Post-infarction ventriculer septal defect – a case report

Pozawałowe pęknięcie przegrody międzykomorowej – opis przypadku

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Abstract

Post-infarction ventricular septal defects (VSD) are rare (1-2%) but often fatal complications of acute myocardial infarction. These post infarction defects require urgent surgical treatment. We report a case unique in being a late presentation of post MI multiple VSDs. The patient survived surgery and a stormy post repair course with an excellent final outcome.

Key words: ventricular septal defect, Dor procedure

Kardiol Pol 2008; 66: 551-554

Case report

A 42-year old male presented with a 2-week history of chest pain with acute shortness of breath on the day of admission. His cardiac risk factors included hyperlipidaemia, hypertension, and smoking.

On examination he looked quite unwell and diaphoretic. His BP was 105/60 and was tachycardic with a pulse of 125. His JVP was distended with a prominent v wave. On cardiac auscultation, he had a grade 5 pansystolic murmur in the left lower parasternal area along with an LVS3. Chest auscultation revealed fine bibasilar crackles.

The ECG recorded one year before admission was normal (Figure 1A). The ECG on admission showed a right bundle branch block with ST elevation (Figure 1B), which persisted on serial ECGs, implying proximal left anterior descending artery occlusion (first septal perforator supplies the right bundle) and post MI aneurysm. His CXR had a cardiothoracic ratio of more than 50% with upper lobe diversion.

The bedside echocardiography study showed two ventricular septal defects – one in the apical septum and the other in the mid septum (Figures 2A and 2B). There was a large apical aneurysm consistent with anterior myocardial infarction. His ejection fraction was 25%. There were features of pulmonary artery hypertension suggested by a right ventricular systolic pressure of 65 mmHg, tricuspid regurgitation and right heart enlargement.

He was referred to a quaternary level hospital with cardiothoracic surgery facilities for urgent repair of his defects. He underwent coronary angiography, which revealed a totally occluded LAD. He was placed on intra-aortic balloon pump (IABP) counterpulsation and started on heparin and frusemide infusion. A week later he had his VSD repair via the Dor procedure using a bovine pericardial patch.

The postoperative period was stormy. He developed a residual VSD with a trivial leak to the aneurysm and went into cardiogenic shock requiring inotropic support, mechanical ventilation and IABP for 6 days.

Atrial fibrillation on the 3rd postoperative day reverted to sinus rhythm after amiodarone infusion. Multi-organ failure developed in the form of acute renal failure and ischaemic hepatitis which settled on the 8th postoperative day with conservative management.

A repeat transthoracic echocardiogram showed an improved ejection fraction from 25 to 48%, normal right ventricular size and a small residual shunt from left ventricle (LV) to right ventricle via apical VSD (Figure 2C and 2D). He received decongestive treatment in the form of an angiotensin-converting enzyme inhibitor and beta-blocker and was discharged on the 15th postoperative day.

He has been reviewed with regular two-monthly follow-ups and his residual leak has been repaired since.

Discussion

In 1845 Latham described a postinfarction ventricular septal rupture at autopsy, but it was not until 1923 that

