Neurocysticercosis (NCC) is a relatively common disease in South Africa. It occurs in the rural areas surrounding Bloemfontein in the Free State province, as well as in neighbouring Lesotho. We report on a 46-year-old female admitted with vertebral and spinal NCC, who was newly diagnosed with human immunodeficiency virus (HIV), and had a CD4 count of 46 x 10⁶ cells/l. She presented with severe pain, associated with spasms and weakness of the lower limbs. She had similar prior episodes and underwent spinal surgery to evacuate cysts. Magnetic resonance imaging revealed a spinal lesion at T12, causing vertebral collapse. During subsequent surgery, cysts were extracted from the subarachnoid space and spinal medulla. Yamshi needle biopsy of the vertebral body was performed and sent with the cysts for histological examination. A diagnosis of spinal NCC was made on the basis of histological findings. The patient had a recurrence of a former disease, leading to the question of whether or not this recurrence was as a result of a weakened immunity, due to her HIV status. The patient was treated with albendazole, steroids, analgesics, and amitriptyline. Her condition improved after surgery, and she was subsequently admitted to a regular ward. To our knowledge, this is the first case in the Free State of spinal NCC that extended into the adjacent vertebral body. The occurrence of cysticercosis is escalating due to factors such as HIV/AIDS, and poor socio-economic conditions. Its widespread distribution poses a research challenge with regard to the true extent of the disease.

Introduction

Cysticercosis is a multi-system disease resulting from the seeding of the larval form of the pork tapeworm, Taenia solium, to various organs of the body. It is contracted via the faecal-oral route, after ingestion of viable eggs in foods contaminated with human faeces. The eggs reach the duodenum and hatch to release the oncospheres, which penetrate the intestinal wall, and enter the mesenteric blood vessels or lymphatic system. Eventually, the eggs are transported to the capillaries in almost any tissue of the body, particularly the brain. The cysticerci are notorious for their encystment in the central nervous system (CNS), known as neurocysticercosis (NCC). Globally, NCC is the most common cause of adult-onset seizures, with greater incidence rates in developing countries.¹² Fifty million people are affected by NCC, while 50 000 deaths directly related to NCC are reported annually worldwide.¹³ NCC is endemic in Central and South America, sub-Saharan Africa, including South Africa, and Asia.¹ Most cases reported in the West pertain to immigrants from endemic areas.⁴⁵ The condition affects all age groups, although children often develop an unusual encephalitis-type variant of the disease.¹

In South Africa, human cysticercosis is reported to be most prevalent in the Eastern Cape province, particularly in the poor, rural areas of the former Ciskei and Transkei, and parts of KwaZulu-Natal, where limited employment and impaired access to clean drinking water, proper sanitation, electricity and medical services equate to challenging socio-economic problems.²⁴ In addition, in these regions, poor methods of pig husbandry and lack of proper meat inspection and disease control measures are often encountered, thus exacerbating disease progression.²⁵ Small-scale subsistence pig farming is popular in the Eastern Cape, and usually pork is the cheapest meat available. Cultural practices and human behavioural patterns also promote dissemination of the disease.²

In India, cysticerci are known to infest the eye more often than in South Africa, while in China, skeletal muscles or
subcutaneous tissue are affected more frequently. South Africa and Latin America report having more multiple intracranial lesions than Asian countries. Spinal cord infestations remain a rarity, both locally and internationally, and involvement of the spinal cord has been reported to occur in 1-5% of NCC cases.

Spinal cysticercosis can present either leptomeningeally, in which the response is similar to that in subarachnoid disease, or intramedullary. The leptomeningeal type is more common. From 1978-1998, fewer than 50 confirmed cases of intramedullary cysticercosis, which presented with either paraplegia or quadriplegia, were documented worldwide. The number of infected individuals is gradually escalating in both developing and developed countries, as well as among human immunodeficiency virus (HIV) positive patients. Since almost all cysticercosis-infected people live in poor socio-economic conditions, a high possibility exists that a patient with HIV/acquired immune deficiency syndrome (AIDS) might concurrently be affected by cysticercosis.

Case report

A 46-year-old female was referred for neurosurgical evaluation by a private general practitioner from a small town outside Bloemfontein. She reported severe back pain radiating to the pelvic region and legs. The pain was associated with spasms and weakness of the lower limbs. The onset of the symptoms was sudden, and often accompanied by cramp-like sensations. Symptoms started three months prior to admission, but later subsided. The pain improved with movement, and became worse with rest. It was intermittent, with each attack lasting a day or two, but never clearing totally.

