Acanthameba Meningoencephalitis: A Case Report


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Summary

A 45 year old lady presented with history of recent surgery for uterovaginal prolapse and retained vaginal tampons following which she developed chronic meningitis due to acanthameba infection. Patient responded to a regimen containing albendazole. She was left with hydrocephalus as a sequelae.

Key words: Acanthameba, Meningoencephalitis, Tampons, Albendazole.

Introduction

Acanthameba meningoencephalitis (AME) is an insidious onset slowly progressive infection of the central nervous system caused by acathamebae. Although the infection occurs mostly in immuno compromised patients like AIDS, immunocompetent individuals are affected as well.1 The usual portal of entry of the infection is through the skin or upper respiratory tract and it reaches the central nervous system through hematogenous route.2 Various drugs have been tried in the treatment of this condition but with very little therapeutic success. A case of AME in an immunocompetent individual, in whom a new therapeutic regime containing albendazole was tried, is reported here.

Case Report

A 45 year old apparently healthy woman underwent Manchester repair for uterovaginal prolapse in a local hospital. A week after discharge from the hospital she developed high grade fever with generalized malaise accompanied by foul smelling vaginal discharge. Patient then presented to a second hospital where per vaginal examination revealed three left over tampons. These were removed and the patient was given a course of oral ciprofloxacin and metronidazole. Vaginal discharge subsided but the patient continued to have fever with malaise. As the symptoms worsened, she presented to a third hospital where she was extensively investigated for fever. At this stage, clinical examination remained unremarkable except for signs of meningeal irritation. Laboratory investigations including complete blood counts, peripheral blood smear, urine examination, blood cultures and liver function tests were normal. Serological studies like rheumatoid factor, antinuclear antibody, widal, ELISA for HIV, HbsAg and brucella antigen were negative. Bone marrow aspiration and trephine biopsy showed normal hematopoiesis with no significant abnormality. Extensive imaging studies performed including roentgenographic studies of
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As the patient could not afford treatment in that hospital she presented to our institute. Clinical examination revealed a conscious, well oriented woman with neck stiffness, bilateral lateral rectus palsy and papilledema. A repeat CSF study confirmed acanthamebae in both wet mount and giemsa staining. However, the organism could not be cultured. HIV serology was repeated and found negative. CT scan of the head was normal.

Specific therapy was started with rifampicin 600 mg once daily and cotrimoxazole 960 mg twice daily orally and fluconazole 200 mg once daily intravenously. After one week of the above therapy, her temperature showed a downward trend from 103°F to 100°F gradually but she developed episodes of complex partial seizures. Intravenous ceftriaxone was added at a dose of two grams 12 hourly and carbamazepline 200 mg three time a day, was introduced to control seizures. High grade fever persisted, even after four days of antimeningitic dose of intravenous ceftriaxone. A course of Tab. albendazole was added on empirical basis, in a dosage of 200 mg twice daily. Simultaneously rifampicin and cotrimoxazole were withdrawn. After 10 days of therapy with albendazole along with ceftriaxone and fluconazole, her temperature touched the baseline and continued to remain so. Once the patient became afebrile, fluconazole and ceftriaxone were stopped, while albendazole and carbamazepline were continued for a period of four weeks.

She remained afebrile and seizure free for initial two weeks, but developed headache, blurring of vision and recurrent episodes of complex partial seizures later. An interictal EEG taken at this point showed non-specific epileptiform discharges and CSF study showed raised protein level, occasional lymphocytes but no organism could be isolated. Gabapentine was added for the control of complex partial seizures. Intracranial hypertension was managed using a course of dexamethasone, along with mannitol and frusemide. There was immediate symptomatic improvement. However, the patient developed right upper motor neuron seventh nerve palsy and urinary incontinence. CT scan of the head at this stage revealed an obstructive hydrocephalus. She was referred to a neurosurgical unit, where she underwent a ventriculo-peritoneal shunt surgery. On one year follow up, her symptoms of urinary incontinence and blurring of vision had improved while she was being maintained on phenytoin 100 mg twice daily.

Discussion

Acanthameba produces subacute to chronic meningoencephalitis. Clinical manifestations range from focal neuro deficit to problems of cognition and even coma. The diagnosis of AME is best confirmed by smear preparation and culture. In the present case, acanthamoeba species could not be isolated by culture probably because she had received antimicrobial agents before she presented to our institute. However, acanthameba trophozoites with characteristic needlelike acanthapodia and large karyosome were seen in both wet mount and giemsa stained preparation. Some investigators feel that careful assessment of morphology, mitoses and motility are adequate criteria for identifying the various genera.3

AME is an infection occurring mostly in immunocompromised individuals, with very few case reports in immunocompetent individuals.4,5 No obvious immunodeficiency states were identified in our patient. Thus, she is probably one of the few immunocompetent individuals in whom this infection has occurred. Foreign body retention, indwelling catheters and in-situ prosthesis can be possible predisposing factors to this infections.6 Retained, vaginal tampon as a source of AME have not been reported. Thus this may be the first case report of accidentally left over vaginal tampons being associated with this infection. She had evidence of chronic meningitis with clinical and EEG evidence of seizure activity suggesting encephalitis. No effective drug regimen has been recommended so far, for the treatment of AME. Several in-vitro studies to map out a sensitivity pattern to the organism have not shown any drug to have persistent amebicidal activity. In an in-vitro study involving several antifungal and
Antiprotozoal agents, only 5-fluorocytosine and hydroxystilbamidine were shown to have amebicidal effect. In human infection, drugs like intravenous pentamidine, rifampicin, cotrimoxazole and fluconazole have been used with variable success. In the present case, we have tried albendazole, along with other previously used drugs such as cotrimoxazole, rifampicin, fluconazole and ceftriaxone. Albendazole, an imidazole derivative with a broad spectrum activity against helminthic parasites, was introduced based on its proven efficacy in neurocysticercosis which again produces a chronic, indolent granulomatous meningoencephalitis.

Several issues in therapy require to be addressed at this juncture. After introduction of albendazole to the existing regimen the patient remained afebrile and repeat CSF examination failed to reveal the organism. However, the therapeutic efficacy of albendazole in our patient remains speculative for two reasons. Firstly albendazole was introduced into the regimen almost after two weeks therapy with other agents. Secondly, it was given along with ceftriaxone and fluconazole for ten days before continuing it as monotherapy. Hence the efficacy of albendazole against acanthamoeba merits further clinical evaluation. In vitro studies are required to prove the amebicidal effect of albendazole to substantiate our findings.

AME has a high fatality rate. Most of the immunocompetent patients who have survived the infection have developed some sequelae. The present case is among the few individuals who have survived the infection with development of hydrocephalus as a complication. AME has been increasingly recognized in immunocompetent individuals. It is necessary to have a high index of suspicion to recognize this infection, especially when CSF examination is negative for routine organisms and conventional anti-meningitis therapy has failed.

References


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