Meningeal and osteoarticular tuberculosis

Marina Pereira*

Abstract

The extrapulmonary involvement of tuberculosis, particularly of the meninges or spinal cord, although uncommon, is a potentially curable condition if recognised and treated adequately at an early stage.

This association was studied in a patient, with epidemiologic, clinical and laboratory data and evolution typical of this disease.

Key words: spondylodiscitis, Claude-Bernard-Horner syndrome, leptomeningitis.

Introduction

Since 1985, cases of tuberculosis have been increasing in the Western world due to the higher number of immunosuppressed patients with AIDS, and the appearance of mycobacterium strains resistant to the usual antituberculostatics. The disease is more prevalent in various parts of the world, such as Africa, Asia, Central America and South America.[1]

The spectrum of the disease has changed, with cases of extrapulmonary tuberculosis increasing at a faster rate than pulmonary tuberculosis.[2] Tuberculous meningitis has high morbidity and mortality rates.

The presence of bacteria in the cerebrospinal fluid is the only sure diagnostic method, and in association with early treatment, it is crucial for a favorable prognosis.[3]

This paper presents a clinical case of a patient admitted for systemic involvement by tuberculous meningitis, with more marked manifestations on the central nervous system and osteoarticular system, an association that is quite rare.

Clinical case

Female patient, aged 52, of Indian race, a native of Pakistan and residing in Mozambique until 1988, when she immigrated to Portugal.

Two years before hospitalization, she began to experience pain in the lumbar spine and right gonalgia with signs of inflammation, periods of remission and exacerbation associated with anorexia, adynamia and weight loss of around 17 kg during this period.

One year before hospitalization, she started suffering diffuse headaches that disappeared spontaneously, accompanied by fever and sweating in the evenings. In the week prior to hospitalization, the pulsating, frontal parietal headaches worsened, accompanied by nausea, persistent fever, dysuria, pollakiuria and terminal hematuria, which worsened and led to hospitalization on the 19th July 1991.

The patient lives with her daughter and granddaughter, who had pulmonary tuberculosis in 1989 and were treated with antituberculostatic drugs for one year.

At the time of hospitalization, the patient had fever (38.5ºC); palpable, painful liver in the right region; dorsal lumbosacral scoliosis; and spinal apophysis of D6-D7 which was painful to touch. There were signs of inflammation in the right knee joint.

From the tests carried out prior to admission, the following are highlighted:

X-ray: incipient discarthrosis in discs C5-C6, initial arthrosis in the sacroiliac and coxofemoral joints, signs of bilateral femorotibial and patellofemoral gonarthrosis, and osteopenia of the pelvis.

A bone scan performed shortly before admission showed hyperfixation of the radioactive drug in D6 and in the right knee. During hospitalization, the following were observed: normochromic and normocytic anemia with Hb: 10.2 g/dL, VS: 76 mm/1st hour, serum iron: 31 µg/dL, serum sodium: 134 mEq/L, and pyuria with isolation of E. coli in the urine culture.

*Resident to the Supplementary Internship in Internal Medicine
Medicine Service IV at the Hospital de Santa Maria, Lisbon

Tests for Tuberculous meningitis in three urine samples and three expectoration samples were negative. The Mantoux test was strongly positive, with a papule of 30 mm. Lumbar puncture revealed: clear, colorless liquid, with protein concentration (120 mg/dL) in the Pandy ++ test, hypoglycorrhachia (54 mg/dL) and pleocytosis (60 cells/mm3). The next day, a new lumbar puncture revealed a darkish, xanthochromic fluid in the Pandy +++ test, 164 cells/mm3 and a slight predominance of PMN cells. Ophthalmologic examination for detection of choroidal tubercles was negative.

X-ray of the lumbar spine revealed narrowing of the intervertebral space D6-D7 with irregularity of the inferior plateau of D6 and a wedge deformation of the vertebral body – aspects suggestive of spondylodiscitis.

On the 14th day of hospitalization, she started experiencing mental confusion and meningeal signs, anisocoria, left central facial palsy and motor signs of focal lesion in the right cerebral hemisphere. Craniocerebral CT revealed probable right posterior parietal infarction and supratentorial ventricle ectasia, suggesting active hydrocephalus.

Given the results of the complementary exams and the patient's clinical evolution, the doctors opted to begin antituberculosis therapy with: rifampicin (600 mg/d), pyrazinamide (1500 mg/d), isoniazid (300 mg/d), ethambutol (1200 mg/d), associated with pyridoxine (40 mg/d) and allopurinol (300 mg/d), as well as corticosteroids (dexamethasone: 20 mg/d).

