and second obtuse marginal branches of the circumflex artery, the posterior descending artery and the posterior left ventricle branch (both arising from the right coronary artery) were bypassed sequentially with a single segment of reversed saphenous vein. Within 20 min of his return to the intensive care unit after the surgery, the patient developed ECG changes suggestive of anterior myocardial ischaemia, with ST-T elevation of 0.2 mV in leads I, aVL, V2, V3, V4 despite glyceryl trinitrate infusion (3 mg h⁻¹). An ultrasound examination carried out an hour after patient's arrival to ITU failed to show any flow in the ITA graft, either by pulsed Doppler or two-dimensional ultrasound with colour flow mapping. The inability to document flow in the ITA prompted coronary angiography which was performed 2 h later. Injection of the saphenous vein graft showed patency of all four anastomoses. Selective angiography showed a patent anastomosis of the ITA to the first diagonal branch but no flow beyond. The first injection of contrast medium (Niopam 370) was followed by another five which showed gradual clearing of the whole segment of the ITA between the first diagonal artery and the LAD. Following this procedure the ST-T segment changes normalized.

Four days later a repeat ultrasound study confirmed excellent flow in the ITA with a typical systolic and diastolic flow pattern (Fig. 1(b)). The diameter of the artery was 3 mm. Peak systolic velocity was 55.6 cm s⁻¹ and peak diastolic velocity 50.6 cm s⁻¹. A total flow of 59 ml min⁻¹ was calculated from time averaged velocity over the cardiac cycle and the cross-sectional area of the ITA.

The patient was discharged home 7 days postoperatively. Seven weeks later, a further ultrasound examination showed good ITA flow, with a diameter of 3 mm, a peak systolic velocity of 53.6 cm s⁻¹, a peak diastolic velocity of 342.8 cm s⁻¹ and a total flow of 50.4 ml min⁻¹.

Our case illustrates the potential usefulness of transcutaneous duplex ultrasound graft examination in the immediate postoperative period, provided that the vessel has previously been shown to be visible preoperatively. In the present case the combination of ECG evidence and failure to image graft flow constituted a powerful argument for emergency angiography.

Intravascular haemolysis in hypertrophic cardiomyopathy

Red cell fragmentation is a well documented complication of mechanical valvular prostheses, which has also been reported in unoperated patients with a variety of conditions such as severe aortic stenosis. Haemolysis has only occasionally been described in patients with hypertrophic cardiomyopathy (HCM)³⁻⁵. We report two cases with this uncommon complication and present the results of a retrospective study on the prevalence of haemolytic hallmarks in HCM patients admitted to our institution during the last 10 years.

References


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The first patient is a 60-year-old man in whom the diagnosis of HCM was made in 1986. Trans-thoracic echocardiography demonstrated asymmetrical hypertrophy of the septum and a systolic anterior motion (SAM) of the anterior mitral leaflet resulting in a left intraventricular pressure gradient (IVPG) of 65 mmHg. Contemporaneously, laboratory investigations disclosed haemolytic hallmarks [haptoglobin <20 mg . dl\(^{-1}\) (NL 80-250) — LDH 615 IU . \(1^{-1}\) (NL 100-340)] which, in the absence of other haemolytic disorders, were ascribed to traumatic red cell fragmentation secondary to HCM. The patient was treated with verapamil (320 mg . day\(^{-1}\)). Two years later, IVPG was 12 mmHg and laboratory studies revealed a concomitant improvement of haemolysis (haptoglobin 88 mg . dl\(^{-1}\) — LDH 360 IU . \(1^{-1}\)).

