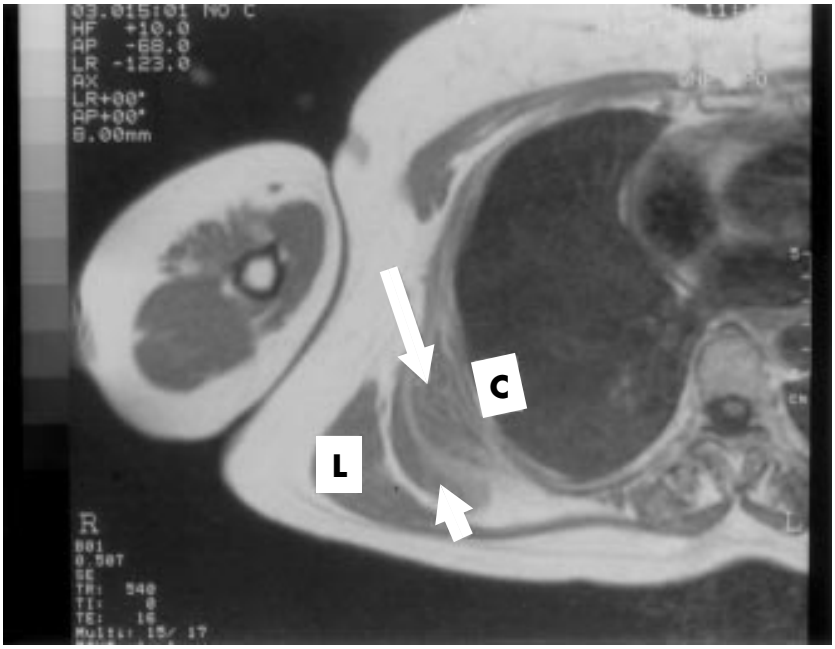

Figure 1 Magnetic resonance image (T_1 weighted axial) of the right infrascapular region showing a poorly defined mass (long arrow) with areas of high signal within. The mass lies between the serratus anterior (short arrow) and the thoracic cage (C). L, latissimus dorsi.

malignant tumours. We report a case of elastofibroma in a patient who presented with shoulder pain to a rheumatology clinic, and review previous publications. Although elastofibroma is uncommon, it has received attention in radiological and orthopaedic publications but not in rheumatology published reports.

A 43 year old Turkish woman, previously fit and healthy, was referred to our outpatient clinic with a two year history of right shoulder pain. The pain was described as a dull ache of gradual onset, around the posterior aspect of the shoulder over the scapula, which was worse on movement of the arm. There was no weakness. Over the preceding four months the patient had noticed a swelling below the inferior angle of the right scapula which would appear and disappear with movement of the arm. The patient had no other medical history or relevant family history.

On examination there was a full range of movement of both shoulders and neck with no wasting or neurological signs. Pain was reproduced around the right shoulder when the arm was circumducted. In this position a firm, poorly circumscribed, and minimally mobile mass of 5×5 cm was apparent underlying the inferior angle of the scapula. The rest of the examination was normal.

Initial investigations showed a normal full blood count, bone profile, and inflammatory markers, and a normal radiograph of the right shoulder and scapula. Subsequent magnetic resonance imaging (MRI) showed a poorly circumscribed heterogeneous soft tissue mass between the chest wall and the scapula (fig 1). The signal intensity was similar to that of adjacent muscles with interspersed strands of high signals similar to those of fat. No significant contrast enhancement was seen. The lesion was biopsied under computed tomography guidance and a histological examination showed elastic fibres within a collagenous fibrous tissue with entrapped adipose tissue, consistent with a diagnosis of elastofibroma. Surgical excision was performed because the mass was causing pain. Postoperative histology confirmed an elastofibroma. The patient has remained asymptomatic after surgery with no recurrence of the mass.

