

Atrial dysrhythmias due to extrinsic left atrial compression by an esophageal stent



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Introduction

Arrhythmogenesis resulting from extrinsic cardiac compression is a well-observed but poorly understood process. The left atrium has generated the most attention given its proximity to several structures, with scattered case reports describing this phenomenon in relation to esophageal malignancy¹ as well as aortic dilation, hiatal hernia, achalasia, and bronchogenic cysts.² Perhaps the best-studied association between mechanical compression of the heart and arrhythmia is pectus excavatum, which is thought to contribute to the development of atrial fibrillation.³ Although an exact mechanism is unfounded, hypotheses include direct epicardial compression and irritation owing to local inflammation, as in the case of pericarditis;⁴ pain and increased sympathetic drive; neural reflexes triggered by distorted esophageal anatomy; and deglutination reflexes, as is hypothesized in swallowing-induced tachyarrhythmia.⁵ We present a patient with multiple supraventricular arrhythmias triggered by compression of the left atrium by an indwelling esophageal stent. We are aware of only 1 previous report of atrial fibrillation occurring in the setting of significant left atrial compression from an esophageal stent.⁶ To our knowledge, this case is the first demonstrating both atrial fibrillation and atrial tachycardia related to extrinsic left atrial compression, and also the first to demonstrate resolution of these arrhythmias after stent removal.

Case report

A 36-year-old man was admitted to our institution with failure to thrive, hypotension, palpitations, and difficulty tolerating oral intake in the setting of a recently implanted esophageal stent. The patient had no significant medical history until discovery of stage IVa esophageal squamous cell carcinoma

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KEY TEACHING POINTS

- Esophageal stenting is an uncommon cause of atrial arrhythmias that should be considered in the appropriate clinical context.
- Extrinsic epicardial irritation is a plausible reason for atrial arrhythmias and should be considered, particularly if a variety of arrhythmias are observed in a relatively short period of time.
- Removal of the extrinsic compression force may alleviate arrhythmias by reducing epicardial irritation and/or limiting atrial stretch.

5 months prior. His condition was complicated by esophago-bronchial fistula to the left main bronchus and resultant left lower lobe pulmonic abscess. At the time of diagnosis, he underwent placement of a partially covered esophageal stent to both palliate esophageal obstruction and exclude the esophago-bronchial fistula. Following this procedure, he developed some intermittent chest pain attributed to his stent placement. Tachycardia was noted on vital signs during routine outpatient follow-up visits, but no electrocardiograms (ECGs) were performed. Chemotherapy with carboplatin and paclitaxel as well as radiation therapy was completed 1 month prior to his presentation. After growth of tumor into the proximal aspect of the original stent, a second, more proximal fully covered stent was placed, which overlapped with the original stent by 7.5 cm. The distal portion of the second inner and more proximal stent terminated in the mid to distal esophagus immediately posterior to the superior left atrium (Figure 1). Soon thereafter, he experienced worsening chest pain and palpitations. He was referred to the emergency department by his outpatient provider, given his failure to thrive, and was noted to have narrow complex tachycardia upon arrival. The differential diagnosis of new-onset supraventricular tachycardia in a young male with failure to thrive and a recent diagnosis of cancer is broad, including but not limited to hypovolemia, hypoxemia, pulmonary embolism, inflammatory or infectious states, metabolic abnormality or electrolyte derangements, hyperadrenergic state, structural heart disease, and toxin ingestion.⁷

