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Brief Report

Late high degree atrioventricular block after percutaneous closure of a perimembranous ventricular septal defect

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Abstract Atrioventricular block is an important complication after percutaneous closure of perimembranous ventricular septal defects. In the majority of cases, it appears in early or in the midterm of closure. We present a patient who developed symptomatic Mobitz 2 second-degree atrioventricular block 3 years following percutaneous closure of such a defect.

Keywords: Conduction delay; cardiac catheterization; Amplatzer septal occluder

Percutaneous closure of perimembranous ventricular septal defect has now become a popular, albeit controversial, alternative to surgical treatment.[1] This method is associated with reduced procedural morbidity compared with the surgical approach, but bears its own complications,[1–3] one of which is atrioventricular block. This phenomenon has been reported as an early or midterm event following the procedure.[4]

Case report

A 30-year old woman was diagnosed with a perimembranous ventricular septal defect. There were no signs of cardiac failure, and her 12-lead electrocardiographic tracing showed sinus rhythm with incomplete right bundle branch block and left anterior hemiblock, so-called bifascicular block, with a QRS duration of 61 milliseconds. Transthoracic echocardiography revealed mild left ventricular enlargement with end diastolic dimensions of 58 millimetres, the normal ranges for the adult being 42 to 54 millimetres, and normal left ventricular systolic function.

Cardiac catheterization revealed normal pulmonary arterial pressures, with a left-to-right shunt of 1.8 to 1 at the ventricular level. Left ventricular angiography and transesophageal echocardiography showed a perimembranous ventricular septal defect of 10 millimetres. The defect was successfully closed with the 10 mm Amplatzer ventricular septal occluder manufactured by the AGA Medical Corporation, Minnesota, United States of America. The procedure was uneventful and the patient was discharged home the next day.

The patient remained asymptomatic at intervals of 10 days, 1 month, 6 months, 12 months, 24 month, and 36 months. Echocardiographic imaging showed a good position of the occlusive device, with no residual shunting and without aortic valvar regurgitation. The consecutive postprocedural electrocardiographic tracings showed a pattern of bifascicular block, but with gradual widening of the QRS complex. The interval was 104 milliseconds at 1 month following the procedure, 116 msec at six months, and 124 msec at 1 year following the procedure, without prolongation of the PR interval. Monitoring using 24 hour ambulatory electrocardiographic tracings performed at six months, one year, and two years following closure disclosed no

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significant arrhythmia or delay in atrioventricular conduction.

After 38 months following closure, the patient was referred for weakness and dizziness, which had appeared several days earlier. An electrocardiographic study now revealed 2 to 1 second degree atrioventricular block, of Mobitz type 2, with a ventricular rate of 45 beats per minute. The patient was hospitalized and medication commenced with oral prednisone at 1 mg/kg/day. On the third day of hospitalization, the second degree atrioventricular block disappeared. The patient was discharged in sinus rhythm, with an unchanged QRS pattern. After discharge, the patient was gradually weaned off the prednisone, and remained asymptomatic.

Continuing 24 hour ambulatory electrocardiographic studies performed 1, 3, and 6 months following discharge were essentially normal, but transient, short-lasting 2 to 1 daytime asymptomatic Mobitz 2 atrioventricular block was recorded 9 months following hospitalization. After an additional interval of 2 months, the patient was referred with similar complaints of dizziness and fatigue, and again we recorded 2 to 1 atrioventricular block at 45 ventricular beats per minute. The patient was readmitted, and a DDDR type permanent cardiac pacemaker was implanted.

Several steps were taken to investigate the cause of this late atrioventricular block. Chest fluoroscopy did not reveal any changes in the form or size of the device, which might have caused mechanical injury to the conduction system. Inflammatory immune response due to nickel allergy was another proposed mechanism, but no clinical or laboratory findings of systemic inflammatory or immune response were detected. A nickel skin patch test was negative, and we failed to detect elevated levels of nickel in the urine.

Discussion

Transcatheter closure of perimembranous ventricular septal defects is associated with excellent rates of closure, no mortality, and low morbidity.[1] Potential complications of this procedure are related to the proximity of the defect to the surrounding structures, such as the aortic and tricuspid valves, the coronary arteries, and the atrioventricular conduction system.[5,6]

Complete atrioventricular block is a well-known complication of closure, being reported in up to one-twentieth of cases.[4] In the majority of cases, it appears in the early or intermediate post-procedural period.[1–4] The proposed mechanisms are mechanical compression of the conducting system and local inflammatory reaction.[5–7] Empirical therapy with non-steroidal anti-inflammatory drugs or corticosteroids is reported to be effective when the arrhythmia occurs in the early post-procedural period.[7]

Our case of high degree atrioventricular block has some unique features. It appeared more than three years following the procedure, the longest period of time ever described. In spite of the late presentation, steroid therapy was effective, and the patient remained asymptomatic for 11 more months. But this improvement was temporary, and the patient had recurrent atrioventricular block, which necessitated the implantation of a pacemaker. The precise mechanism of the atrioventricular block in our patient is unclear. The late development of atrioventricular block, the flat initial appearance of the occlusive device, and the lack of further deformity on comparative fluoroscopy, do not support the theory of mechanical compression.[6] Gradual widening of the QRS complex detected during follow-up might have been a hint reflecting its slow development. The late response to corticosteroid therapy may have been a reflection of an ongoing inflammatory process. In view of recent publications suggesting that nickel allergy may be a trigger of an inflammatory response,[8] we tested for this complication, but no signs of nickel allergy were noted. Empirical corticosteroid therapy was effective in spite of the late presentation, but the improvement was temporary, probably due to progressive ongoing inflammatory process.

Our experience shows that high degree atrioventricular block may appear several years following transcatheter closure of perimembranous ventricular septal defects. It implies that long-term follow-up, perhaps life-long, may be mandatory. Further clinical, immunological and pathological studies are needed to clarify the nature of this complication.

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