She reported no systemic symptoms, except sudden weight loss. She had no paraesthesia and there was no subjective sensory involvement. At her physical examination, she couldn’t walk due to the spasms and weakness associated with the pain. She reported that the pain had worsened and become unbearable over the preceding three days. She had experienced similar episodes in 2005, prior to her initial surgery to remove the spinal cysts.

The patient was diagnosed with hypertension three months prior to admission. She underwent back surgery, during which a laminectomy was carried out to evacuate the cysts, and took acetaminophen for pain control. The patient had a family history of hypertension and cerebrovascular accidents, and had been inhaling dry tobacco snuff for more than 10 years, although she didn’t consume alcohol. She had two children, worked as a domestic worker, and lived in a four-room shack with no basic sanitation.

On physical examination, the patient was alert and conscious. She appeared cachectic and restless. She was well hydrated and no lymphadenopathy was observed. Her blood pressure was 169/80 mmHg, pulse rate 80 beats/minute, respiratory rate 22 breaths/minute, temperature 36.5°C, and she had an oxygen saturation (SaO₂) of 96%. Orientation was normal for person, time and place, and communication, emotional state and intellectual function were intact. Cranial nerves, funduscopv and visual field examination were normal. On motor examination of the limbs, the muscle bulk showed general wasting with normal tone and power of 5/5 for both upper limbs, but 3/5 for the lower limbs. Both ankles had a power of 2/5 with medial deviation, and slight stocking sensory deficit for light touch, pain, vibration and proprioception. The left knee joint demonstrated obvious crepitations. The patient displayed no cerebellar signs or meningism. Examination of other systems proved to be unremarkable.

The differential diagnosis included:

- Recurrence of previous spinal cysticercus infestation
- Spinal cord tumor, e.g. meningioma, neurofibroma
- Extrapulmonary tuberculosis
- Pathological fracture, due to metastatic diseases or osteoporosis
- Spinal cord compression, due to spondylolisthesis
- Spinal cord trauma
- Musculoskeletal problems
- Neuropraxia
- Conversion disorder
- Frontal lobe lesion of the brain
- Spinal vasculitis.

Special preoperative medication included:

- Intravenous dexamethasone 8 mg eight hourly
- Hydrochlorothiazide 12.5 mg daily per os
- Enalapril 5 mg daily per os
- Acetaminophen 1 g six hourly, when necessary, per os
- Amitriptyline 25 mg nocte.

Table I: Laboratory investigation findings

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Patient’s result</th>
<th>Normal rangea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neutrophil count</td>
<td>1.77 x 10⁹ cells/l</td>
<td>2.00-10.00 x 10⁹ cells/l</td>
</tr>
<tr>
<td>Akaikne phosphatase</td>
<td>138 U/l</td>
<td>40-120 U/l</td>
</tr>
<tr>
<td>Lactate dehydrogenase</td>
<td>220 U/l</td>
<td>100-190 U/l</td>
</tr>
<tr>
<td>Erythrocyte sedimentation rate</td>
<td>40 mm/hour</td>
<td>0-20 mm/hour</td>
</tr>
<tr>
<td>HIV serology</td>
<td>Positive</td>
<td>Negative</td>
</tr>
<tr>
<td>CD4 count</td>
<td>46 x 10⁶ cells/l</td>
<td>500-2 010 x 10⁶ cells/l</td>
</tr>
<tr>
<td>Red cell morphology</td>
<td>Rouleaux formation</td>
<td>–</td>
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</tbody>
</table>

**Notes:**

- a: normal values used by the local National Health Laboratory Service (NHLS) diagnostic laboratories
- b: normal range for females 0-50 years of age
A laminectomy at T12 was carried out under general anaesthesia. No complications occurred intraoperatively. Five oval cysts were extracted from the subarachnoid space and spinal medulla at position T12-L2. The cysts had thin walls, and were translucent with a whitish tinge. Yamshidi needle biopsy of the vertebral body was performed, and cysts and tissue specimens obtained for histological investigations. Histology results reported cysts consistent with cysticerci. Inflammatory cells, connective tissue and a lining with foamy and multinucleated histiocytes were seen in the specimens. Ziehl-Neelsen stain was negative. Based on the histological findings, the diagnosis of NCC with surrounding immune reaction was made.

Postoperative medication in the ward was similar to the preoperative regimen, except that oral albendazole 15 mg/kg/day in three divided doses was added for its antiparasitic effects. The patient’s clinical condition improved because of the decompressive effect of evacuating the cysts. She reported a decrease in pains and spasms of her lower limbs. Muscle power in the limbs remained unchanged, and she still could not walk.

