

Persistent Atrial Standstill in Acute Myocarditis

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Background: Atrial standstill manifests as absence of any atrial electrical activity in the surface ECG leads. Persistent atrial standstill secondary to acute myocarditis is extremely rare. **Case report:** 10-year-old girl had atrial standstill and heart failure due to acute myocarditis. After recovery from myocarditis, heart failure resolved, but the atrial standstill persisted. **Outcome:** Persistent atrial standstill was treated with permanent pacemaker and anticoagulation. **Message:** Acute myocarditis may rarely cause atrial standstill that can last even after recovery from myocarditis.

Keywords: Cardiac arrhythmias, Permanent pacemaker.

Atrial standstill or paralysis has been defined as the absence of any electrical or mechanical activity of atria [1-3]. In atrial standstill, there is no evidence of atrial electrical activity in the surface ECG leads or on electrophysiological evaluation. We report atrial standstill in acute myocarditis that persisted even after the resolution of myocarditis.

CASE REPORT

A 10-year-old girl presented with congestive heart failure (CHF) following an episode of low grade fever. She had no family history of cardiac illness, pacemaker implantation, embolic events or skeletal muscle disease. Investigations revealed a normal total leukocyte count ($7 \times 10^9/L$) with 18% neutrophils. Serum electrolytes were in the normal range. The ECG at the time of presentation showed absent P waves and a wide QRS regular escape rhythm at 40/min (**Fig. 1**). Cardiac troponin T was raised (1793 ng/L). Echocardiogram showed hypokinesia of the left ventricle and mild left ventricular (LV) dysfunction. Her Anti-ds DNA and anti-nuclear antibody titres were normal. She had received intravenous immunoglobulins, ampicillin and ranitidine before she was referred to our unit. We continued conservative management with bed rest, diuretics, and digoxin. Her CHF improved, and subsequently she was discharged. At review visit 6 weeks later, she had no CHF but had the same ventricular escape rhythm and exertional fatigue. Echocardiogram showed improved LV function (ejection fraction 51%). Mitral and tricuspid inflow Doppler study showed absence of “a” wave

suggestive of total atrial standstill (**Fig. 2**). An evaluation done for neuromuscular disease showed normal muscle strength and normal electromyography.

Electrophysiology study (EPS) showed no evidence of atrial electrical activity. There was no atrial capture even at maximum pacing output (25mA), from multiple atrial sites (**Web Fig. 1**), consistent with atrial standstill. She underwent a pacemaker implantation, and subsequently the right ventricular pacing parameters were normal. Her ECG after 10 months showed no p waves, and the fluoroscopy and echocardiography showed no suggestion of atrial contraction. Interrogation of the pacemaker confirmed that she was totally pacemaker-dependent.

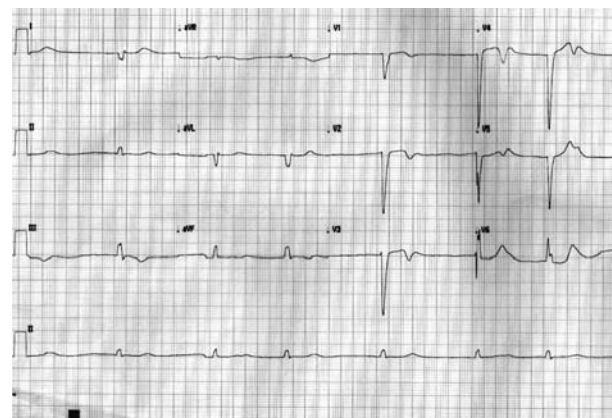


Fig. 1 The 12 Lead ECG showing idioventricular rhythm with absence of P wave and wide QRS escape rhythm.

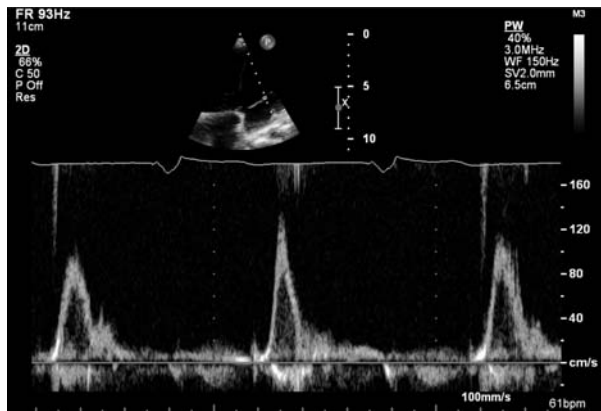


Fig. 2 Mitral inflow pulse wave Doppler showing absence of 'a' wave and paced rhythm at 60/minute.

