Late appearance of traumatic ventricular septal defect

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A ventricular septal defect with delayed appearance is described following stab wound of the heart in a woman. The diagnosis was confirmed by left ventricular angiography. Damage to the left anterior descending coronary artery, documented by selective coronary arteriography, might have played an important role in late development of the shunt. The septal defect was closed successfully by a patch using extracorporeal circulation.

Penetrating wounds of the heart may be complicated on rare occasions by creation of ventricular septal defects (Cary, Hurst, and Arentzen, 1958; Williams, Hara, and Bulloch, 1966; Wilson and Bassett, 1966). Delayed appearance of a typical murmur has been described incidentally (Cary *et al.*, 1958; Lui, Glas, and Bercu, 1965; Miller, Crockett, and Potter, 1962). A further case with late onset of symptoms and physical signs of a ventricular septal defect following stab wound of the heart and angiographic proof of additional involvement of the anterior descending branch of the left coronary artery is presented.

Case report

A 47-year-old woman was admitted unconscious and in profound shock 25 minutes after having been stabbed in the chest with a butcher's knife. There was a 2 cm long linear laceration in the fourth intercostal space 4 cm to the left of the sternum. As cardiac tamponade and left haemothorax were obvious from clinical signs and chest x-ray, emergency thoracotomy was performed. A wound, 1.5 cm in length, situated in the apical third of the right ventricle, was closed with three atraumatic sutures. The left anterior descending coronary artery had been severed and was bleeding freely. It was ligated.

The patient's postoperative course was uneventful except for transient pericarditis and a localized myocardial infarction demonstrated by the electrocardiogram (Fig. 1). A cardiac murmur was not heard at discharge 7 weeks after operation, or at follow-up examination 6 and 9 months later.

It was not until 2 years after the assault that a heart murmur was first heard. The patient then complained of shortness of breath and left-sided chest pain upon strenuous exercise. As symptoms deteriorated review of cardiac status was advised.

On admission to the section of cardiology, University of Vienna, in May 1972, 6 years after trauma, physical

examination showed a dyspnoeic, obese patient with peripheral cyanosis but without oedema. At the left sternal edge there was a systolic thrill and a harsh, grade 4/6 holosystolic murmur with late systolic accentuation on the phonocardiogram suggesting ventricular septal defect or mitral incompetence (Fig. 1). The electrocardiogram and vectorcardiogram indicated incomplete right bundle-branch block and slight biventricular hypertrophy; T waves had become abnormal. Chest x-ray showed moderate biventricular enlargement of the heart, normal hilar vessels, and absence of left atrial dilatation.

Cardiac catheterization revealed normal pressures in both ventricles and in the pulmonary artery and a left-to-right shunt of 1:1:1 at ventricular level. Left ventricular angiograms did not show regurgitation into the left atrium but there was rapid opacification of the right ventricle and pulmonary vessels. Selective coronary arteriography showed a 1.5 cm long interruption in the distal third of the left anterior descending artery with good opacification of the distal segment via collaterals (Fig. 2). Despite the rather unimpressive haemodynamic findings, closure of the septal defect was recommended because of the disabling symptoms. At operation, under cardiopulmonary bypass, an oval-shaped defect 4 × 7 mm in diameter was seen in the apical third of the interventricular septum corresponding to the site of coronary artery damage and was closed with a 'teflon' patch.

Postoperative recovery was essentially uncomplicated; the murmur completely disappeared. Serial electrocardiograms showed development of complete right bundle-branch block. Two months after operation the patient had substantial relief of symptoms and increased working capacity.

Discussion

Ventricular septal defects secondary to penetrating or blunt injuries of the heart are uncommon. Wilson and Bassett (1966) reported only one case of resultant interventricular septal defect in a series of 200 patients with penetrating heart injuries. On the

FIG. 1 Electrocardiograms on the second and twenty-first days after trauma, indicating peri-carditis and circumscribed anterior myocardial infarction. Electrocardiogram and phonocardiogram 6 years after trauma.

FIG. 2 Left coronary arteriogram showing interruption of the anterior descending branch (right anterior oblique position).

other hand, Samson (1948) stated that all patients with perforation of the septum observed by him had died before operation could be performed. In some instances, however, patients with these defects have survived even without surgical intervention for many years (Derra, 1959).

The onset of clinical signs suggesting a shunt may be delayed for some days up to several weeks making repeated examinations after trauma absolutely necessary (Cary *et al.*, 1958; Miller *et al.*, 1962; Lui *et al.*, 1965). The cause of late appearance of a typical systolic murmur remained obscure in most cases. Feruglio, Bayley, and Greenwood (1960) believed additional damage of a coronary artery to cause late perforation of the septum. Summerall, Lee, and Boone (1965) thought that some defects, especially when subjected to high pressure gradients, enlarged after the initial injury. In the case described by Russe *et al.* (1968), who resembled our patient in many respects, the exact mechanism of late development of a septal defect was ascertained by necropsy: this revealed a non-perforating laceration of the interventricular septum congruent with the damaging knife and surrounded by scar tissue secondary to the intersection of the perforating septal branches of the left anterior descending coronary artery; the thinned and partially necrotic area of the septum had been ruptured by the high left ventricular pressure, thus leading to an interventricular septal defect 4 weeks after trauma. In our patient all the evidence for this genesis of the interventricular septal defect is provided: the knife entering the right ventricle and damaging the left anterior descending coronary artery possibly also injured the underlying ventricular septum. Serial electrocardiograms were consistent with circumscribed myocardial infarction. Assuming that the loud and harsh murmur was not overlooked during 7 weeks in hospital and on several follow-up examinations, the defect probably originated between 9 months and 2 years after assault.

Thus, appearance of the interventricular septal defect in our patient is likely to have been caused by direct laceration as well as by secondary damage of the septum after intersection of the left anterior descending coronary artery. Injuries to coronary vessels are a rare sequel of penetrating heart traumas, leading to myocardial infarction in few instances (Wilson and Bassett, 1966; Maynard,

Cordice, and Naclerio, 1952) and coronary arteriovenous fistulae in some exceptional cases (Tsagaris and Bustamante, 1966; Siepser *et al.*, 1972). Rapid and ample development of collaterals cannot always prevent myocardial infarction (Siepser *et al.*, 1972) but might be responsible for the normal electrocardiogram seen in our case.

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