From the age of 18 this had become gradually more severe, and he had observed progressive coarsening of his features. At referral he was noted to have severe facial acne and acromegalic features. Acromegaly was confirmed by basal growth hormone concentrations of 16·7-20 mU/l, which failed to decrease after oral glucose. Serum concentrations of luteinising hormone (4 U/l) and follicle stimulating hormone (3 U/l) rose to 12 U/l and 14 U/l respectively in response to gonadotrophin releasing hormone. Serum concentrations of testosterone (21·6 nmol/l (6·2 ng/ml)) and prolactin (295 mU/l) were normal. Attempted surgical removal of a suprasellar pituitary adenoma only partially suppressed secretion of growth hormone (to 13·6-14·6 mU/l); because medical treatment also failed to give adequate control external radiotherapy (4000 rads) was given. Meanwhile his acne remained active, the acromegaly became more gross, and he developed hidradenitis suppurativa of the left axilla and perineum.

Comment

Patients with acromegaly often have a greasy skin and, in common with patients with severe acne,[1] have a greatly increased rate of excretion of sebum.[2] Acne is not, however, a recognised feature of acromegaly and, as in a recent British study of 155 patients with acromegaly,[3] is not mentioned in most reviews of the disease. Severe acne was present in three of 35 Polish patients with acromegaly reported on in 1968,[4] and one French report described a 49 year old woman with acromegaly who presented with rapidly progressive nodulocystic acne.[5] Acne thus appears to be a surprisingly uncommon feature of acromegaly. Hidradenitis suppurativa may accompany severe acne, but the influence of endocrine factors on this condition has received scant study. We suggest that the pituitary endocrine abnormalities in our patients were important in the aetiology of their skin disease and, therefore, that acromegaly may on occasion present with severe acne vulgaris.

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Obscure splenomegaly in the tropics that is not the tropical splenomegaly syndrome

The tropical splenomegaly syndrome originally referred to cases of splenomegaly in the tropics for which no cause was found even after full investigation.[1] Since then it has come to refer specifically to a condition resulting from an aberrant immunological response to malaria.[2] Criteria for diagnosis include immunity to malaria, raised serum IgM concentrations, response to antimalarial drugs, and hepatic sinusoidal lymphocytosis on liver biopsy.[3] Patients with undiagnosed splenomegaly were studied to see whether these criteria were fulfilled and whether malaria was aetiologically relevant in all cases.

Patients, methods, and results

Altogether 131 adult patients with chronic splenomegaly were investigated at Kenyatta National Hospital, Nairobi. They were allocated to diagnostic groups on the basis of clinical, haematological, and parasitological findings and the results of liver biopsy. The tropical splenomegaly syndrome and hepatosplenic schistosomiasis were diagnosed specifically on the results of liver biopsy, and visceral leishmaniasis on the results of splenic aspiration. In this way 41 patients were considered to have the tropical splenomegaly syndrome, 23 hepatosplenic schistosomiasis, and seven visceral leishmaniasis. Specific, less common, diagnoses were made in 44 cases and included haematological disorders and lymphomas, liver disease with portal hypertension, tuberculosis, sarcoidosis, heart failure, and malaria. In 16 patients no diagnosis was reached. In these patients, whom we diagnosed as having "indeterminate splenomegaly," examination was inconclusive, peripheral blood films showed no relevant abnormality, results of liver biopsy were non-diagnostic, barium swallow or endoscopy, or both, did not show oesophageal varices, and visceral leishmaniasis was excluded on results of splenic aspiration or geographical background. Only one patient (6·7%) in this group had ova of *Schistosoma mansoni* detected on examination of stools or rectal snip, compared with 17 (73·9%) of those with histologically diagnosed hepatosplenic schistosomiasis ($p < 0.001$).

Malarial antibody titres were determined by an indirect fluorescence antibody test[4] and IgM concentrations by kinetic turbidimetry.[5] The table compares results in patients with the tropical splenomegaly syndrome and those with indeterminate splenomegaly.

Results of malaria indirect fluorescence antibody test (IFAT) and estimation of IgM concentrations in patients with the tropical splenomegaly syndrome and indeterminate splenomegaly

	Tropical splenomegaly syndrome (n = 38)	Indeterminate splenomegaly (n = 15)	p value
Mode of IFAT titre	1/4096	1/256	
No (%) with IFAT titre of 1/1024 or greater	35 (92·1%)	4 (26·7%)	<0·001
Geometric mean IgM (g/l) (95% confidence limits for geometric mean)	4·9 (0·7-33·7)	1·6 (0·4-7·1)	<0·001

Four patients with indeterminate splenomegaly had titres of 1/1024 or above on indirect fluorescence antibody testing, with IgM concentrations 2 SDs above the local mean in one case and 1 SD above the local mean in two cases. In the patient with the highest IgM concentration regression of splenomegaly occurred with proguanil 100 mg once daily. In the only other four patients followed up for at least six months no response to proguanil was observed.

Comment

The patient with the highest IgM concentration and a high malarial antibody titre who responded to proguanil probably had the tropical splenomegaly syndrome without the changes characteristically found on liver biopsy. The same may possibly apply to the two other patients with high titres and IgM concentrations 1 SD above the local mean. For the other patients with indeterminate splenomegaly, however, the low malarial antibody titres, low IgM concentrations, non-diagnostic liver biopsies, and failure to respond to proguanil in those followed up make the tropical splenomegaly syndrome an unlikely diagnosis. There was no evidence in these patients of any other specific condition, and we are forced to conclude that cases of obscure splenomegaly that are distinct from the tropical splenomegaly syndrome still occur in the tropics.