Elastofibroma dorsi, first described in 1961,[2] is a benign, slow growing, mesenchymal soft tissue lesion.[3] They usually occur in active subjects above the age of 50 with a male:female ratio of 1:5.[4] Most (99%) occur in the subscapular region, usually on the right side. The lesions have occasionally been found in the extremities, head, abdominal and thoracic cavities.[5] Of those in the subscapular region approximately 10% are bilateral.[5] The cause and pathogenesis are unclear, but it is suspected that subclinical microtrauma may lead to reactive hyperplasia of elastic fibres with consequently increased production of fibrous tissue.[6] Clinically, over 50% of subjects are asymptomatic and may present with a painless swelling; approximately 25% present with a clicking sensation when the arm is moved, while fewer than 10% present with pain.[7]

Plain radiographs may be normal or may show soft tissue density in the periscapular region when the scapula is raised.[5] Computed tomography usually shows a heterogeneous soft tissue mass with poorly defined margins.[5] MRI is the best non-invasive technique and most useful for diagnosis. Elastofibromas appear as poorly circumscribed soft tissue lesions with similar signal intensity to that of skeletal muscle but interspersed with high signal intensity areas representing adipose strands.[8] The differential diagnosis includes desmoid tumours, neurofibroma, and liposarcoma. However, these tumours usually show strong enhancement after gadolinium injection. Usually faint enhancement is seen with elastofibromas, although marked enhancement, mimicking malignant tumour, has been occasionally reported.[9] Biopsy should therefore be undertaken as the confirmatory procedure and to exclude sarcoma.

In cases where the patient is asymptomatic excision is unnecessary. Malignant transformation is unknown. In symptomatic cases local excision is the best treatment.[10] Recurrence has not been reported.

We conclude that elastofibroma should be considered in the differential diagnosis of subscapular pain. Although an uncommon lesion with a variable clinical presentation, the site and MRI appearances are characteristic. Awareness of the benign nature avoids unnecessary surgery and reassures a symptomatic patient.

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Olecranon bursitis due to *Candida parapsilosis* in an immunocompetent adult

Septic bursitis (SB) mainly affects the olecranon and patellar bursae. Subcutaneous localisation predisposes to trauma and may subsequently lead to infection. Most cases of SB are related to the subject's occupation (roofing, gardening, plumbing), but surgical interventions (aspiration, intrabursal injection) are among other probable causes.[1] Bacteria account for most cases, *Staphylococcus aureus* being the most commonly found (80%).[1 2] Fungal isolation is quite rare and always associated with immunosuppresion or debilitating conditions,[3] but some species of *Candida, Cryptococcus, Penicillium*, and *Sporothrix schenckii* have been described.[1] These atypical organisms usually develop in a late indolent pattern, and a delay in diagnosis and treatment may lead to considerable difficulties in eradication of infection. We report a case of SB caused by *Candida parapsilosis* in a previously healthy man, with no underlying disease or any risk factors, including HIV infection, who probably acquired joint infection at the hospital secondary to local steroid injection.

Case report

A 32 year old man with a one month history of mild inflammation of the right elbow presented to our hospital on 19 May 2000. He had

Table 1 Main clinical features of candida bursitis

Case [ref]	Age/sex	*Candida* strains	Localisation	Underlying disease/ risk factors	Probable source	Treatment	Outcome
1 [3]	73/M	*C albicans*	Subacromial	SLE/steroids	Candidaemia	AMB	Cure
2 [5]	77/M	*C tropicalis*	Olecranon	Bladder carcinoma	Candidaemia	AMB + bursectomy	Cure
3 [6]	48/M	*C tropicalis*	Popliteal	Lymphoma/ immunosuppresive drugs	Candidaemia	AMB + surgery	Cure
4 [7]	64/M	*C albicans*	Popliteal	Alcoholism/steroids, antibiotics	Candidaemia	AMB; ketoconazole	Cure
5 [8]	59/F	*C lusitaniae*	Olecranon	SLE, diabetes, asthma/ steroids, immunosupresive drugs	Superficial trauma (Lunger's elbow)	Fluconazole; 5-FC	Failure
6 [CR]	32/M	*C parapsilosis*	Olecranon	None	Steroid injection	Fluconazole + bursectomy	Cure