On the following day, patient presented right ptosis, right enophthalmos and miosis, symptoms that characterize Claude-Bernard-Horner syndrome. 48 hours later, she presented acute pulmonary edema, generalized seizures and deep coma. A ventriculoperitoneal shunt was immediately placed.

Neurological evolution was good, with the signs of Claude-Bernard-Horner syndrome persisting for a few months.

The results of the first cultures of liquor in Lowenstein-Jensen media were positive.

A CT scan of the spinal cord revealed, at this point, besides lytic lesions in D6, with the disappearance of disc D6-D7, a paravertebral mass with density of soft parts, involving vertebrae D5, D6, D7, prolapsing into the spinal cavity, surrounding the dural sac, with slight obliteration of the D6-D7 right conjugation hole compatible with paravertebral and epidural abscess.

NMR study of the dorsal region, performed two weeks later, confirmed spondylodiscitis, but the paravertebral abscess was no longer detected.

Clinically, the patient had no signs of spinal cord compression, and the initial therapy was maintained.

The patient was discharged after three months. She received antituberculostatic drugs for twelve months and remained asymptomatic, with no neurological or osteoarticular sequelae, but with permanent ventriculoperitoneal drainage.

Discussion

The patient presented in this paper has associated osteoarticular and meningeal tuberculosis. Tuberculosis of the CNS is generally associated with active extrapulmonary tuberculosis, generally presenting skeletal and urogenital lesions.

Acute inflammatory caseous meningitis, in its diffuse form, is responsible for clinically recognizable meningeal tuberculosis and the most common form of neurotuberculosis. It corresponds to a meningoencephalitis with filling of the basal cisterns by a flexible, exudative tissue, which is more marked in pontine and interpeduncular cisterns, extending along the third ventricle towards the chiasmatic cistern. The extension of the exudate in the cerebellar-medullar cistern can block the Luschka's foramen, resulting in hydrocephalus, as occurred in this patient.

More commonly, hydrocephalus occurs due to a blockage in the basal cisterns by the exudate in the acute stage, or by adhesive leptomeningitis in the chronic stage.[4]

As in the case described, the most frequent ischemic lesions and infarcts occur in the middle cerebral artery region, and can be superficial or affect the basal nuclei or hypothalamus, being secondary to vasculitis of the perforating vessels. The most commonly affected vessels are the terminal branch of internal carotid and the proximal 2 cm of the middle cerebral artery in the Sylvian fissure.

The patient's clinical condition was as would be expected after the prodromal phase, with headaches, vomiting and fever.[5,6] Photophobia, aphasia, paresis, seizures and focal neurological deficits, such as palsy of the more severely affected cranial nerves, may also occur (III, IV, and VII).[6,7]

This may be followed by neck stiffness and other meningeal signs.

Later, as the adhesive leptomeningitis progresses, signs of hydrocephalus may be prevalent, with progressive deterioration of consciousness.[8] The rapid improvement in focal deficits in the studied patient – observed after ventricular drainage – suggested that the dilated ventricles were compressing the vessels affected by vasculitis, worsening the ischemia.

Characteristically, the cerebrospinal fluid is clear, with pleocytosis and a predominance of PMN cells in the initial stage, followed by lymphocytosis, increased protein and glycopenia.

Spinal tuberculosis affects 50% of the locations of tuberculosis in the bone.[9] As occurred in our patient, the process began in the vertebral body, spreading to the intervertebral disc and, subsequently, to other adjacent vertebra. With the progression of the disease, an abscess is formed which, fistulating through the anterior and posterior longitudinal ligaments, penetrates the muscle-facial regions, and continues towards the paraspinal region, generally in the dorsal region.

X-rays and NMR characteristically show the involvement of two adjacent vertebrae, periarticular osteopenia, narrowing of joint space, cortical irregularity, subchondral and vertebral body erosions, periosteal bone neoformation, subligamentary extension of the paraspinal abscesses, and spinal cut.[10]

The cure consists of bone fusion of the damaged vertebral bodies, with permanent deformation of the spine, which may lead to gibbosity – Pott's disease. Given the fact that a knee was affected in this patient, it is noted that joint generally affects the hips, thighs and knees.[10]

The patient was treated with a quadruple antituberculosis regimen, as follows:[9] pyrazinamide, rifampicin, isoniazid and ethambutol in the first two months. The pyrazinamide was withdrawn in the following four months, but all other drugs were continued. The ethambutol was then withdrawn, while the rifampicin and isoniazid were continued for about 12 months. The active hydrocephalus required urgent intervention, with ventricular drainage. ■

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