Primary psoas abscess

Primary psoas abscess is a rare infection with an often vague and non-specific clinical presentation, especially in children. In Asia and Africa 99.5% of all psoas abscesses are primary, compared with 61% in the United States and Canada and 18.7% in Europe. \(^1\) Approximately 70% of psoas abscesses occur in patients younger than 20 years of age, with a male preponderance of 3:1. \(^1\) Fifty seven per cent of psoas abscesses occur on the right side, 40% on the left side, and 3% bilaterally. \(^1\) We present the following case and show the magnetic resonance imaging to emphasise the presenting signs, symptoms, and findings of this unusual infection.

A 13 year old white girl was in excellent health until she developed a dull ache in the right hip. She was non-contributory; she denied smoking, walked with a limp. Her past medical history was unremarkable. Over five days she developed progressively severe, dull pain, localised to the right hip. The patient deferred any direct trauma or excessive strenuous activity. Laboratory tests were within normal limits. A blood cell count of 15.2 \(\times 10^9\) /l (77% neutrophils, 14% lymphocytes, 8% monocytes) and platelets were 415 \(\times 10^9\) /l. An erythrocyte sedimentation rate was 115 mm/1st h (normal <20 mm/1st h). Urine analysis disclosed trace blood and protein; the remainder of the laboratory tests were within normal limits. Blood and cerebral cultures were negative.

Magnetic resonance imaging (MRI) of the abdomen and pelvis showed grossly abnormal signal intensities of the left psoas muscle (figs 1 and 2). Although a discrete abscess cavity was identified, fine needle aspiration under imaging guidance, and microbial cultural evidence of the causative organism. \(^1\) If abdominal CT or MRI is unavailable, ultrasonography may demonstrate the inflammatory mass. \(^1\) Gallium-67 scanning may be useful in the diagnosis of psoas abscesses and detection of concomitant infectious foci. \(^1\) Diffuse symmetric signal decreases of psoas abscess include bacterial infection of the hip, avascular necrosis of the hip, irritable hip, necrotising fasciitis of the psoas muscle, myonephritis, pelvic inflammatory disease, retrocaecal appendicitis, iliopsoas abscess, disc, avascular necrosis, vertebral or pelvic osteomyelitis, and epidural abscesses. \(^1\) These entities should be distinguishable upon the correlation of history, physical examination, laboratory tests, and imaging studies.

The cause of primary psoas abscess remains uncertain. Proposed mechanisms of psoas abscess formation include haematogenous spread from primary infectious foci or local trauma with intramuscular haematoma formation predisposing to abscess development. \(^1\) In secondary psoas abscess the most commonly associated disorder is Crohn’s disease; other disorders include appendicitis, colonic inflammation or neoplasm, disc infections, and a variety of intra-abdominal or retroperitoneal infections. \(^1\) Primary psoas abscesses are caused by a single organism in 87.5% of cases; \(^2\) \(75\%\) of cases: primary Staphylococcus aureus (88.4%), streptococci (4.9%), and Escherichia coli (2.8%). \(^3\) Blood cultures are positive in 41.7%, usually for Staphylococcus aureus. \(^4\) In the past decade the majority of patients with a primary psoas abscess were intravenous drug users (86%) infected with the human immunodeficiency virus (57%). \(^5\) Treatment for primary psoas abscess includes percutaneous drainage combined with systemic antibiotic administration. \(^6\) Surgical drainage is preferred for the patients in whom the psoas abscess is associated with underlying bowel disease. \(^7\) With appropriate treatment, psoas abscess rarely results in death (2.5%). \(^7\) Death from psoas abscess is associated more commonly with inadequate or delayed drainage, or both. \(^7\) The patient responded well to antibiotic treatment and recovered completely.


Figure 1 Coronal magnetic resonance imaging scan of the abdomen showing abnormal signal intensity in the inferior pole of the left psoas muscle (arrow). Figure 2 Cross sectional magnetic resonance imaging of the pelvis showing abnormal signal intensity of the psoas close to approximating the bladder (arrow).
Klippel-Feil syndrome in the prehispanic population of El Hierro (Canary Islands)

Klippel-Feil syndrome is an uncommon autosomal trait (1:10,000 births), mainly affecting the cervical spine. The classic clinical triad described by Klippel and Feil in 1912—short neck, low dorsal hairline, and restricted neck mobility—is the result of the fusion of a variable number of cervical vertebrae, sometimes reducing their number, and cervical spine bifida. Extraosseous changes, hemivertebra, vertebral body clefts, and thoracolumbar syndrholembar abnormalities, are sometimes seen.