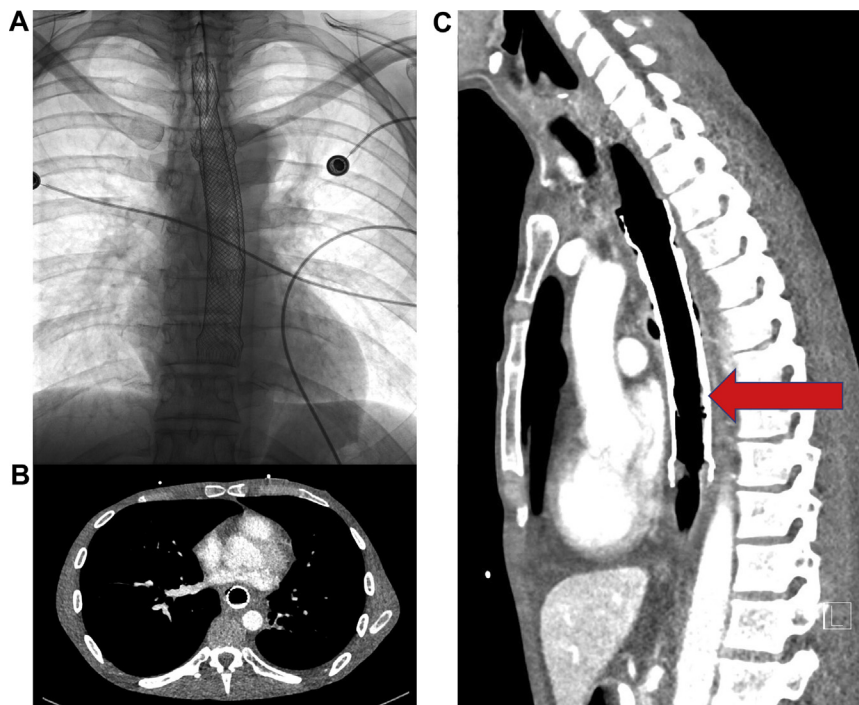


Figure 1 Imaging of esophageal stent. **A:** Radiologic findings of esophageal stent with stent-in-stent seen on fluoroscopy at the time of implantation. Stent is further visualized on computed tomography demonstrating its proximity to the heart and compression of the posterior left atrium in the transverse (**B**) and sagittal (**C**) planes. Note the termination of the inner stent at the level of the left atrium (*red arrow*).

Laboratory findings on admission demonstrated potassium (4.7 mmol/L), magnesium (2.2 mmol/L), thyroid stimulating hormone (2.472 mIU/mL), white blood cell count ($5.5 \times 10^9/L$), troponin I (<0.034 ng/mL), D-dimer (169 ng/mL), and unremarkable urine toxicology screen. Apart from his tachycardia, his vital signs were stable, with a blood pressure of 132/87, a temperature of 36.8°C, a

respiratory rate of 20, and an oxygen saturation of 100% on room air. His physical exam was most notable for cachexia. He was treated with aggressive fluid resuscitation, without improvement in his tachycardia.

At the time of the initial cardiology evaluation, electrocardiographic monitoring showed a narrow complex regular tachycardia at 125 beats per minute. Adenosine was

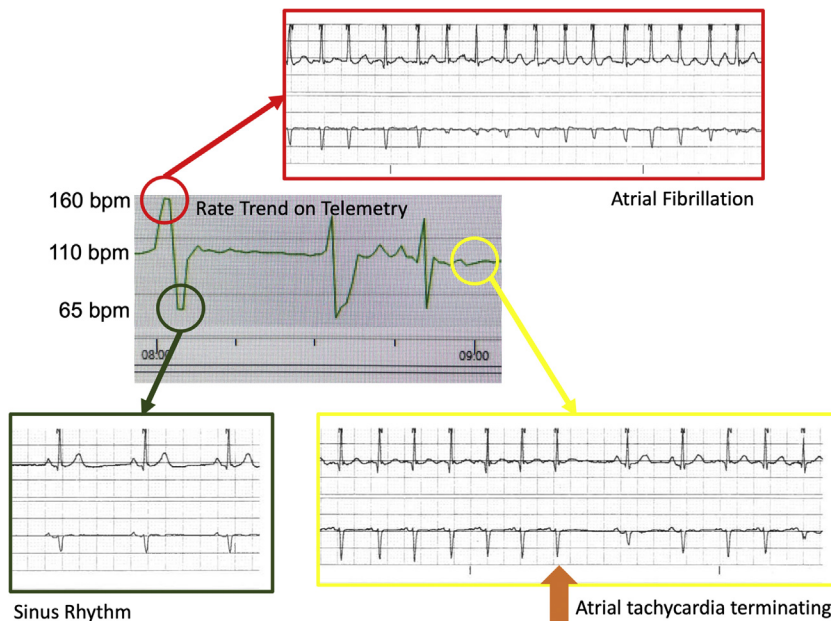


Figure 2 Telemetry demonstrating various atrial dysrhythmias. As demonstrated on this representative sample of telemetry strips, our patient was experiencing predominantly atrial tachycardia (*yellow*) with bouts of atrial fibrillation (*red*) and brief periods of normal sinus rhythm (*green*). Note that the atrial tachycardia example briefly terminates after a QRS complex (*orange arrow*) briefly to normal sinus rhythm.

