roentgenogram. An electrocardiogram showed sinus tachycardia and elevation of ST segment in leads I, II, aVL, and V6. Ultrasonography revealed a 10 × 6 × 5 cm sized relatively well-marginated 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 abscess 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 myocardial compression has occurred. Despite drainage of pus and removal of fibrin clots in the pericardium is necessary, controversy still exists about which method of surgical treatment is combined with medical therapy, the mortality rate decreases to 20% or less[3].

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 constriction[3]. 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.

References

Successful lysis of mobile right heart and pulmonary artery thrombi, diagnosis and monitoring by transesophageal 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]. The recommended therapeutic regimen has changed within the last decade. Before thrombolysis, surgical embolectomy was the treatment of choice[5]. Current therapeutic strategies favour fibrinolytic therapy with consecutive anticoagulation[6]. We assessed the value of transesophageal 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 thromboemboli following a systemic intravenous, lysis therapy with rt-PA was documented by transesophageal 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 transesophageal 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 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 3—4%[8]. 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%[9]. The present therapeutic strategies favour fibrinolytic therapy with consecutive anticoagulation with heparin and
Pulmonary embolectomy and complete exploration of the right heart as an alternative treatment strategy has an extremely high mortality rate and should only be performed in patients with contraindications for fibrinolytic therapy or after ineffective lysis.

The efficacy of intravenous thrombolytic therapy in the lysis of right-sided heart thrombi has only been documented in a few cases without significant complications.[1-4] Based on these experiences, we chose intravenous thrombolytic therapy in all four cases as the primary treatment. In contrast to surgery, this strategy has the advantage that it also resolves the origin of the thrombus formation, which is most often located in the deep venous system of lower extremities.

The echo findings, in context with the clinical symptoms, allowed immediate initiation of thrombolytic therapy without pulmonary angiography, thus preventing a potentially fatal diagnostic procedure in these patients, namely right heart catheterization with consecutive embolization of thrombotic material into the pulmonary artery. Although we cannot rule out the occurrence of embolization of the thrombotic masses or pulmonary artery. Although we cannot rule out the occurrence of embolization of the thrombotic masses or parts thereof into the peripheral pulmonary circulation, absence of clinical signs of embolization suggest that most of the thrombi underwent local lysis.

In summary, the use of thrombolytic therapy is highly efficacious for the therapy of patients with pulmonary embolism and concomitant right atrial or ventricular thrombus formation, if there are no contraindications for such a therapy. Transthoracic and especially transesophageal echocardiography, is a powerful bedside diagnostic tool for the immediate diagnosis and follow-up of successful treatment in this life-threatening condition.

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References

Biphasic P wave masquerading as a retrograde positive P wave during atrioventricular nodal reentrant tachycardia

Retrograde impulses exiting from the atrioventricular (AV) node and spreading to the atria generally result in an inverted, negative P wave in leads II, III and aVF.[1-3] We report a patient with AV nodal reentrant tachycardia who, during tachycardia, exhibited apparently positive retrograde P wave in inferior leads occurring immediately after the QRS complexes.[4]

A 51-year-old woman with a history of recurrent sustained supraventricular tachycardia was referred to our hospital to undergo radiofrequency catheter ablation. A pseudo r' deflection in lead V1 (Fig. 1A) suggests AV nodal reentry as a mechanism of tachycardia.[1-3] It should also be noted in Fig. 1A that in leads II, III and aVF each QRS complex is followed by an apparently 'positive' retrograde P wave,[4] as indicated by an arrow. Sustained supraventricular tachycardia with a cycle length of 315 ms (AH=265 ms, HA=50 ms) was reproducibly induced by atrial extrastimulation and rapid atrial pacing. AV nodal reentrant tachycardia was diagnosed based on standard electrophysiologic diagnostic criteria. Application of radiofrequency current at a site where the so-called slow pathway potential was recorded abolished the tachycardia.

Apparently positive P waves during AV nodal reentrant tachycardia will be explained in three ways: (1) the whole positive deflection represents a truly positive retrograde P wave,[4] and is preceded by a small 'S' deflection of the QRS complex; the occurrence of an S wave may be

Figure 1 12-lead ECGs. Each panel indicates AV nodal reentrant tachycardia (Panel A), sinus rhythm (Panel B) and ventricular extrastimulus testing (basic cycle length, 600 ms) at the extrastimulus coupling interval of 400 ms (Panel C). In Panel C, the biphasic (- +) nature of retrograde P wave is more clearly characterized during S2 stimulation as compared with S1 stimulation. AVNRT=AV nodal reentrant tachycardia; RV=right ventricle; S1=basic drive stimulus during ventricular extrastimulus testing; S2=extrastimulus during ventricular extrastimulus testing. Paper speed is 25 mm . s⁻¹ in each panel.

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