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 mm Hg. Contemporaneously, laboratory investigations disclosed haemolytic halmarks [haemoglobin <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 [haemoglobin 88 mg . dl~1 (NL 100-140) — 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 halmarks [haemoglobin 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.

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 halmarks 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 abscesses

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[1]. Survival has been improved with early diagnosis, combined medical and surgical treatment, but pericardial constriction may develop suddenly or later[2]. 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 man 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 hepato-megaly was noted. Haemoglobin was 9.3 g . dl~1 — leucocyte count 33 700 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.
Cardiac tamponade. Clinical features are lacking, physicians decreases to 20% or less. Even
an infective focus, and some signs of pericardial abscess if there is fever, should be alert to the possibility of
3'. Although compression has occurred. Despite 4 weeks on antibiotics, but Doppler echocardiography revealed an immediate result and to prevent peri-
ment is optimal to obtain the best about which method of surgical treat-
necessary, controversy still exists of fibrin clots in the pericardium is still carries a high mortality rate. If medical progress, pericardial abscess is
abscess is difficult because usual symp-

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[1-3]. Due to the high rate of consecutive massive pulmonary embolism with antiocoagulation therapy only, the mortality rate is high and ranges from 40-50%[11]. The recommended therapeutic regimen has changed within the last decade. Before thrombolysis, surgical embolectomy was the treatment of choice[3]. Current therapeutic strategies favour fibrinolytic therapy with consecutive anticoagulation[4].

We assessed the value of transoesophageal echocardiography for diagnosis and follow-up of a mobile right heart and pulmonary artery thrombi under thrombolysis with recombinant tissue-type plasminogen activator (rt-PA). In four patients (4 men, 55-74 years old) with suspected PE diagnosis and regression of right heart and pulmonary thromboembolism following a systemic intravenous, lysis therapy with rt-PA was documented by transoesophageal echocardiography (e.g. Fig 1 A-D). A submassive PE occurred in three patients. One patient had a massive PE with cardiac arrest followed by cardiopulmonary resuscitation over 40 min. In all four cases transoesophageal echocardiography clearly identified the extensive, hypermobile, worm-shaped thrombus formation in the right-sided cavities of the heart and in the central pulmonary artery in two cases. All patients were treated with 100 mg rt-PA, three patients in a front-loaded regimen over 90 min, and due to the life-threatening situation in one case, a bolus injection as ultimatum was performed with no intracerebral bleeding complications. Regression of thromboembolic masses after fibrinolytic therapy was demonstrated by transthoracic and transoesophageal echocardiography after 1 to 15 h. All patients survived and were put on coumadine; one patient developed an intracerebral bleeding with persistent hemiplegia.

In the majority of cases, right atrial or ventricular thrombi represent pulmonary emboli in transit. These may be fatal in patients treated conservatively with anticoagulation only. In the literature, the incidence of right heart thrombi in patients with proven pulmonary embolism ranges from of 3-49%[5]. Extremely mobile, long, worm-shaped masses in the right heart cavities carry an especially high early thrombus-related mortality rate which ranges from 40-50%[31]. The present therapeutic strategies favour fibrinolytic therapy with consecutive antiocoagulation with heparin and