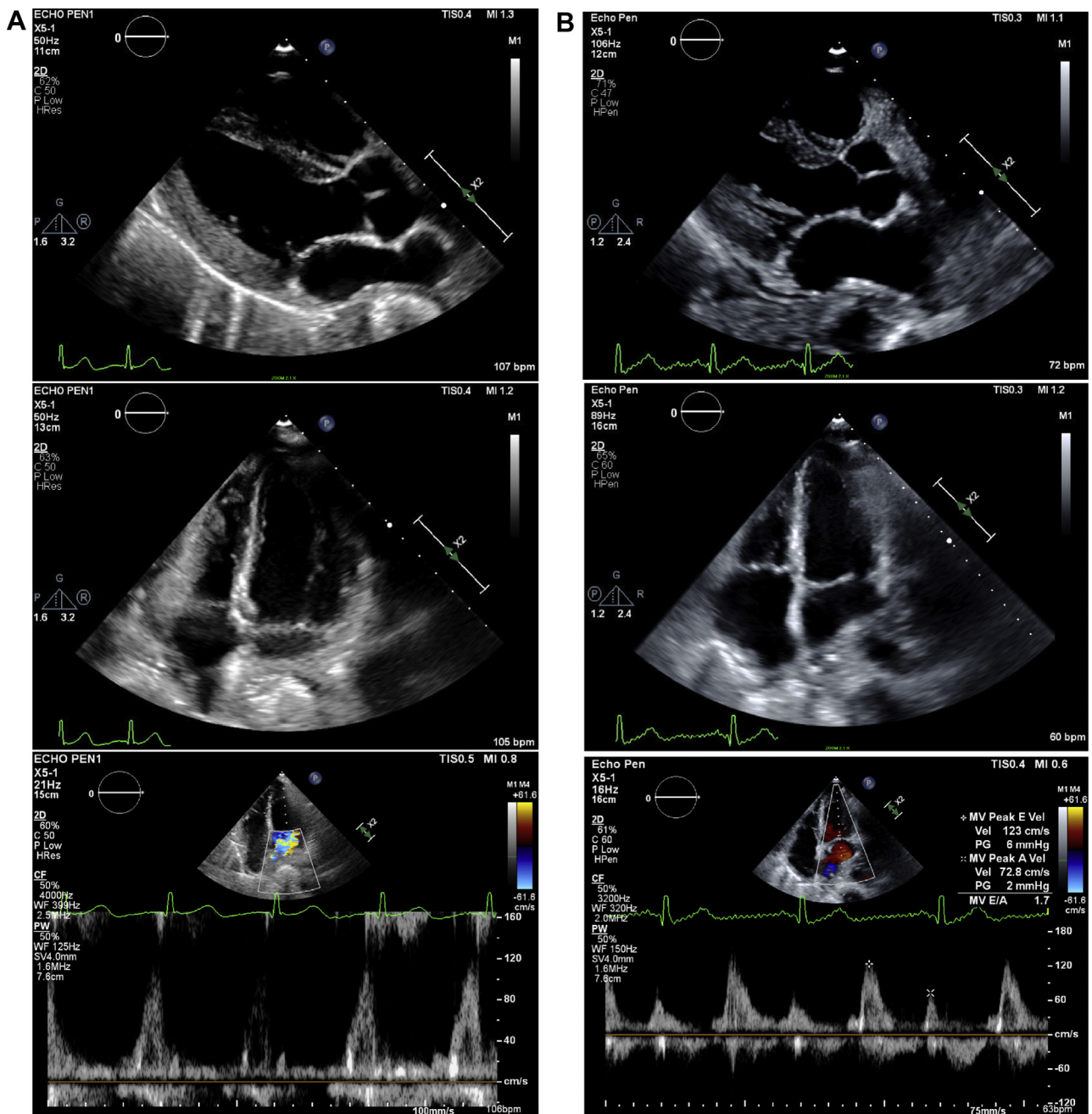


Figure 3 Echocardiographic demonstration of left atrial compression. **A:** Transthoracic echocardiogram obtained on admission showing extrinsic compression of the posterior left atrium in parasternal long and apical 4-chamber views. Notably there is a lack of E-wave on mitral inflow pulse wave Doppler examination. **B:** After esophagectomy, left atrial end-diastolic dimensions are more favorable with a return of E wave.

administered, resulting in transient atrioventricular block revealing 8 regular P waves, which were isoelectric in aVL and positive in V₁, suggesting a left-atrial focus at a rate of 125 beats per minute. This was followed by conversion to sinus rhythm (Supplemental Figure 1). The P-wave morphology during atrial tachycardia is positive in V₁ with a duration less than 80 ms, with an amplitude in lead I less than 50 μ V, and an amplitude in lead II of less than 100 μ V, all suggestive of a non-pulmonary vein left atrial focus. Review of 12-lead ECGs at various points during the initial workup demonstrated both sinus tachycardia, normal sinus rhythm, and atrial

fibrillation with rapid ventricular response. Detailed review of continuous telemetry monitoring revealed evidence of the above rhythms as well as suggestion of atrial tachycardia (Figure 2), which was not captured on 12-lead ECG other than the adenosine strip described above and in Supplemental Figure 1.