These findings emphasise that the tropical splenomegaly syndrome cannot be diagnosed by exclusion alone. There is a need for discussion and agreement about diagnostic criteria. Finally, as the tropical splenomegaly syndrome is now accepted to have a malarial aetiology, perhaps the time has come to abandon this confusing term in favour of one that is more specific and informative.

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Pericardiocentesis in myocarditis: the protective role of the pericardium in severe heart failure

The development of tamponade in patients with pericardial effusion is an indication for urgent pericardiocentesis. This decompresses the heart and is potentially life saving. We describe an unusual case of massive pericardial effusion with tamponade complicating myocarditis. Pericardiocentesis was undertaken but led to rapid cardiac dilatation that was ultimately fatal.

Case report

A previously healthy 23 year old woman presented with dyspnoea on exertion and central chest discomfort. The symptoms had started during a brief influenza like illness 10 weeks previously and had gradually worsened. She had not, however, required time off from her secretarial job before admission.

On examination she was tachypnoeic but not otherwise distressed. The pulse rate was 100 beats/minute with 35 mm Hg of paradox. Blood pressure was 160/90 mm Hg. The jugular venous pulse was raised to the angle of the jaw so that evaluation of the wave form and of Kussmaul's sign was not possible. Heart sounds were faint. Chest radiography showed an enlarged globular cardiac silhouette and clear lung fields. An electrocardiogram showed sinus rhythm and low voltage complexes. Echocardiography confirmed the presence of a massive pericardial effusion (figure). The dimensions of the ventricular cavity, however, were normal, and the motion of the walls appeared vigorous. The results of routine blood tests were normal, and virological screening yielded negative results.

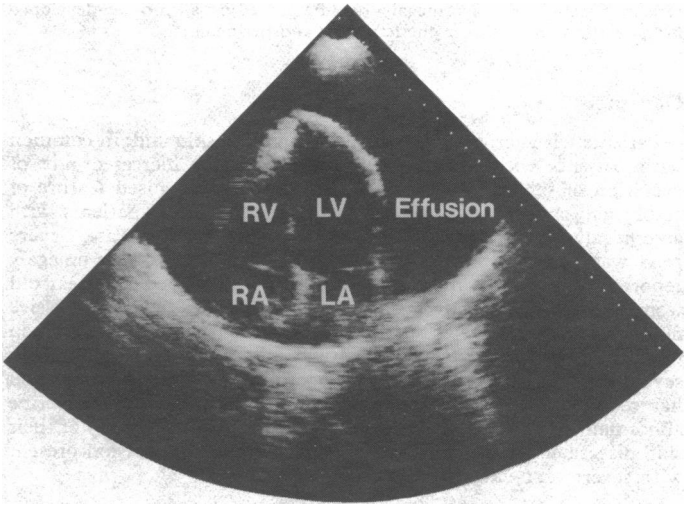
Pericardial aspiration by the subxiphisternal route yielded 1900 ml bloodstained serous exudate. Thirty minutes after the pericardiocentesis the jugular venous pulse remained raised and blood pressure had fallen to 105/60 mm Hg. Echocardiography showed successful pericardial aspiration but that considerable cardiac dilatation had occurred. The interventricular septum moved paradoxically, and the free wall of the left ventricle was hypokinetic.

During the next 12 hours cardiogenic shock developed. Right heart catheterisation showed mean right atrial and pulmonary capillary wedge pressures of 11 and 15 mm Hg respectively. The radial artery pressure was 69/40 mm Hg. An echocardiogram showed further dilatation of the four chambers and contractile failure. Despite full resuscitative measures she died 22 hours after admission.

Postmortem examination showed pericardial inflammation and a flabby, dilated heart with ventricular scarring and mural thrombosis. Microscopy of the pericardium and myocardium showed widespread inflammatory changes with round cell infiltration and patchy myocyte necrosis. Specimens sent for microbiological examination failed to yield evidence of a specific infective agent.

Comment

Massive pericardial effusion is rare in myocarditis, and the findings in this case were strongly suggestive of early cardiac tamponade. Though the pericardiocentesis was uncomplicated, it unmasked the underlying myocardial impairment: a paradoxical lowering of blood pressure occurred without correction of the jugular venous pressure. Thereafter progressive cardiac dilatation was associated with worsening ventricular function, a vicious circle described by the "descending limb" of the Starling curve.[1]



Two dimensional echocardiogram at end diastole showing large pericardial effusion (calculated volume 2 l) surrounding small heart. Dimensions of the left and right ventricular cavities are 3·2 and 2·2 cm respectively. Dots on the right indicate a centimetre scale.
LV=Left ventricle. RV=Right ventricle. LA=Left atrium. RA=Right atrium.

Experimental studies have shown that the pericardium plays an important part in preventing cardiac dilatation particularly when ventricular end diastolic pressures are raised.[2 3] The chronic effusion in this case, however, had clearly stretched the pericardium considerably, rendering it overly compliant and unable to restrain the heart on relief of the tamponade. The diseased myocardium, therefore, was unprotected against the consequences of uncontrolled dilatation.

This physiological explanation best accounts for the rapid worsening of the patient's condition after the pericardiocentesis. Trauma to the heart, kinking of the great vessels, and volume depletion are other potential causes of haemodynamic deterioration in such patients, but the clinical and postmortem examination findings provided no evidence of these complications.

This report emphasises the important constraining influence of an intact pericardium on the failing heart. In this unusual case cardiac tamponade had compensated for the pericardial dysfunction by effectively splinting the myocarditic heart. Gradual drainage of the effusion over several days might have permitted some recovery of normal pericardial compliance and prevented irrecoverable cardiac decompensation.

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