CR, current report; AMB, amphotericin B; SLE, systemic lupus erythematosus; 5-FC, 5-fluorocytosine.

an unremarkable past medical history, which did not include any toxic habits or recent trauma. Bursal aspiration showed that the synovial fluid had inflammatory characteristics (leucocyte count 4.9×10^9 cells/l (54% neutrophils), and a glucose level of 3.8 mmol/l), but there were no crystals and a fluid culture was negative. A diagnosis of olecranon bursitis was established, and conservative management (fluid aspiration) was decided on. Bursal effusion was repeated over the next four days, so a further aspiration was carried out and local injection with triamcinolone acetate (20 mg) was given. However 24 days later the pain worsened and swelling of the elbow recurred; laboratory synovial findings showed a leucocyte count of 15.7×10^9 cells/l (60% neutrophils) and a low glucose level (0.8 mmol/l). Culture yielded a few colonies of *Candida* spp, but antifungal treatment was not started because it was considered that this might be caused by contamination. One month later (28 July), the patient presented to the emergency room owing to development of a new extremely painful episode of bursitis. After joint aspiration, a steroid injection was again given, but this time a fluid culture was not carried out.

On 1 August clinical symptoms persisted. Physical examination showed an increase in the size of the olecranon bursa. The patient had never presented with fever, arthralgias, or any general complaints. Laboratory studies, including a test for antibodies to HIV, were normal or negative. Magnetic resonance imaging was performed showing multiseptate bursitis; the adjacent structures were normal. A removal of 10 ml bursa fluid again yielded a positive culture for *Candida* that was later identified as *C parapsilosis* (Majadahonda (Madrid), National Centre for Microbiology). Antifungal sensitivity testing showed a minimal inhibitory concentration for amphotericin B of 1 mg/l, 5-fluorocytosine 0.25 mg/l, fluconazole 0.25 mg/l, itraconazole 0.03 mg/l, and ketoconazole 0.015 mg/l. By the end of August, oral fluconazole was started at a dose of 400 mg/day for seven days, and then 200 mg daily. Recovery was slow and the patient needed repeated drainage. As follow up cultures were still positive, on 27 September it was decided to carry out surgical debridement with complete excision of the olecranon bursa. This material was not cultured, but histopathological analysis was performed demonstrating pseudohyphae structures, without granulomatous reaction or foreign bodies. After bursectomy, the patient continued fluconazole treatment (same maintenance dose) for six weeks more. Six months later he is completely asymptomatic.

Infection of superficial bursae (olecranon, prepatellar, and infrapatellar) is generally associated with different occupations or physical activities. Local trauma may predispose micro-organisms to penetrate by the transcutaneous route.[1] Similarly, intrabursal steroid injection, a habitual therapeutic procedure, may lead to infection. Weinstein *et al* noted that development of infection after this procedure occurred in 12% of a series of cases.[4] Most frequently bacteria cause infections, but unusual pathogens like fungi have also been described.[1] *Candida* septic bursitis is extremely rare. After a thorough review of the Medline database (from 1966 to January 2001) using medical subject headings, and keyword searches that included "septic bursitis" and "*Candida*", we found only five reports.[3 5–8] Two caused by *C albicans*, two by *C tropicalis*, and another one by *C lusitaniae* (table 1). Characteristically, in all the cases, and in the present report, different risk factors or underlying diseases were found. Four cases were caused by haematogenous spread and two induced by direct penetration, including our case. The olecranon bursa was affected in three cases, including the present report.

C parapsilosis is a well known cause of arthritis that has been described secondary to systemic dissemination in intravenous drug users,[9] and also by direct inoculation secondary to catheterisations[10] or intra-articular injections.[7] It is not strongly associated with immunocompromised hosts, but rather with invasive procedures or prosthetic devices.[11] More recently *C parapsilosis* has emerged as an important nosocomial pathogen. This is the *Candida* species that is most commonly isolated from the hands of healthcare workers.[12] In contrast with other *Candida* species, colonisation with *C parapsilosis* rarely occurs before the onset of invasive infection, suggesting an exogenous source of infection.