In a prehispanic ossuary containing remains of at least 121 subjects in the island of El Hierro (Canary Islands), we noted:

1. A C2-3 block, with both the vertebral bodies and the medial ends of the arches, well preserved right intervertebral foramina, and foramina transversaria (fig 1), and a normal medullary canal. The body of the third vertebra shows intense degenerative signs.

2. A C5-6 block, consisting of two vertebrae with intense degenerative changes fused both by the vertebral bodies and the medial part of the arches. Both cervical blocks seem to belong to the same subject.

3. A well preserved atlas bone, with an incomplete anterior part of the left arch, with sharp, fine proximal, and distal ends (fig 2A), supporting an underlying developmental defect rather than an acquired one.

4. A left hemiasis (fig 2B); although it is possible that the right body of the bone was partially fused with the left one (and was not recovered in the archaeological excavation), the posterior end of the arch was neither fractured nor fused to any other bony structure, thus pointing to a developmental defect.

5. Fusion of C2 and C3 (and C5-6), hypoplasia of the arch of the atlas, and complete bipartition of the atlas' constituent distinct features of Klippel-Feil disease. Thus the subject with the fused C2-3 and C5-6 blocks and the hemiasis was probably affected by this disease. Possibly, the second atlas belongs to another subject with the same disease, though this possibility should be cautiously admitted. The two atlas bones show different developmental abnormalities. In the newborn, the ossification of the cartilaginous anterior and posterior arches of the atlas takes place progressively from the already ossified lateral masses. Often, especially in the anterior arch, secondary ossification centre(s) appear. In our case it seems that hypoplastic development of the anterior left arch took place. Because the hypoplastic part of the arch is in its middle part, probably, a second ossification centre was present, but ossification was never completed; in this sense, it is similar to the case described by Chigira et al., which also showed fusion of C5-6.

The “hemiatlases” perhaps is really an atlas with a midline cleft and a lost half, though the posterior arch does not reach the midline, so it never became fused with the right half of the bone. A secondary posterior ossification centre sometimes appears during the first years of life. In this case, it was absent, in contrast with the anterior secondary ossification centre which was surely present in the former case.

Perhaps familial links existed between the two subjects. Klippel-Feil syndrome is a heterogeneous disorder, showing different alterations in different families. The simultaneous finding of different developmental abnormalities of the atlas in our two cases—assuming that the second one truly represents a case of Klippel-Feil—may either reflect an unusually high prevalence of this entity in the prehispanic population of El Hierro, or may also indicate that even in the same family clinical expression of the Klippel-Feil syndrome is variable.

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Haematopoietic stem cell transplantation (HSCT) in a patient with Sjögren’s syndrome and lung malignant lymphoma cured lymphoma not the autoimmune disease

Haematopoietic stem cell transplantation (HSCT) has been used in an attempt to control autoimmune diseases that respond poorly to conventional treatment, or as a way to...
of readjusting the immunological balance. As far as we know, only one case of primary Sjögren's (SS) has been reported, with an unfavourable outcome. Another patient received an allogeneic bone marrow transplant and also had an unfavourable outcome. We describe here a further patient with primary SS who underwent HSCT for a non-Hodgkin's lymphoma affecting the lung (large cell, mucosa associated lymphoid tissue (MALT) lymphoma) and review the literature on the effects of HSCT on the autoimmune features and histopathological changes in primary SS.