A transthoracic echocardiogram obtained the morning after presentation was remarkable for extrinsic compression of the left atrium by the distal portion of the esophageal stent (Figure 3), also evident on computed tomography of the chest as shown in Figure 1. Compared to computed tomography of

the chest following the first stent placement, the second stent increased the external diameter of the distal esophagus by approximately 15–20 mm at the level of the left atrium, resulting in a subtle increase in left atrial compression. Given the largely noncontributory laboratory workup above and the temporal association of chest discomfort worsened by the second stent placement, we theorized epicardial atrial irritation from the stented mid and distal esophagus as well as inflammation generated by the esophageal tumor to be the primary driver for his multiple atrial dysrhythmias.

Treatment was initiated with oral metoprolol tartrate 12.5 mg every 6 hours, which was gradually titrated to 50 mg every 6 hours over the ensuing 10 days with improvement in the frequency and duration of atrial ectopy. Owing to ongoing pain and concerns regarding the esophagobronchial fistula, he underwent salvage esophagectomy (including removal of the stents) on hospital day 20. After surgery, beta-blocker therapy was no longer required and there was no recurrence of atrial dysrhythmias in the immediate postoperative period. Repeat echocardiogram after esophagectomy revealed improvement in left atrial volume (Figure 3B). Additionally, cardiac output based on echocardiogram increased from 2.69 to 3.41 L/min/m² after stent removal. Unfortunately, disruption of the left main bronchus repair led to pneumothorax and persistent air leak without available surgical intervention. The patient was transitioned to comfort care on hospital day 24 and cardiac monitoring was discontinued.

Discussion

Esophageal stents can provide palliation of dysphagia and in certain instances close fistulae affecting the esophagus and adjacent mediastinal structures.⁸ Iatrogenic induction of supraventricular arrhythmia by esophageal stenting can lead to significant unintended morbidity but is rarely considered, nor counseled, given its rarity. Compression of the left atrium after esophageal stent deployment was noted in only a single case cited in a series of 30 patients (3.3%),⁹ and a pooled cohort study found only 1 case in 599 patients (0.2%).¹⁰ Although many complex neurohormonal pathways have been suggested to explain the phenomenon of dysrhythmia in association with left atrial compression, we believe that the resolution of atrial arrhythmias after removal of the source of extrinsic compression strongly supports the hypothesis that direct epicardial disruption is a pathophysiologic driver. A study by Ong and colleagues¹¹ demonstrated iatrogenic irritation of the epicardium after epicardial implantable cardioverter-defibrillator implantation is associated with increased risk of atrial fibrillation compared with transvenous approaches. Lastly, atrial stretching caused by direct compression of cardiac myocytes may also contribute to atrial arrhythmia. Atrial dilation and stretch are commonly associated with atrial fibrillation. Several animal studies have demonstrated electrophysiologic changes associated with atrial stretching that predispose to, or directly trigger, the genesis of atrial arrhythmias.^{12,13}

The left atrium is a vulnerable structure to extrinsic compression, given its proximity to other structures in the mediastinum, gastrointestinal tract, pericardium, lungs, and aorta. Severe compression can lead to cardiac insufficiency, but mild-to-moderate compression can increase morbidity through precipitation of atrial arrhythmias. Mediastinal pathology with proximity to the heart should be included in the differential diagnosis when evaluating new-onset atrial arrhythmia, particularly if multiple underlying rhythms are newly detected. Although the indications for surgical manipulation and device implantation of these adjacent structures are often compelling, it should warrant consideration of the proximity to the left atrium and the potential to trigger unintended arrhythmogenesis.

Conclusion

Extrinsic left atrial compression has previously been suspected to be a cause of atrial fibrillation. Attribution of a precise pathophysiologic mechanism to a single clinical scenario is often elusive, but the resolution of atrial arrhythmias after removal of the source of extrinsic compression in this case appears compelling.

Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2021.01.006>.

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