Extradural guinea-worm abscess

Report of two cases

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Two cases are reported of extradural abscess from guinea-worm infection, one that resulted in paraplegia, and the other in motor weakness of the legs.

KEY WORDS • extradural abscess • guinea worm • paraplegia

Infection by *Dracunculus medinensis* is a major cause of morbidity in certain parts of tropical Africa and Asia. An extremely high incidence of infection in both children and adults has been reported from various parts of West Africa. However, despite the prevalence of this infection in endemic areas only six cases of extradural guinea-worm abscess have been reported between 1961 and 1970.

Clinical diagnosis of this complication is rarely possible and the prognosis is unpredictable. Early surgical intervention may be rewarding, but the existence of vascular involvement may determine the final outcome. We are reporting our experience with two such cases and the pathogenesis and diagnosis are discussed.

Case Reports

Case 1

This 8-year-old boy came from a village in a dry savanna area well known for its high incidence of guinea-worm infection. He had been completely well until 8 days before his admission when he had a sudden attack of abdominal pain associated with leg weakness; over the next hour he developed complete paralysis of both legs and loss of all sensation in the legs and lower half of the trunk. There was no past history of guinea-worm infection.

He was admitted the following day to a small rural hospital where he was catheterized for urine retention and treated with penicillin and streptomycin for fever. Three days later he developed marked neck pain and rigidity; on the eighth day after the onset of symptoms he was transferred to our hospital in Zaria.

Examination. On admission to this hospital the patient was found to have sensory loss below T-6 and a temperature of 104°F. The neck stiffness was causing some distress. The liver was enlarged by 3 cm, and the spleen was palpable 8 cm below the left costal margin. A provisional diagnosis of Burkitt's lymphoma was made.

Initial laboratory investigations revealed...
the following counts: hemoglobin, 10 gm%; total white cells, 7300; neutrophils 67%; eosinophils 1%; lymphocytes, 29%; and monocytes 3%. Blood urea was 10 mg% and serum electrolytes were normal. Malaria parasites were not seen in the peripheral blood, but the bone marrow contained malaria pigment and an excess of eosinophils. Cerebrospinal fluid (CSF) showed 10 lymphocytes, protein 360 mg% and glucose 23 mg%. No Burkitt's cells were demonstrated. A lumbar myelogram showed a block at T-10 (Fig. 1).

Since no definite cause had been established for paraplegia, and in view of the myelographic findings, a laminectomy was performed 12 days after the onset of symptoms.

Operation. The laminae of T5-10 were removed. There was a tough pyogenic membrane in the extradural space with two abscess cavities containing pus under tension. In the lower and larger abscess, the head of a guinea worm was seen protruding after the pus had been sucked out. The worm itself was found coiled up in a confined space below the level of the T-10 lamina. It was extracted without difficulty, and was found to be alive; it measured 70 cm in length (Fig. 2).

A specimen of pus was collected for microscopic and bacteriological examination. The upper abscess cavity, obviously multiloculated, was excised, but no other worm was found. The abscess wall was closely adherent to the dura mater and could be separated with difficulty. No pulsations of the dura mater were seen even after the evacuation and excision of the abscess cavities. Apart from generalized hyperemia of the dura mater, no other gross pathological lesion was demonstrated. The wound was closed in layers and a vacuum drain left in place.

Histological Examination. The abscess cavity contained acute and chronic inflammatory cells. The wall consisted of fibrous, fatty tissue infiltrated by chronic inflammatory cells and eosinophils, lined by granulation tissue and numerous extraneous giant cells (Fig. 3). This finding is common in a case of guinea-worm abscess.

Microscopic examination of the pus after gram staining revealed myriads of larvae of Dracunculus medinensis and numerous polymorphonuclear leucocytes (Fig. 4). The pus was cultured and reported sterile.

Postoperative Course. Postoperative recovery was uneventful, although there was no improvement in the neurological state. Mark-
Extradural guinea-worm abscess

Withdrawal reflex was noted in the legs a few days after surgery and this persisted. Tendon reflexes were brisk and plantar reflexes extensor. The patient was treated with a course of niridazole, 500 mg twice daily, and chloramphenicol, 250 mg every 6 hours for a week.

His pyrexia initially subsided but then recurred, associated with urinary infection by *Pseudomonas pyocyanea*, which proved resistant to all available antibiotics. A postoperative cisternal myelogram showed no mechanical block. His condition continued to deteriorate, with uncontrolled urinary infection. He died 10 weeks after the operation; permission could not be obtained for autopsy.

Case 2

A 25-year-old farmer was seen in the emergency unit on June 1, 1975. He had been injured by a falling sack of grain and had backache and weakness of the legs.

**Examination.** The patient had a cauda equina lesion, and extreme motor weakness of the legs without any sensory loss. While waiting for myelogram he became incontinent of urine and feces. During the second week of his hospital stay, a guinea worm emerged through a peripheral lesion on the right leg. Blood examination at that time revealed peripheral eosinophilia.

**Operation.** We performed an immediate laminectomy of T-11 through L-2. A single thick-walled abscess (7 x 2 cm) containing a 70 cm-long live guinea worm was excised.

**Postoperative Course.** The patient's recovery has been good. He is fully continent of urine and feces and walks with only a slight degree of residual motor weakness.

**Discussion**

Although infection by guinea worm is common in West Africa, extradural abscess resulting from it is apparently rare. The

![Fig. 3. Case 1. Photomicrograph of the abscess wall, showing extraneous giant cell reaction and infiltration with chronic inflammatory cells. H & E, x 450.](image1)

![Fig. 4. Case 1. Photomicrograph of pus smear showing numerous *Dracunculus medinensis* larvae and inflammatory cells. Gram's stain, x 175.](image2)
parasite can migrate freely throughout the tissue planes of the retroperitoneal spaces and may set up a chronic abscess at any site, normally with only minimal symptoms. The pathway by which the worm enters the spinal canal remains obscure.

Although the inflammatory process is a low-grade infection, the paraplegia is usually sudden in onset and may be due to ischemia. This fact is supported by the existence of a critical zone of vascular anastomosis at the level of T4–8 (the most common site of abscess formation) and by the lack of recovery from paralysis following laminectomy in more than half of the patients reported.

The prognosis is not directly related to the duration of illness. Analysis of previous reports showed that three out of six patients had some degree of recovery from their paraplegia following laminectomy.

A clinical diagnosis of extradural guinea-worm abscess is rarely made, and it is not possible to define any characteristic or distinguishing feature which differentiates this form of extradural abscess from any other. In most of the reported cases the diagnosis was established at laminectomy. This alone is a reasonable justification for early surgical exploration in all cases of undiagnosed paraplegia in the tropics.

Similar presentation of paraplegia, particularly in children, may be due to Burkitt's lymphoma, tuberculosis, pyogenic infections, and schistosomiasis, all of which may be difficult to diagnose on clinical grounds. Possible factors leading to recognition of this syndrome may be peripheral eosinophilia or eosinophilic hyperplasia of the bone marrow, previous infestation with guinea worm, the presence of extraspinal lesions, and most important, paravertebral calcification seen on films, indicating a dead guinea worm.

The purpose of this communication is to stimulate awareness of the possibility of such a diagnosis, since many patients with obscure paraplegia or leg weakness in the tropics are never diagnosed.

Addendum

We have recently treated two more patients with paraplegia due to guinea-worm infection. One of them was diagnosed preoperatively and recovered after laminectomy, while the other did not show any postoperative recovery.

References

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