to be increasing in young women and this change has been linked to the increasing prevalence of human papilloma virus infection and vulval intra-epithelial neoplasia (VIN). A causal link is unproven but both LS and VIN may be associated with the development of carcinoma.\(^2\)\(^3\) Published data have either assessed the skin changes in patients with a primary diagnosis of carcinoma of the vulva\(^2\)\(^4\) or have observed cohorts of patients with LS for the development of carcinoma of the vulva.\(^5\) Buscema \textit{et al} in a series of 98 patients with vulval carcinoma identified four patients in whom LS was found adjacent to the carcinoma.\(^5\) Zaino \textit{et al} noted the presence of adjacent LS in 15 of 60 patients with vulval carcinoma.\(^4\) Leibowitch \textit{et al} described a high proportion of patients with vulval carcinoma associated with LS. In their series of 78 patients with vulval carcinoma 61% had LS identified histologically adjacent to the tumour. Many of these patients also had squamous hyperplasia or VIN \textit{II}.\(^2\)

The observation that LS may be present in association with a vulval carcinoma does not establish a cause and effect relationship. Studies in the 1950s and 1960s suggested that carcinoma of the vulva occurred in up to 10% of patients with LS. Of 465 patients with LS in published series 16 had coexistent carcinoma of the vulva.\(^4\) The precise relationship between symptomatic and asymptomatic LS and the development of vulval carcinoma remains unclear. In Leibowitch’s series of 78 patients with carcinoma of the vulva 43 patients had undiagnosed LS.\(^2\)

This case is a reminder that squamous cell carcinoma should be remembered in the differential diagnosis of a solitary vulval swelling, particularly if there is associated LS. Aggressive treatment of LS with topical steroids and careful long-term follow-up should be instituted,\(^7\) but whether this will reduce the incidence of vulval carcinoma remains an untested hypothesis.

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\textit{Staphylococcus aureus} pericarditis in a patient with AIDS

Bacterial pericarditis are rare in patients with HIV infection. Recently we have seen a 36 year old homosexual man who had pericarditis due to \textit{Staphylococcus aureus}.

The patient, who had never injected drugs, presented with a 24 hour history typical of acute pericarditis. On examination he was pyrexial with a temperature of 38.7°C and had a pericardial friction rub. A chest radiograph showed a normal sized heart and an ECG showed widespread concave ST segment elevation. An echocardiogram showed a moderate pericardial effusion and an indium-111 labelled human polyclonal immunoglobulin \(^{111}\)In-HIIG) scan showed pericardial accumulation, suggesting an infective aetiology.

He had been HIV-1 seropositive for three years and \textit{Pneumocystis carinii} pneumonia and cutaneous Kaposi's sarcoma were diagnosed 15 months before this admission. Eight weeks before presenting with pericarditis the patient had bronchitis due to \textit{Haemophilus influenzae} and \textit{Streptococcus pneumoniae}; at this time fibreoptic bronchoscopy had confirmed endobronchial Kaposi's sarcoma. His CD\(_4\) count was 0.04 (normal range 0.35–2.20 \(\times\) \(10^3\)) Chemotherapy with vincristine 2mg and bleomycin 30 mg once every three weeks was commenced. At the time of his admission with pericarditis, further bloods showed a white blood cell count of 2.8 \(\times\) \(10^9\) (69% granulocytes), no elevation of cardiac enzymes and no rise in viral titres in paired sera; Coxackie B IgM was negative. Sputum samples grew \textit{H. influenzae} and \textit{Str. pneumoniae}, which were treated with tetracycline in conventional doses; blood cultures were negative. A chest radiograph taken four days after admission showed an increase in the cardiac diameter and a second echocardiogram showed a pericardial effusion with pericardial thickening and diastolic collapse of the right atrium, but not of the right ventricle. The diagnosis was thought to be of chronic pericardial effusion due to \textit{H. influenzae} and further intervention was not thought to be required.

Sixteen days after admission signs of impaired right ventricular filling developed with pulsus paradoxus of 15 mm Hg. Repeat echocardiography showed no right ventricular diastolic collapse but there was evidence of restrictive cardiomyopathy, suggesting pericardial infiltration. Neither computerised tomography nor gated magnetic resonance imaging (MRI) showed evidence of pericardial Kaposi's sarcoma. On the 22nd day after admission pericardiacentesis was performed; Gram-stain of the pericardial aspirate demonstrated numerous pus cells, and culture of the fluid yielded a heavy pure growth of \textit{Staphylococcus aureus}, sensitive to penicillin.

Prolonged culture for mycobacteria was negative and cytology revealed no malignant cells. Despite IV teicoplanin and oral rifampicin he developed increasing peripheral oedema and

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ascites: echocardiography showed a small pericardial effusion with extensive pericardial thickening and restrictive left ventricular function. His condition steadily deteriorated and he died 36 days after admission. An autopsy was not performed.

Cardiac disease is increasingly recognised in patients with HIV disease. Several clinical syndromes occur, including cardiac tamponade due to pericardial effusion or haemorrhage, dilated cardiomyopathy, refractory ventricular tachycardia and/or sudden death and systemic thromboembolic disease. Pericardial pathology in association with HIV infection is not uncommon, but is usually asymptomatic: in one study of 60 asymptomatic HIV positive patients, nine had pericardial effusion on echocardiogram. An autopsy series of 115 patients with AIDS or AIDS-related complex found pericardial effusion in 35, fibrinous pericarditis in two and Kaposi’s sarcoma in seven. It is thought that pericarditis is a frequent concomitant of the terminal illness rather than contributing to death. However, where pericarditis is a cardiac symptom it is likely to be due to opportunistic infection or neoplasia, and here appropriate treatment may modify the disease course.

A review of 16 cases of pericarditis in HIV positive patients in whom an infective agent was identified included Mycobacterium tuberculosis, M. avium-intracellulare, Cryptococcus neoformans, Nocardia asteroides, and cytomegalovirus. In a previous report of S. aureus pericarditis in a patient with AIDS-related complex, a preceding episode of sinusitis was thought to be the primary focus of infection. He did not respond to antibiotics and pericardectomy was performed; he made a full recovery. The scarcity of reports of HIV-associated S. aureus pericarditis contrasts with its relative frequency in immunocompetent patients, accounting for up to 22% of cases.

Our patient is unusual in that no primary source of S. aureus infection was found either by multiple bacteriological cultures or by use of 111In-HIG scanning. It is possible that he had viral pericarditis which became secondarily infected, but against this was the fact that paired sera showed no rise. Alternatively the effusion might have been caused by pericardial Kaposi’s sarcoma. Epicardial Kaposi’s sarcoma, frequently found at autopsy, is not usually associated with pericardial effusion. Our patient had no evidence of pericardial Kaposi’s sarcoma on CT or MRI, and the pericardial fluid was not haemorrhagic, which mitigates against this diagnosis.

In conclusion, S. aureus may cause pericarditis in HIV positive patients. Pericardiocentesis enables a specific bacteriological diagnosis to be made and appropriate treatment to be instituted.

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