Progressive multifocal leukoencephalopathy: differential diagnosis in HIV

- lymphoma
- toxoplasmosis
- HIV encephalopathy
- cryptococcus

Box 4

progressive multifocal leukoencephalopathy may result from reactivation of an otherwise dormant infection. In practice, the clinical picture with supportive radiological evidence is usually sufficient for diagnosis. The characteristic radiological changes of progressive multifocal leukoencephalopathy are non-enhancing, low density CT lesions without mass effect. These lesions are high signal on T2-weighted MRI. Clinical features at presentation are typically far more striking than the more subtle CT scan changes. Routine CSF microscopy and biochemistry are usually normal or may show only non-specific changes. Recent work suggests that the polymerase chain reaction to identify JC virus DNA in CSF is a highly specific and moderately sensitive technique for positive diagnosis without the need for biopsy.

In HIV-infected patients the diagnosis of progressive multifocal leukoencephalopathy without pathological/polymerase chain reaction confirmation demands exclusion of alternative diagnosis (principally toxoplasmosis, HIV encephalopathy and CNS lymphoma). HIV encephalopathy (AIDS dementia complex) does not usually produce focal signs and the other two disease processes are associated with ring-enhancing lesions on neuroimaging. However, as toxoplasmosis responds relatively well to treatment, HIV-positive patients with presumptive progressive multifocal leukoencephalopathy are often given a course of pyrimethamine and sulfadiazine so that treatable pathology is not overlooked.

There is no useful treatment for progressive multifocal leukoencephalopathy and the prognosis is poor, patients rarely surviving more than a few months. Even in the absence of known HIV infection and/or risk factors the possibility of an HIV-related illness should be considered early in the work-up of patients with unusual neurological disease. Once HIV-related CNS disease has been diagnosed prompt investigation is essential.

Final diagnosis

Progressive multifocal leukoencephalopathy presenting as cortical blindness in a patient with AIDS

Keywords: progressive multifocal leukoencephalopathy, cortical blindness, AIDS, MRI

The post-traumatic painful testis

FI Chinegwundoh

A 13-year-old schoolboy presented to the Accident and Emergency department having awoken with marked left testicular pain and vomiting. The previous day he had suffered a blow to his scrotum whilst engaged in a sporting activity. The immediate discomfort was insufficient for him to seek medical attention. A month previously he had experienced a short-lived episode of left scrotal pain. Examination revealed a hard, tender left scrotal swelling.

Questions

1. Suggest two differential diagnoses?
2. Which investigation may help in the diagnosis?
3. What is the treatment of the condition?
Questions

**Question 1**
The differential diagnosis includes any cause of a scrotal swelling: testicular contusion, haematocoele, torsion, orchitis and tumour. In view of the antecedent history, the first two options would spring to mind.

**Question 2**
Possible investigations include scrotal ultrasound, isotope scrotography, and surgical exploration. Ultrasound is particularly good in the diagnosis of hydroceles and mass lesions of the testes, such as tumours. It is less helpful in the diagnosis of torsion. Isotope scanning is good at diagnosing or excluding torsion. With a torsion there is decreased radioisotope uptake by the testicular vasculature. The radioactive dose is minimal. If the suspicion of a torsion is high, surgery should not be delayed by undue investigations.

**Question 3**
Examination revealed a hard, tender left scrotal swelling. The presumptive diagnosis was testicular contusion. Ultrasound examination was equivocal but raised the question of torsion. At scrotal exploration an intravaginal torsion was identified within a tense hydrocele. The ischaemic testis was adjudged viable. Treatment therefore consisted of eversion of the tunica and bilateral testicular fixation was undertaken. The patient made an uneventful recovery.

**Discussion**
Trauma is an infrequently reported precipitant of testicular torsion. Most studies have found the incidence to be approximately 5–6%. The "bell-clapper" anatomic predisposition to torsion, classically described by Muschat and most frequently identified with nontraumatic torsion, also appears to best explain the findings of intravaginal trauma-induced torsion. It is proposed that trauma produces torsion through induction of cremasteric muscle spasm in the presence of a high investment of the tunica vaginalis.

In the case reported such an abnormality was detected at operation. The left testicular pain experienced a month previously may well have been a torsion that spontaneously resolved. The elicitation of such a history may aid the clinician in the differential diagnosis of post-traumatic testicular pain. Failure to consider torsion may lead to testicular loss.

**Final Diagnosis**
Testicular torsion.

**Keywords:** scrotal trauma, differential diagnosis, torsion of testis, testicular fixation

The post-traumatic painful testis.

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doi: 10.1136/pgmj.72.846.251

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