alleviated her symptoms. Postoperative CT imaging revealed previously undiagnosed pectus excavatum with significant right heart compression and a Haller Index of 3.67. These findings suggest that the patient experienced acute right-sided heart strain due to mass effect from her chest wall anatomy, unmasked by anesthetic-induced changes in thoracic dynamics.



Discussion

Pectus excavatum can cause significant cardiovascular compromise during anesthesia due to mechanical compression. The sternal depression reduces the anteroposterior diameter of the chest, resulting in right ventricular compression and decreased right and left ventricular stroke volume. During induction, anesthetics may exacerbate the symptoms of reduced right heart function through muscle relaxation and positive pressure ventilation, which can both further reduce venous return and cardiac output.

The degree of impairment typically correlates with the severity of the chest wall deformity, measured by the Haller index. Our patient had a Haller index of 3.67, placing her in the severe category. One study demonstrated that an elevated Haller index is inversely correlated with right ventricular ejection fraction. This anatomic finding put our patient at high risk for severe hemodynamic instability during induction.

In response to decreased stroke volume and cardiac output, the body may compensate with reflexive tachycardia to maintain perfusion. Following induction, our patient developed hypotension and tachycardia consistent with acute right heart failure. She was subsequently diagnosed with right heart failure secondary to mass effect from previously undiagnosed severe pectus excavatum.

This case highlights the importance of preoperative recognition of pectus excavatum and its potential impact on anesthetic management.