**Discussion**

Patients with NCC may present with seizures, encephalopathy, obstructive hydrocephalus, meningoencephalitis, cranial nerve palsy, hyperactive reflexes, visual field defects, nystagmus, papilloedema, cerebrovascular accidents, chronic headaches, nausea and vomiting, and changes in mental status. Seizures or headaches are the most common presentation of NCC, and are often due to brain parenchymal involvement. The cysticerci are known for their compression effects on surrounding structures, as well as their cytotoxic effects due to the release of toxins when they eventually die. The individual’s immune system and the consequent inflammatory response mainly determine parenchymal disease symptomatology. Involved calcified lesions from dead cysts may be epileptogenic foci, and thus lead to considerable morbidity.

Extraparenchymal ventricular and subarachnoid cysts are an alternative presentation of NCC with a much poorer prognosis, due to the resulting obstructive hydrocephalus that requires neurosurgical intervention. The cysts can become enormous in the basilar cisterns or Sylvian fissures before becoming symptomatic. Otherwise, particularly for those in the cisterns, this may lead to vasculitis and stroke. Spinal NCC remains a rare finding, and invasion of the adjacent vertebral body can be regarded as an exceptionally rare condition. NCC with a fatal outcome is generally uncommon, and is likely to be caused by complications of hydrocephalus, cerebral oedema, stroke and encephalitis.

**Medical therapy and prophylaxis**, with either praziquantel or albendazole, are recommended. Praziquantel is the medication of choice for intraparenchymal NCC. Albendazole is the drug of choice in cases of non-response to praziquantel, and is highly recommended in intraventricular NCC without intraparenchymal cysts. Most patients require urgent neurosurgical intervention for spinal cord decompression, but in resource-scarce areas, medical treatment can be a second best alternative.

The best management of this condition is prevention. The most important approach is education regarding possible contamination of drinking water, and the role of hygiene at both community and personal level.

Simultaneous diagnosis of HIV/AIDS and cysticercosis is an unsurprising finding, because of the gradual increase in the magnitude of HIV infection in regions where cysticercosis is endemic. It is unclear whether co-infection of the two diseases occurs by mere coincidence, or if HIV infection predisposes to cysticercosis. Of interest, is that the majority of NCC cases are known to have occurred in the late stages of HIV infection, which further strengthens the idea of a possible direct link between the two diseases. HIV/AIDS can modify the clinical presentation of NCC, causing it to manifest in either of the two opposite extremes; that is, either as a life-threatening condition, or as an incidental finding. The variation in clinical features is determined largely by the number, size, and location of CNS cysticerci, and the extent of the host’s inflammatory response.

In AIDS patients who have a compromised cell-mediated immunity, NCC may present silently, without a considerable inflammatory response in the host. The sensitivity of antibody detection is often lowered in these patients, because of a weakened immune system. The symptomatology of cysticercosis is mainly driven by the host's inflammatory response. Decreased cell-mediated immunity from advanced HIV infection often leads to an asymptomatic presentation of NCC.

Uncontrolled parasitic growth, due to a defective immune
response associated with HIV infection, may also result in the recurrence of NCC, and this could be particularly damaging, especially in delicate structures such as the brain and spinal cord. In this regard, HIV can fuel the destructive potential of NCC. Interestingly, NCC presenting with giant cysts and racemose forms appearing like clusters of grapes in the basal subarachnoid space, seems to be much more common in HIV-infected patients.12

Longstanding parasitic infection with *T. solium* suppresses the cellular immune response required to prevent HIV infection. The parasite achieves this effect by inhibiting complement and lymphocyte activation and cytokine production, as well as by surviving longer in HIV patients.4 Cysticercosis can also contribute toward the development of AIDS in HIV-infected patients by negatively interfering with the immune system.5

**Conclusion**

In practice, cysticercosis is frequently observed, with an escalating occurrence.4 This increase can be attributed to factors such as:

- HIV/AIDS4
- Poor socio-economic conditions4
- Immigration of individuals from endemic areas1,2
- Lack of awareness and education5
- Poor methods of pig husbandry.5

Contrary to what Foyaca-Sibat et al postulated,4 cysticercosis does not seem to be limited to the Eastern Cape and KwaZulu-Natal regions of South Africa exclusively, as high rates are also observed in the Free State. It is expected that patients in other provinces could be having cysticercosis foci,2 with general widespread distribution throughout the country, which poses a research challenge with regard to the true extent of this disease.

**References**