Appropriate antifungal drugs to treat *Candida* infections are available, but appropriate drug levels in osteoarticular structures are difficult to achieve. So for successful treatment of this infection, surgery is sometimes required. Half of the patients with *Candida* SB reviewed needed surgery for complete resolution (table 1).

We would like to summarise several aspects of the present report: Firstly, steroid injection must be carefully prescribed in order to avoid probable side effects like infection.[4] Secondly, most cases of *Candida* SB are produced by haematogenic spread, secondary to disseminated infection, whereas the present case was almost certainly through direct inoculation. Thirdly, isolation of *C parapsilosis* was neglected at the start so that antifungal treatment was delayed, leading to the need for surgery. We consider that the diagnostic delay together with a rather low maintenance dose of fluconazole were critical for the very slow resolution of the infection; probably 400 mg/day would have been more suitable for an infection in a deep compartment.

Because unusual micro-organisms are difficult to recognise and anti-inflammatory drugs may mask the symptoms, a higher degree of awareness is necessary to achieve prompt diagnosis and successful treatment. Nevertheless, special care must be taken to avoid complicating side effects in iatrogenic manipulations, so preventive measures to reduce the incidence of infection must never be omitted.

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Prevalence of allergic respiratory diseases in patients with RA

The balance between Th1 and Th2 cell activity is considered crucial in many autoimmune disorders.[1 2] It has been suggested that rheumatoid arthritis (RA) is T1 cell predominated,[3] whereas atopic diseases are T2 cell directed.[4] Some recent observations[5 6] of a decreased prevalence of atopy in patients with RA have received a lot of attention.[7] It has been suggested that a T2 cell related disorder such as atopy might have a protective role against the onset of a T1 cell mediated disease such as RA,[8] and the biological importance of the Th1/Th2 paradigm has been emphasised.

We evaluated the prevalence of atopic respiratory diseases in 126 consecutively observed outpatients with RA (diagnosed according to the American College of Rheumatology (ACR) criteria). The presence of allergic respiratory diseases was investigated in all patients by an exhaustive interview and the administration of skin prick tests by a trained allergologist. Skin prick tests were made according to the EAACI guidelines,[9] with a panel including the most common airborne allergens of our area. A diagnosis of allergic rhinitis was made in 21 patients (16.6%). The diagnosis was based on a suggestive clinical picture associated with the positivity of skin prick tests. Seven of 21 patients also had symptoms of asthma and 3/21 had undergone specific immunotherapy before the onset of RA symptoms. In 20/21 patients allergic respiratory symptoms had started before the onset of RA symptoms. In 5/21 patients atopic symptoms had totally disappeared by the time of this study.

Patients with RA with associated atopic disease did not differ from other patients with RA in the following characteristics: (*a*) sex (76.2% female *v* 75.2%); (*b*) positivity of rheumatoid factor (71.4% *v* 63.8%); (*c*) presence of subcutaneous noduli and/or other extra-articular manifestations (14.3% *v* 21.9%); (*d*) functional class according to the ACR revised criteria (class I-II: 64% *v* 60%); (*e*) current treatment with two or more disease modifying antirheumatic drugs in combination (57.1% *v* 60.9%); (*f*) current steroid treatment (57.1% *v* 54.3%). Notably, most patients from both groups (90.9% *v* 76.8%) were taking steroids at a low dose—namely, not more than 5 mg daily of prednisone, when they were evaluated for this study.

Patients with atopic diseases were younger (mean age 53.8 *v* 57.5) and had a shorter average duration of RA (4.5 *v* 9.7 years) than those without.

We found a rather high prevalence of allergic respiratory diseases in our patients with RA (16.6%), comparable with that expected in the general population.[10] Moreover, the presence of atopic disease did not seem to influence the severity of RA.

The difference between our data and other reports[5 6] may be due to the methods used to determine the presence of atopic diseases. Those other studies started from the administration of standardised questionnaires to patients with RA and this method might have caused an underestimation of atopic symptoms. Conceivably, prolonged steroid treatment, as well as the systemic symptoms and disability associated with RA, may often cause occult symptoms of rhinitis and asthma that only emerge at deeper analysis.