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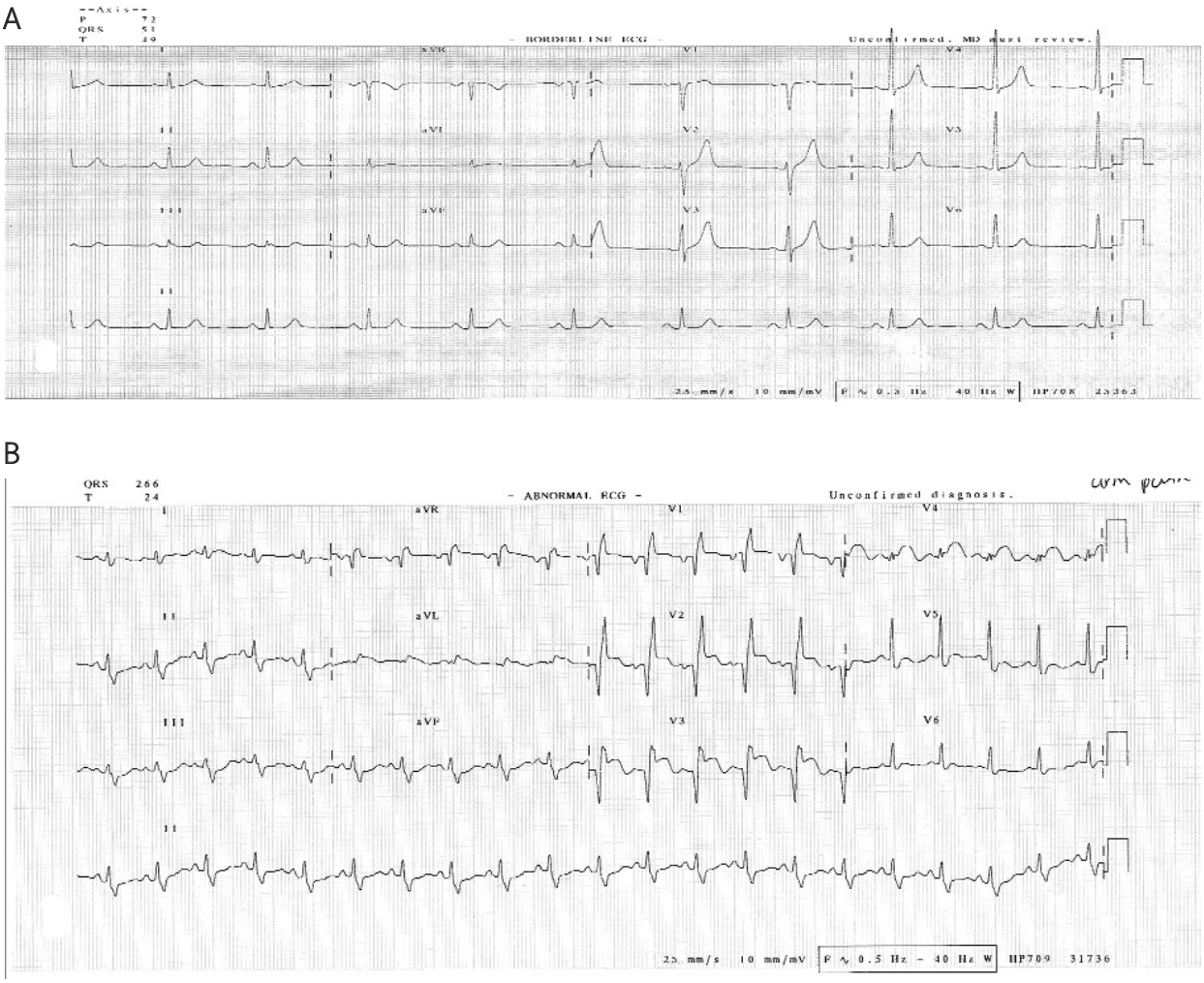

Figure 1. A. ECG 12 months before current presentation. **B.** ECG at presentation

Brunn made the first ante-mortem diagnosis. Sager in 1934 established specific clinical criteria for diagnosis, stressing the association of postinfarction septal rupture with coronary artery disease.

Ventricular septal defects rupture can occur from a few hours to a few weeks post MI but occurs most commonly by the end of the first week. It is usually followed by low cardiac output and multi-organ failure [1].

Reviews [2] reveal that nearly 25% of patients with postinfarction septal rupture and no surgical intervention died within the first 24 hours, 50% died within 1 week, and 80% died within 4 weeks; only 7% lived longer than one year. Of 25 patients with postinfarction ventricular septal defects treated medically, 19 died within one month [3]. Thus, the risk of death following postinfarction ventricular septal defect (VSD) is highest immediately after infarction and septal rupture, and then gradually declines. Interestingly, there are reports of spontaneous closure of small defects, though this is so rare that it would be unreasonable to manage a patient with the expectation of closure.

Recently, the SHOCK Trial (SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK) provided intriguing data on the outcome of medically managed patients with shock and postinfarction VSD. The multi-institutional study tracked 55 patients in cardiogenic shock from postinfarction VSD. Rupture occurred a median of 16 hours after infarction, and the median time to the onset of shock was 7.3 hours. Twenty-four patients were managed medically; the remaining 31 patients comprised a high-risk surgical group. There were only 7 survivors, of whom 6 had surgery to repair the defect.

Despite the many advances in the nonoperative treatment of congestive heart failure and cardiogenic shock, including the intra-aortic balloon pump and a multitude of new inotropic agents and vasodilators, these do not supplant the need for operative intervention in these critically ill patients [4].

