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## Case Report

# Atrial flutter ablation in a case of diuretic resistant constrictive pericarditis



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## ABSTRACT

We present a 66 year old gentleman with constrictive pericarditis and persistent atrial flutter. Initial management with oral loop diuretics was successful until he developed persistent atrial flutter. Once in atrial flutter the patient developed progressive signs of right heart failure resistant to high dose intravenous loop diuretics. He was referred to a tertiary electrophysiology service where he underwent successful isthmus catheter ablation and reverted to sinus rhythm. His responsiveness to diuretics improved immediately. His symptoms improved and he was discharged 48 h later on oral diuretics. He remains well one month after discharge.

This is the first reported case of symptomatic improvement in a patient with constrictive pericarditis and persistent atrial flutter with targeted treatment of the dysrhythmia. This offers a possible short-term palliation option in a group of patients where definitive surgical management carries too high a risk.

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## 1. Background

Constrictive pericarditis is a fibrous thickening of the pericardial sac leading to chronic compression of the heart and impaired diastolic function. It is a rare condition believed to occur in 1 in 10,000 people in the western world.<sup>1</sup> The incidence of the disease is linked to a variety of aetiologies seen in different parts of the world. In the western world, it is commonly idiopathic whilst tuberculosis is the most common cause in the developing world.

Definitive treatment is pericardial surgery which is associated with a high mortality and morbidity. In idiopathic disease the mainstay of medical treatment is symptomatic, treating fluid overload with oral loop diuretics.

Concomitant dysrhythmias are a recognized finding, with atrial fibrillation occurring in one third of cases and atrial flutter in up to 5% of patients.<sup>2</sup> There is little in the literature regarding the effectiveness of treating the dysrhythmia in improving patient's symptoms.

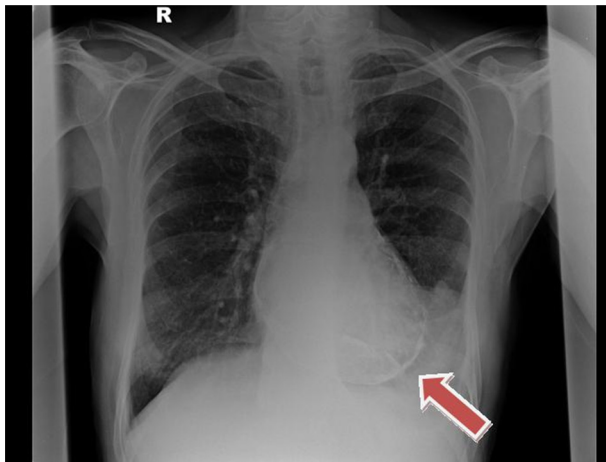
We present the first reported case of short-term palliation treatment of atrial flutter with ablation resulting in

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**Fig. 1 – Pericardial calcification (red arrow) seen on chest radiography.**

symptomatic relief in a patient who had developed diuretic resistant constrictive pericarditis.

## 2. Case presentation

A 66 year old gentleman presented to a vascular surgery clinic with an episode of cellulitis. He was noted to have marked bilateral peripheral edema up to the scrotum. In view of unexplained bilateral peripheral edema, he underwent an X-ray (Fig. 1) and then a CT scan of his thorax and abdomen (Fig. 2).

This identified calcified and thickened pericardium which the surgeons suspected constrictive pericarditis and urgently referred him to our cardiology outpatients clinic.

An echocardiogram showed a normal sized left ventricular with preserved systolic function along with mild bi-atrial dilatation. The inferior vena cava was dilated and did not collapse on inspiration. Initial management was with loop

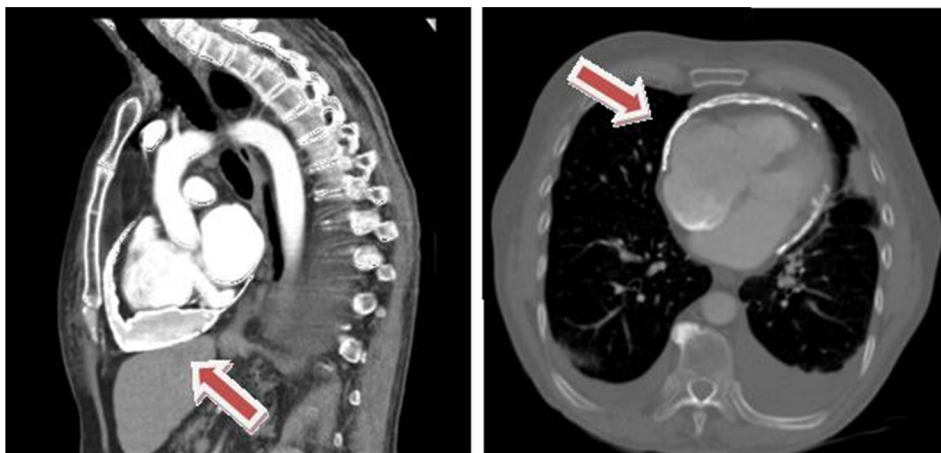
diuretics (*furosemide 40 mg od*). This was uptitrated (*furosemide 80 mg od*) with a good response and the patient remained symptom free for a period of three years.

Later on routine follow up he was identified to have an irregular heart beat with a rate of 110 bpm. Electrocardiography at the time showed sinus rhythm. He was fitted with a 7 day event recorder to rule out paroxysmal atrial fibrillation. The analysis revealed short runs of irregular tachycardia consistent with atrial flutter. He was referred for outpatient oral anticoagulation with warfarin in view of a significant risk of thrombo-embolic stroke.

The following year, he had developed increased peripheral edema with a raised JVP. Electrocardiography at this time identified typical atrial flutter with 2:1 block (Fig. 3). He was started on spironolactone (*25 mg od*) to aid the diuresis and bisoprolol (*5 mg od*) for rate control. This treatment could not control the ventricular rate and despite the addition of digoxin he developed worsening right sided congestive heart failure and presented to emergency department within the period of a month.

He was admitted urgently and commenced on high dose intravenous diuretics (*240 mg/24 h uptitrated to a maximum of 480 mg/24 h*) with adjunctive metolozone 2.5 mg. He was considered and referred for ultrafiltration at a tertiary centre as he was found resistant to diuretic therapy. Meanwhile, given the suspicion that the atrial flutter was likely to be contributing to his decompensation of congestive cardiac failure and with limited responsiveness to high dose diuretics and medical control of his heart rate, Electrophysiologist opinion was sought. Since his ECG (Fig. 3) suggested typical atrial flutter the Electrophysiologist suggested conversion of dysarrhythmia into stable sinus rhythm by radiofrequency ablation. The atrial arrhythmia was indeed confirmed to be typical atrial flutter and cavo-tricuspid isthmus ablation resulted in stable sinus rhythm (Fig. 4).

His responsiveness to diuretics improved immediately after the procedure and he was switched to oral diuretics within 48 h. He was discharged after a couple of days, to be followed by our community heart failure nurse specialists and with arrangements for review in our clinic.



**Fig. 2 – Pericardial calcification (red arrow) seen on CT Thorax.**

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