The second patient is a 56-year-old man in whom HCM was diagnosed in 1991 and treated with a combination of atenolol and nifedipine. He was admitted in September 1994 because of Streptococcus faecium septicaemia. At echocardiography, no vegetation was seen; IVPG was 100 mmHg associated with a SAM of the anterior mitral leaflet and a moderate mitral insufficiency. Pertinent laboratory findings were decreased haptoglobin level (<2 mg . dl\(^{-1}\)) associated with increased reticulocytosis and serum LDH values (609 IU . \(1^{-1}\)). All other haematological investigations were negative. Antibiotherapy associating penicillin and gentamycin was given for 6 weeks. In January 1995, control laboratory studies revealed the persistence, but to a lesser extent, of haemolytic hallmarks (haptoglobin 46 mg . dl\(^{-1}\) — LDH 434 IU . \(1^{-1}\)). At that time, mean IVPG was decreased to 42 mmHg after increasing the doses of atenolol and nifedipine.

These two observations prompted us to review the medical notes of all patients with HCM seen in the Saint Luc University hospital from 1984 to 1994. Twenty-seven of the 61 identified cases met our inclusion criteria: adult patients with unoperated HCM confirmed by echocardiography and/or heart catheterization, and in whom sufficient haematological data were available at the time of pressure gradient determination. Among the 27 included patients, 12 (45%) had normal laboratory tests whereas 15 (55%) had abnormalities suggestive of haemolysis: decreased (<80 mg . dl\(^{-1}\)) haptoglobin level and/or increased LDH (>340 IU . \(1^{-1}\)) associated with normal transaminases values. Nine out of these 15 patients had a haemoglobin concentration below 12 g . dl\(^{-1}\) (mean ± SEM 11.4 ± 0.2). Our findings stand in sharp contrast with those of Shapiro et al. who found no evidence of haemolysis in their 39 HCM patients. On echocardiography, no differences were noted among patients with and without haemolysis with respect to left ventricular septal thickness, internal dimensions and fractional shortening. There was, however, a significant difference in mean IVPG which was significantly higher in patients with haemolysis that in those without (73.3 ± 7.5 vs 46.1 ± 9.5 mmHg, \(P<0.03\)).

In patients with acquired valvular disease, intravascular haemolysis has been related to turbulence and shear stress produced by flow through stenotic or regurgitant orifices. In vitro studies have shown that red cell damage occurred at shearing stresses between 1500 and 3000 dynes cm\(^{-2}\). Using the Bernoulli's equation, it was suggested that in HCM an IVPG of 50 mmHg could exert a shearing stress of about 4000 dynes cm\(^{-2}\) and might therefore be accompanied by haemolysis\(^{[1]}\). This is supported by our retrospective study which found higher IVPG values in the patients with haemolysis than in those without. Furthermore, an attenuation of the haemolytic process concomitant with the reduction of IVPG was demonstrated in our two patients after optimization of their therapy.

In conclusion, the prevalence of haemolysis in HCM is probably higher than previously estimated. The presence and severity of red cell fragmentation in this disorder appears to be correlated essentially with the magnitude of IVPG. Prospective studies could possibly conclude that in HCM, haemolytic hallmarks represent valuable tools in assessing average IVPG and response to therapy.

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References


Pericardial abscess due to transdiaphragmatic perforation of the pyogenic liver abscess

Pericardial abscess is a rare complication of pyogenic liver abscesses, and if untreated all patients die due to cardiac tamponade, septicaemia or complications of underlying disease\(^{[4]}\). Survival has been improved with early diagnosis, combined medical and surgical treatment, but pericardial constriction may develop suddenly or later\(^{[1]}\). Herein, we present a successfully treated patient with pyogenic liver abscess which was complicated by pericardial abscess and tamponade due to the perforation of diaphragm.

A 32-year-old male was admitted because of right upper quadrant pain, fever and a chilling sensation. He had previously been healthy. His blood pressure was 120/70 mmHg, pulse rate 107 beats . min\(^{-1}\), and body temperature 38 °C. Jugular venous pressure was elevated. Lung sounds were clear and no murmur or pericardial friction rub were heard. Tender hepatomegaly was noted. Haemoglobin was 9.5 g . dl\(^{-1}\) — leucocyte count 33 700 \(1^{-1}\) — AST 28 IU . \(1^{-1}\) and ALT 31 IU . \(1^{-1}\). Enlargement of cardiac shadow and pleural effusion were seen on the chest