Case report

A white woman, aged 42, developed recurring parotid swelling and symptoms of Sjögren syndrome, with a Schirmer's test I of 5 mm in the right eye and 4 mm in the left eye. Break up time was 6 s and sialometry was <1 ml. She had periodic relapses of her parotid swelling. In August 1994 (aged 57) lung x-rays and computed tomography disclosed a parenchymal nodule of 3 cm in diameter in the basal left lobe. She underwent a lobectomy that disclosed a MALT, of the large cell B lymphoma histotype, stage IE. In December 1994 two more nodules in the right lobe, with hilar bilateral adenomegaly, led to the diagnosis of a relapse of her lymphoma, which had progressed to stage IV. She then received six courses of F-MACHOP (vincristine 0.5 mg/m² at hours 0 and 12; cyclophosphamide 800 mg/m² intravenous (IV) bolus at hour 36, 5-fluorouracil 15 mg/kg IV for six hours at hour 36, cytarsine-arabinoside 1000 mg/m² IV for six hours at hour 42, doxorubicin 60 mg/m² IV bolus at hour 48, methotrexate 500 mg/m² IV for six hours at hour 60, prednisone 60 mg/m² from day 1 to 14), and folinic rescue (20 mg/m²/day from day 1 to 14), and folicin rescue (20 mg/m² IV bolus at hours 60, 94, 86, 120, 120), with a prompt reduction of hilar adenopaties and a net decrease of pulmonary nodule size. However, no complete remission was recorded. She was then offered the possible chance of an HSCT. The patient (patient 1) reports the myeloablation, conditioning, recovery, and reinfusion of stem cells. After three years of follow up no relapse of the lymphoma has occurred. Sjögren syndrome after transplantation was unmodified, however, with a persistently poor function of the salivary glands, an unchanged serology (antinuclear antibody titre 1/2560), and an unchanged histopathology (Chisholm-Mason grading = 4) despite having mild fibrrosis of the salivary glands.

In table 1 we give the characteristics of the other patient with primary SS (No 2), previously reported. It can be seen that the conditioning regimen, pretransplantation treatment, stem cell rescue, and bone marrow reconstitution were different. However, in this case, also, SS was not cured and there was no remission. An immunological reassessment showed persistence of the immunological imbalance and poor function of the salivary apparatus.

Table 2 shows the results for patients with three more common autoimmune rheumatic diseases (rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), and scleroderma (SSc)) treated with HSCT and who received an adequate follow up. A total of 270 such patients are registered so far in the European Bone Marrow Transplant/EBMT registry, but the number who have received adequate follow up is much smaller.

Current data suggest that best results have been obtained in RA, the worst in SSc, suggesting that T helper 2 oriented diseases have a poorer response.

Results for SS seem to confirm this because HSCT cured lymphoma but did not improve the autoimmune disease. No changes were recorded in the function of salivary glands, or in the synthesis of ANA, or the histopathology. The other case reported did show some early improvement in the function of the glands, but no improvement afterwards and an infection leading to death. Early recurrence of autoimmune features and of autoantibodies was seen in patients with SLE and CREST. We do not know whether various conditioning regimens or myeloablation approaches (with or without T cell depletion) might result in different outcomes. It seems unlikely that T cell depletion would offer a better prospect, especially in view of the increased risk of long term immunosuppression, lymphoproliferative diseases, and infections. On the other hand, allogeneic bone marrow transplantation, even though clearly having intrinsic stem cell defect, does not represent a definite cure either and the related morbidity-mortality still remains too high to be accepted as a possible alternative. As benefits have been seen in around two thirds of the cases treated so far, controlled trials in the three major rheumatic diseases are eagerly awaited.

We gratefully acknowledge the invaluable help we received from Professor A Tyrndall, who provided us with the latest data available of the EBMT/EULAR registry on haematopoietic stem cell transplantation in autoimmune diseases.

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5 Euler HH, Marmont AM, Bacigalupo A, Fastenrath S, Dreger P, Hofnicht M, et al. Intrinsic stem cell defect, or does not represent a definite cure either and the related morbidity-mortality still remains too high to be accepted as a possible alternative. As benefits have been seen in around two thirds of the cases treated so far, controlled trials in the three major rheumatic diseases are eagerly awaited.

