The first patient is a 60-year-old man in whom the diagnosis of HCM was made in 1986. Transthoracic 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 mm Hg. 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 gentamicin 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.

M. LAMBERT
F. BOSLY
B. BOLAND
P. HAINAUT
J. L. VANOVERSCHELDE*

Divisions of General Internal Medicine
and *Cardiology.
Department of Internal Medicine.
Cliniques Universitaires Saint-Luc.
University of Louvain Medical School.
Brussels, Belgium

Pericardial abscess due to transdiaphragmatic perforation of the pyogenic liver abscess

Pericardial abscess is a rare complication of pyogenic liver abscess, and if untreated all patients die due to cardiac tamponade, septicaemia or complications of underlying disease$^{[11]}$. Survival has been improved with early diagnosis, combined medical and surgical treatment, but pericardial constriction may develop suddenly or later$^{[12]}$. 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 hepatojugular was noted. Haemoglobin was 9.5 g . dl$^{-1}$, leucocyte count 33700 µl$^{-1}$, AST 28 IU . 1$^{-1}$, and ALT 31 IU . 1$^{-1}$. Enlargement of cardiac shadow and pleural effusion were seen on the chest.
roentgenogram. An electrocardiogram showed sinus tachycardia and elevation of ST segment in leads I, II, aVL, and V_{3,4,6}. Ultrasonography revealed a 10 × 6 × 5 cm sized relatively well-margined mass with heterogeneous internal echoes in the left lobe of liver. Echocardiography demonstrated a swinging heart and massive pericardial effusion with diastolic collapse of both the right atrial and ventricular wall. Pus drainage of the liver abscesses and pericardiectomy were performed. A small perforation, 1 cm, with an irregular border was observed on the posterior aspect of right diaphragm. The pericardium was thickened and 600 ml of foul odorous pus and necrotic debris were drained. Fibrin clots were firmly attached to the underlying myocardium. Histological examination of the pericardium showed acute inflammation with microabscesses and *Escherichia coli* was cultured from both blood and pericardial pus. He was discharged after 4 weeks on antibiotics, but Doppler echocardiography revealed a slightly thickened pericardium with a mild constrictive physiology.

Early diagnosis of pericardial abscess is difficult because usual symptoms and signs of pericarditis may be absent and diagnosis may not be made until a significant degree of cardiac compression has occurred. Despite medical progress, pericardial abscess is a serious, life-threatening illness and still carries a high mortality rate. If surgical treatment is combined with medical therapy, the mortality rate decreases to 20% or less[^3,4]. Even though drainage of pus and removal of fibrin clots in the pericardium is necessary, controversy still exists about which method of surgical treatment is optimal to obtain the best immediate result and to prevent pericardial constrictions[^3,5]. Although clinical features are lacking, physicians should be alert to the possibility of pericardial abscess if there is fever, an infective focus, and some signs of cardiac tamponade.

**Successful lysis of mobile right heart and pulmonary artery thrombi, diagnosis and monitoring by transoesophageal echocardiography**

Right-atrial and/or ventricular thrombi are precursors of pulmonary embolism (PE) and are associated with a poor clinical outcome[^1]. Due to the high rate of consecutive massive pulmonary embolism with anticoagulation therapy only, the mortality rate is high and ranges from 40–50%[^1]. Even though drainage of pus and removal of fibrin clots in the pericardium is necessary, controversy still exists about which method of surgical treatment is optimal to obtain the best immediate result and to prevent pericardial constrictions[^3,5]. Although clinical features are lacking, physicians should be alert to the possibility of pericardial abscesses if there is fever, an infective focus, and some signs of cardiac tamponade.

**References**


**Figure 1 (A)** Transoesophageal examination, four-chamber-view with large, hypermobile mass in the right atrium. (B) Transoesophageal short-axis view of right ventricle and left ventricle showing the diastolic prolapse through the tricuspid valve. (C and D) same transducer position as in (A) and (B) 15 h after systemic lysis; complete resolution of the right atrial mass.