DISCUSSION

Atrial standstill has been reported to occur in inherited myopathies, valvular cardiomyopathies, digitalis or quinidine intoxication, hypoxia, hyperkalemia, myocardial infarction, systemic lupus erythematosus, and Chaga's disease. Atrial standstill secondary to myocarditis, persisting long after the acute phase, is extremely rare.

Atrial standstill could be defined as partial or total [4]. In partial atrial standstill, conduction disturbance within the right atrium alone is a more common finding. The absence of P waves in surface ECG occur in sinus node arrest with junctional escape rhythm which is thus a differential diagnosis of atrial standstill. However, in sinus arrest, the atria can be shown to be excitable by pacing whereas in atrial standstill, atria are non-excitabile and cannot be captured even with high pacing outputs. This results in lack of mechanical functioning of atria, and predispose to intra-atrial thrombus formation.

Our patient had a wide QRS escape rhythm with no P waves. This indicates an escape rhythm from below the level of His bundle (Infra-Hisian). The inability to capture the atria even at high current output during the EPS confirmed atrial standstill in our case. We used digoxin for LV dysfunction in spite of patient having bradycardia because digoxin has minimal action on infra-Hisian tissues, and is less likely to suppress the escape rhythm. However, a stand by temporary pacemaker was kept ready, and patient was closely monitored. Isoprenaline, which can be used to increase the heart rate, can also worsen the conduction in infra-Hisian disease, and hence was not used. She was closely monitored in the acute phase, and as there was a possibility that her rhythm may normalize once her myocarditis resolves, we waited for six weeks before implanting a permanent pacemaker.

Persistent atrial standstill is a rare disorder [5], and that occurring after myocarditis is even rarer. Straumanis, *et al.* [6] have reported a 11-year-old child with biopsy-proven necrotizing acute myocarditis causing atrial standstill. However, this was transient and resolved after 3 days of treatment with methylprednisolone. Our patient had persistence of atrial standstill, even after the ventricular dysfunction recovered. Abdelwahab, *et al.* [7] have reported a case of multiple atrial arrhythmias (atrioventricular node re-entry and two different focal atrial tachycardias) originating from the remaining atrial myocardium after global scarring of both atria following a remote viral myocarditis. Talwar, *et al.* [8] reported a case series in which two of the patients had lymphocytic infiltrates on right ventricular endomyocardial biopsy. In another case series of 11 patients with atrial standstill reported by Nakazato, *et al.* [9], three had histological evidence of chronic myocarditis. None of their patients had a presentation suggestive of acute myocarditis.

Pathological involvement of atria in atrial standstill can be localised or diffuse. Our patient likely had a total atrial standstill as evidenced in mitral pulse wave Doppler showing absence of 'a' wave. As atrial standstill is a well-known cause of cardiogenic embolism [9], anticoagulation is mandatory; our patient was started on Warfarin. The management of patients with atrial standstill include anticoagulation in all patients, and pacemaker implantation in patients who have symptomatic bradycardia due to insufficient rates of escape rhythm.

We conclude that acute myocarditis can rarely cause extensive damage to the atria causing loss of electrical and mechanical function. This may herald atrial standstill, which can persist even beyond the acute phase when other changes have recovered.

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REFERENCES

1. Surawicz B. Electrolytes and the electrocardiogram. *Am J Cardiol.* 1963;12:656.
2. James TN. Myocardial infarction and atrial arrhythmias. *Circulation.* 1961; 24:761.
3. James TN, Rupe CE, Monte R. Pathology of the cardiac conduction system in systemic lupus erythematosus. *Ann Intern Med.* 1965;63:402.
4. Lévy S, Pouget B, Bemurat M, Lacaze JC, Clementy J, Bricaud H. Partial atrial electrical standstill: report of three cases and review of clinical and electrophysiological features. *Eur Heart J.* 1980;1:107-16.
5. Allensworth DC, Rice CI, Lowe G. Persistent atrial

- standstill in a family with myocardial disease. *Med Am J.* 1969;47:775-84.
6. Straumanis, John PW, Henry BC, Christopher LR. Resolution of atrial standstill in a child with myocarditis. *PACE.* 1993;16:2196-202.
 7. Amir A, John LS, Ratika P, Magdy B, Martin G. Mapping and ablation of multiple atrial arrhythmias in a patient with persistent atrial standstill after remote viral myocarditis. *PACE.* 2009;32:275-7.
 8. Talwar KK, Dev V, Chopra P, Dave TH, Radhakrishnan S. Persistent atrial standstill — clinical, electrophysiological and morphological study. *PACE.* 1991;14:1274-81.
 9. Yuji N, Yasuro N, Teruhikoa H, Masataka S, Shunsuke O, Hiroshi Y. Clinical and electrophysiological characteristics of atrial standstill. *PACE.* 1995;18:1244-54.
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