The culprit artery in the setting of VSD is usually the LAD. Several reports have questioned the role of concomitant CABG along with VSD repair, there being no difference in early or late outcomes [2]. The tissue in the LAD territory becomes obliterated by the sutures that close the LV. Therefore nothing is gained by grafting the LAD. Commonly, after the 2nd or 3rd day of post MI VSD repair, the patch blows up and the shunt re-opens, as happened in our case too, in spite of using an exclusion technique. The reason is that although the surgeon stitches onto tissue that appears solid enough to hold the sutures at the time of operation, surgery is performed in the setting of an acute infarct. Over the next few days additional myocardium may become necrotic and friable as a result of developing inflammation at the infarct margin. This results in stitches pulling out of the septal tissue, allowing the shunt to re-open. The technical problems are largely different when the necrotic muscle is replaced by fibrous tissue [5].

Risk factors for mortality include preoperative and postoperative evolution of clinical status and right ventricular functions. Two patch repair has a better prognosis than 1 patch repair [5].

A shorter interval between perforation of the septum and surgical repair has been found to be an increased risk factor for mortality, however, it is ethically unacceptable to delay surgery in these brittle patients [5].

Management

It has become clear that the early practice of waiting for several weeks after ventricular septal rupture before proceeding with surgery only selects out the small minority of patients in whom the haemodynamic insult is less severe and is better tolerated [6]. Likewise, it has also become clear that to manage most patients supportively, in the hope of deferring operation, is to deprive the great majority of those with postinfarction ventricular septal rupture of the benefits of definitive surgery before irreversible damage due to peripheral organ ischaemia has occurred [7,8].

Rarely, because of a delayed referral, a patient who is already in a state of multisystem failure or who has developed septic complications will be seen for surgical therapy. Such a patient is unlikely to survive an emergency operation and thus may benefit from prolonged support with an intra-aortic balloon pump before an attempted operation [4].

Dor Procedure

It is LV reconstructive surgery. The surgeon adopts an infarct exclusion technique removing the dead/infarcted area of heart tissue and/or the aneurysm, thereby returning the LV to a more normal shape. The goal is to improve the heart failure and the angina and possibly improve the pumping ability of the heart.