In conclusion, our data question the hypothesis of a mutual antagonism of RA and atopy, suggesting caution in interpreting previous data and confirming that things are often not as simple as they can seem at first glance.

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Henoch-Schönlein purpura: a possible complication of hepatitis C related liver cirrhosis

Henoch-Schönlein purpura (HSP) is a systemic small vessel vasculitis predominantly affecting children and, less commonly, adults. Classical HSP includes a tetrad of palpable purpura, arthritis, abdominal pain, and glomerulonephritis. Adults may present with any two of the four criteria in the tetrad (87% sensitivity and specificity). Gastrointestinal disease has been recorded in up to 82% of adult patients in one series[1] and is usually self limiting with colicky abdominal pain, but may progress to ischaemic bowel perforation.[2]

We present the case of a 63 year old man with IgA vasculitis, probably HSP, confounded by undiagnosed hepatitis C related cirrhosis.

He was admitted with a two week history of dyspnoea, malaise, cough, fevers, and chills, myalgias, one day of a non-blanching erythematous rash on his legs, and an ileus. His hepatitis C antibody was positive; table 1 shows the results of other laboratory studies. Cultures of cerebrospinal fluid, blood, and urine were negative. A colonoscopy was non-diagnostic.

Leucocytoclastic vasculitis was confirmed by skin biopsy, and direct immunofluorescence staining was positive for IgA deposits consistent with HSP (fig 1).

Treatment with high dose (1 mg/kg/day) intravenous corticosteroids was started. A computed tomographic (CT) scan of the abdomen showed portal hypertension, a small cirrhotic liver, small spleen, omental and perisplenic varices, an atrophic pancreas, and modest ascites. The purpuric lesions and ileus improved; however, on day 4 he became tachycardic and developed a tender abdomen. A second CT scan showed massive ascites, a partial superior mesenteric vein thrombosis, thickening, and focal and nodular irregularities throughout the small bowel (probable ischaemia), and pneumoperitoneum. Blood cultures disclosed septicaemia with *Bacteroides fragilis*. His clinical course rapidly deteriorated and he died on day 8.

There are two previous case reports of the association between HSP and hepatitis C.[3 4] The diagnosis of HSP in our patient is most likely, given palpable purpura, haematuria, abdominal pain, and a skin biopsy demonstrating IgA complexes (fig 1). However, the possibility of hepatitis C associated IgA/IgM mixed cryoglobulinaemia cannot be ruled out despite a negative cryoglobulin screen[5] on two occasions. In this patient an IgA mediated vasculitis may have been the nidus for thrombus formation and abdominal catastrophe.

The role of liver cirrhosis in the development of HSP is intriguing. Patients with cirrhosis may develop HSP as a consequence of defective liver metabolism of IgA circulating immune complexes (CICs), resulting in tissue deposition, although this is known to occur without overt vasculitis.[6]

Adult and paediatric HSP differ in the incidence and severity of renal involvement, with nephropathy and progression to renal insufficiency being greater in adult HSP,[7] which is associated with a poor outcome.[8] Gastrointestinal manifestations vary widely and include abdominal pain, nausea/vomiting, intestinal haemorrhage and, rarely, perforation.

There have been no large clinical trials in adults with complicated HSP. Corticosteroids used in a series of children have been shown to relieve symptoms,[9] but fail to deal prospectively with the prevention of abdominal complications. Adults respond favourably to corticosteroids and may be managed with short courses of treatment,[10] but corticosteroids may also mask severe abdominal catastrophe.

Several important points can be learnt from this case report:

- Although nephritis is the most important long term prognostic factor in HSP, in the short term, gastrointestinal disease can lead to death despite early therapeutic intervention
- Liver cirrhosis secondary to hepatitis C may precipitate development of HSP or mixed cryoglobulinaemic vasculitis through the defective metabolism of CICs
- Given the increasing incidence of hepatitis C related liver disease world wide, the association of these diagnoses and their clinical implications should be considered more often.

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