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Table 1  Lymphoma characteristics, conditioning regimens, side effects and outcome of the two female patients so far studied, after haematopoietic stem cell transplantation

<table>
<thead>
<tr>
<th>Variables</th>
<th>Patient 1</th>
<th>Patient 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>57</td>
<td>34</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>NHL* of the lung (large cells MALT* lymphoma)</td>
<td>Immunoblastic B lymphoma</td>
</tr>
<tr>
<td>First line treatment</td>
<td>6 cycles of F-MACHOP</td>
<td>VACOP-B* followed by VIPF*</td>
</tr>
<tr>
<td>Conditioning regimen</td>
<td>ARA-C* 200 mg × 2/day × 4</td>
<td>BCNU 300 mg/m²</td>
</tr>
<tr>
<td></td>
<td>VP16* 200 mg × 2/day × 4</td>
<td>Etoposide 800 mg/m²</td>
</tr>
<tr>
<td></td>
<td>CTX 1500 mg/day × 4</td>
<td>Cytarabine 1600 mg/m²</td>
</tr>
<tr>
<td></td>
<td>BCNU* 200 mg</td>
<td>Melphalan 140 mg/m²</td>
</tr>
<tr>
<td>Refusion</td>
<td>MNC* 4.5 × 10⁹/kg</td>
<td>MNC 2.42 × 10⁹/kg</td>
</tr>
<tr>
<td></td>
<td>CD4 0.9 × 10⁹/kg</td>
<td>CD4 4.60 × 10⁹/kg</td>
</tr>
<tr>
<td></td>
<td>CD3 not counted</td>
<td>CD3 37.03 × 10⁹/kg</td>
</tr>
<tr>
<td>Literature on chemotherapy</td>
<td>Days to PMN* &gt;10 × 10⁹/l = 10 days</td>
<td>Days to PMN &gt;10 × 10⁹/l = 11 days</td>
</tr>
<tr>
<td></td>
<td>Days to PMN* &gt;10 × 10⁹/l = 8 days</td>
<td>Days to PMN &gt;10 × 10⁹/l = 18 days</td>
</tr>
<tr>
<td>Sides effects/outcomes</td>
<td>Alive in complete continuous lymphoma remission after 3 years</td>
<td>No remission of autoimmune disease</td>
</tr>
<tr>
<td></td>
<td>Died 20 months after transplantation for Pneumocystis carinii pneumonia</td>
<td></td>
</tr>
</tbody>
</table>

* NHL = non-Hodgkin's lymphoma; MALT = mucosa associated lymphoid tissue; F-MACHOP: F = 5-fluorouracil, M = methotrexate, A = Adriamycin, C = cyclophosphamide, H = doxorubicin, O = Oncovin, P = prednisone; ARA-C = arabinoside-C; VP16 = etoposide; CTX = cyclophosphamide; BCNU = carmustine; MNC = mononuclear cells; PMN = polymorphonuclear cells; Plt = platelets; VACOP-B: V = Vepepids, A = Adriamycin, C = cyclophosphamide, O = vincristine, P = prednisone, B = bleomycin; VIPF: V = vinblastine, I = ifosfamide, P = prednisone, F = etoposide.

Castleman’s disease

A 65 year old woman presented in February 1998 with joint pains, mild weight loss, and a low grade irregular fever. Initially, pain was localised around both shoulder joints. Subsequently, elbows, wrists, hips, and knees were affected, with morning stiffness, but without particular morning stiffness. The symptoms were accentuated by movement, but also persisted during the night, often keeping the patient awake. On clinical examination, there was limited painful movement of the shoulders and hips with a marked reduction in strength. The small joints of the hands and feet were not affected. No other pathological conditions were found. Laboratory findings showed a marked increase in erythrocyte sedimentation rate (ESR; >100 mm/1st h), hyper-globulinaemia and a mild anaemia, whereas enzymatic activity (serum aspartate aminotransferase, serum alanine aminotransferase, alkaline phosphatase, lactate dehydrogenase, and creatine kinase) was within the normal range. A diagnosis of polymyalgia rheumatica was made and a rapid and marked clinical improvement was obtained with low dose steroid treatment (prednisone 12.5 mg/day). Pain disappeared, muscle strength and joint function became normal within a week. A decrease in ESR (to 40 mm/1st h) and η globulinaemia was noted at one month. The clinical condition remained satisfactory during 1998, with a complete normalisation of ESR and η globulinaemia after three months.

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