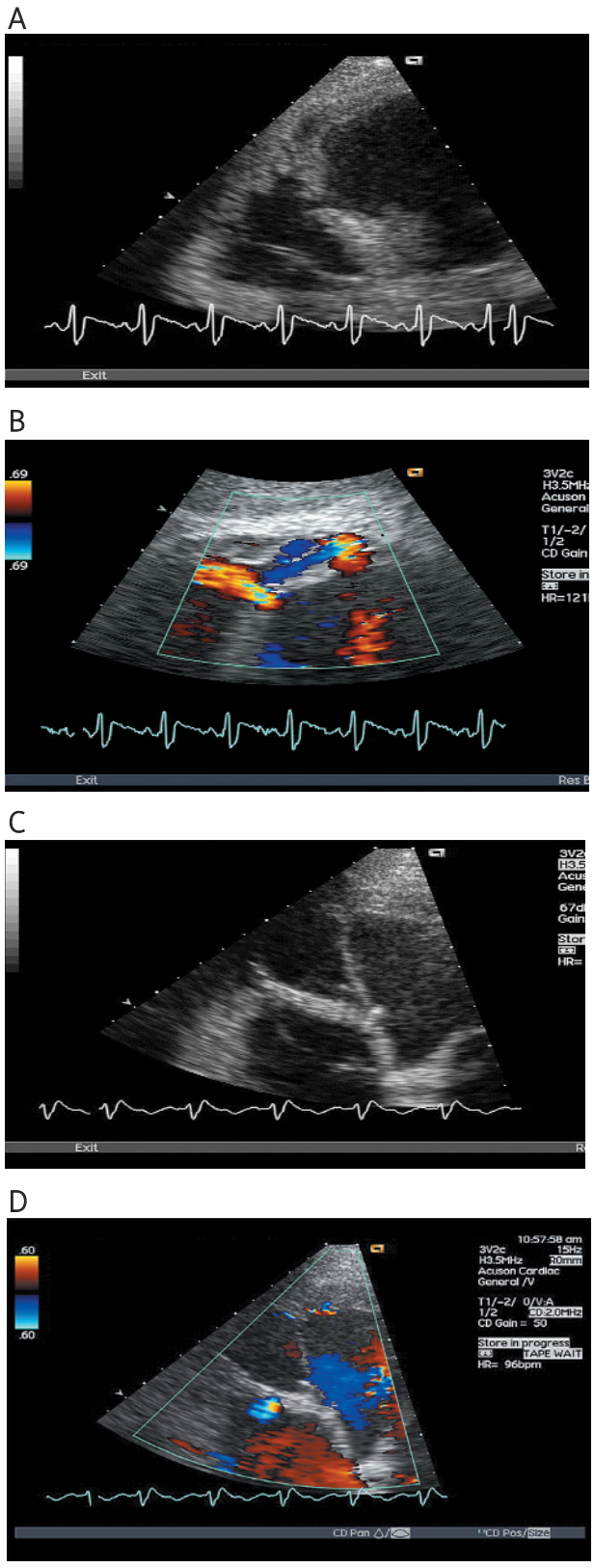

Figure 2. A. Mid septal and apical VSD. **B.** Left to right ventricle shunting across VSD. **C.** Apical aneurysm and VSD excluded with Dor procedure. **D.** No flow across mid septal VSD but a small residual leak in apical VSD

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Komentarz redakcyjny

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Operacyjne zamykanie pozawałowych ubytków przegrody międzykomorowej (VSD) wciąż jest jedną z najtrudniejszych operacji kardiochirurgicznych i – mimo postępów w kardiochirurgii i intensywnej terapii – jest związane z dużą śmiertelnością okołooperacyjną. O wynikach leczenia decyduje przede wszystkim wielkość obszaru lewej komory (LV) uszkodzonego w wyniku zamknięcia tętnicy dozawałowej. Tak więc stan kliniczny chorego przed zabiegiem i wielkość frakcji wyrzutowej LV są istotnymi czynnikami prognostycznymi. Jednakże na stan kliniczny wpływa bezpośrednio wielkość przecieku determinowana wielkością pozawałowego ubytku międzykomorowego. Tak więc możemy stwierdzić, iż chory z dużym ubytkiem i stosunkowo ograniczonym uszkodzeniem ściany przedniej [np. zamknięcie tętnicy przedniej zstępującej (LAD) po pierwszej tętnicy diagonalnej (D1)] będzie bardziej niewydolny przed zabiegiem niż chory z małym, tworzącym się dopiero ubytkiem i dużym uszkodzeniem LV, jednak wynik leczenia operacyjnego w tym pierwszym przypadku będzie lepszy.

Ponieważ mamy duże doświadczenie w chirurgicznej rekonstrukcji pozawałowo uszkodzonej LV, także u chorych z pozawałowymi przednimi ubytkami po zamknięciu LAD, w Śląskim Centrum Chorób Serca w Zabrzu wykonujemy operacje metodą wprowadzoną przez francuskiego kardiochirurga, prof. V. Dora. Pozwala ona nie tylko zamknąć ubytek, ale również wyeliminować zmienioną martwiczo tkankę ściany przedniej serca. Trzeba jednak pamiętać, że wczesna operacja, wykonana ze wskazań życiowych, wiąże się z dużo trudniejszą dla chirurga identyfikacją zasięgu zmian niedokrwiennych ściany przedniej i trudną decyzją o tym, jak duży obszar powinien być wykluczony poprzez wszycie łaty osierdziowej.

Gratulujemy Autorom pracy, jak zawsze gdy uda się nie tylko uratować chorego trudnego, chociaż typowego, ale także przywrócić dobrą kurczliwość serca po zabiegu. Na szczęście w Polsce i na świecie, w związku z upowszechnieniem przez kardiologów inwazyjnych mechanicznej reperfuzji w zawale serca, obserwujemy znacznie mniej mechanicznych powikłań zawału, w tym pozawałowego VSD. Jednocześnie cieszy nas – zarówno kardiochirurgów, jak i kardiologów – rosnące doświadczenie naszych zabrzańskich Kolegów w nieoperacyjnym zamykaniu pozawałowych ubytków międzykomorowych. Trzeba wspólnie podejmować trudne wyzwania u trudnych chorych – jest to prawda oczywista, jeżeli zależy nam na rozwoju nowoczesnej medycyny, wszystko jedno, czy dzieje się to w Australii, czy w Polsce.