

Psychiatric manifestations of toxocara

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Psychiatric patients are more likely than the general population to be at risk from toxocara, due to behavioural and social disturbance. Here, the authors discuss a case that highlights that it is prudent to consider atypical pathogens in patients presenting with an unexplained high eosinophil count, or if there has been a history of homelessness, farm work or contact with animals.

We present a particularly complicated case of a male patient who became psychotic with marked cognitive decline during an acute toxocara infection. He was a homeless man, estimated to be in his mid-to-late forties, and was found to be acting strangely whilst on remand in prison. He claimed to have been homeless for several years and happily so, with no significant psychiatric or medical history and no history of alcohol or substance abuse.

Following various psychiatric assessments in prison, the patient was transferred to a psychiatric intensive care unit (PICU) in Somerset under the Mental Health Act.

Toxocara

The genus of helminths, toxocara, is ubiquitous amongst domestic dogs, cats and wild animals such as foxes, all of which act as hosts.^{1,2} Humans can become accidental (paratenic) hosts when they ingest toxocara larvae, often from soil contaminated by faeces or from unwashed vegetables or undercooked meat. These larvae are unable to develop into mature worms

Sample date	ELISA toxocara IgM serum levels (normal range <0.844)	Western blot
28/07/15	2.006	5 bands
15/10/15	1.555	5 bands

within a human, but may migrate around the body.

In most cases toxocariasis is asymptomatic as the larvae are unable to reproduce and subsequently die, but local inflammation can lead to distinct syndromes, depending on where in the body the larvae reside. In rare cases these larvae may migrate to the central nervous system where they may cause a constellation of neurological symptoms, including meningoencephalitis, focal deficits and seizures.^{3,4}

Studies have estimated seroprevalence in humans to be between 35%–42% in hot and humid agricultural regions (2%–5% in urban areas) of more economically developed countries, and even higher in less economically developed countries.⁵ In one study conducted in Turkey, toxocara seroprevalence was much higher in schizophrenic patients (45.9%) than in the general population (2%).⁶ In another, eosinophilia in peripheral blood was detected in 61.9% of seropositive schizophrenic patients; this is significantly different to the non-psychiatric population ($p=0.0015$).⁷

Thus far there are few, if any, reports of toxocariasis causing psychiatric symptoms.⁷ Several studies have highlighted the increased seroprevalence of antitoxocara antibodies in psychiatric populations, particularly schizophrenia, although it is uncertain whether

this is a causative effect, or secondary to this population's increased likelihood to behave strangely or be homeless. Recently, there has been interest in the possible association between neurotoxocariasis and an immune-mediated dementia.^{4,5,8}

Presentation

The patient was on remand at a prison when staff noticed a series of bizarre behaviours including: drinking from (and soaking his clothes in) the toilet bowl; destroying his cell's furniture; and self-injurious behaviour including banging his head against his cell's walls or scratching at his eyes.

When questioned about his injuries, he would deny them or respond with fantastical confabulations such as being trampled by a herd of cows.

Mental state examination revealed no signs of any mood disturbance or signs of psychosis, other than the possible social drift and isolation seen in chronic schizophrenia. With a differential diagnosis of learning difficulties or Ganser's syndrome, he was transferred to our psychiatric hospital for further assessment.

Findings from assessment and investigations on a PICU

In addition to the above, we noted the following:

1. A significant cognitive deficit,

including retrograde amnesia, particularly for personal details such as his name, childhood and date of birth. A mini-mental state examination (MMSE) scored him at 12/30 and Addenbrooke's Cognitive Examination (ACE-III) highlighted particular deficits in orientation and visuospatial domains. Despite this, there was no evidence of any anterograde amnesia (other than the confabulations).

2. There was no evidence of the approximate answers associated with Ganser's syndrome.

3. Physical examination revealed marked clubbing and an elongated skull with prominent facial features, but was otherwise normal.

4. Blood tests highlighted a persistent eosinophilia (22% of white cell count), so a stool culture and serological screen for parasites were sent to the lab for analysis. All other blood tests, including B12 and folate levels, HIV, syphilis, NMDA receptor, voltage-gated K⁺ channel and various other autoantibodies, were normal. A blood sample was sent for a genetic array, but also came back negative.

5. An MRI of his head showed small, equivocal frontotemporal lobe degeneration and an incidental finding of empty sella syndrome, but was otherwise normal. Hormone levels were normal so empty sella syndrome was discounted as a contributing factor.

A full neurological assessment highlighted a possible diagnosis of frontotemporal or Korsakoff's dementia, perhaps secondary to the poor diet. He was started on thiamine and other vitamin replacement therapy.

Change in presentation

After several months on the wards, our patient deteriorated rapidly, becoming far more agitated and confused. He also occasionally complained of visual hallucinations of a 'red man'. On several occasions he needed secluding and was treated

with intramuscular haloperidol or lorazepam.

A diagnosis of toxocariasis was made after both western blot and ELISA returned positive results for high titres of toxocara IgM antibodies, indicating an acute infection (see Table 1). The patient was therefore started on albendazole.

Within a month, he had improved markedly: his MMSE had risen to 29/30, the psychosis and behavioural problems disappeared and the eosinophilia normalised. His ACE-III results also improved dramatically in every domain, although deficits persisted both in visuospatial perception and frontal lobe function. On interaction, he seemed superficially competent, if a little odd, but on closer inspection his memory remained impaired to the extent that he struggled with various activities of daily living. His subsequent social rehabilitation has been slow, yet we have not seen a re-emergence of any psychotic phenomena

Discussion

Toxocariasis is under-diagnosed due to non-specific symptoms and poor awareness of its epidemiology,⁵ and is more common in schizophrenic patients and those that have spent a lot of time in rural settings^{2,6,8}. It is therefore the responsibility of the clinician to consider it as part of a differential diagnosis.

This particular patient proved a challenge as we were unable to ascertain a complete history due to his cognitive deficit and lack of family or GP to provide a collateral history. Subsequent investigations via the police provided us with contact details for his sister, who revealed a history suggesting a schizophreniform psychotic illness. but during assessment the patient had fled to begin his nomadic lifestyle of homelessness and self-neglect. As such, it is impossible to ascertain when he became infected

with toxocara. We did see a remarkable improvement in his cognitive function after treatment with albendazole, but not complete. This may have been secondary to a toxocara-induced delirium superimposed upon a pre-existing dementia or psychotic illness. His MRI head did not show any evidence of cerebral larvae, but it was taken before any positive psychotic symptoms evolved. Larvae are mobile, and are not always visible on imaging. Ideally, diagnosis of neurotoxocariasis is confirmed with biopsy or analysis of CSF,^{8,9} but it was not deemed appropriate to perform a lumbar puncture when he was so agitated.

Declaration of interest

No conflicts of interest were declared.

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