Tumour-like presentation of central nervous system tuberculosis: A retrospective study in Kingdom of Saudi Arabia

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Abstract

Objective: To differentiate the tumour-like presentation of central nervous system (CNS) tuberculosis (TB) from CNS tumours.

Methods: We conducted a retrospective chart review of all cases of CNS TB seen at King Abdulaziz Medical City, Jeddah, between January 2002 and January 2012. No

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Introduction

Although tuberculosis (TB) is a curable disease, it is the world’s second commonest cause of death from infectious disease, the first being infection with human immune deficiency virus (HIV). The World Health Organization (WHO) estimated that nearly one third (1.9 billion people) of all the people in the world are infected with Mycobacterium tuberculosis, and it has probably killed more than 100 million people over the past 100 years. It is endemic in most developing countries and is resurgent in countries with high rates of HIV infection.

In the Kingdom of Saudi Arabia, according to WHO, the incidence of smear-positive TB and deaths from TB each rose by 6.2% between 1990 and 2004. Although 85% of cases of TB occur in the lungs, 15% occur outside the respiratory system, the central nervous system (CNS) being the second commonest site of involvement. CNS TB accounts for approximately 1% of all of disease caused by M. tuberculosis and carries the worst prognosis of any other form. Tuberculous meningitis, the commonest form of CNS TB, may result in hydrocephalus, brain infarction and death if left untreated. CNS TB can present either as diffuse forms, such as basal exudative meningitis, or as localized forms, such as tuberculoma, abscess or cerebritis.

Tuberculomas are the commonest manifestation of parenchymal TB. They can occur at any age and can be solitary or multiple. Tuberculomas may affect the brain (meningeal, parenchymal or ependymal), spinal cord, subarachnoid, subdural or epidural space. Cerebral tuberculomas can occur anywhere in the brain parenchyma but are usually located at the corticomedullary junction and pteriventricular region, as expected for haematogenous dissemination. In children, they predominate in the infratentorial compartment, whereas in adults, the supratentorial compartment is more commonly affected. Rarely, they are found within the ventricle (the lateral ventricle being the commonest site), the cavernous sinus, the sella turcica, the hypophysis, the hypothalamus, the sphenoid sinus or mastoid air cells. The frontal and parietal lobes are the most commonly affected regions, especially on the left, probably due to greater blood flow to the dominant hemisphere. Tuberculomas arise when tubercles in the parenchyma of the brain enlarge without rupturing into the subarachnoid space. They usually occur in the absence of meningitis, but they may coexist because of extension of cerebrospinal fluid infection into the adjacent parenchyma via cortical veins or Virchow-Robin spaces.

Tuberculomas consist of epithelioid cells and giant cells mixed with lymphocytic inflammatory cells, forming a non-caseating granuloma. It subsequently develops a central area of caseating necrosis, which is initially solid and may liquefy later.

Patients usually present with headache, seizures, fever, focal neurological deficit and features of raised intracranial pressure. Infratentorial tuberculomas may present with brainstem syndromes, cerebellar symptoms and multiple cranial nerve palsies. Clinical findings suggestive of extraneural TB are frequently subtle or absent, and fewer than 50% of patients have a history of TB. In patients with CNS tuberculoma, typical cerebrospinal fluid findings might be absent, and immunological studies and adenosine deaminase are neither specific nor sensitive; culture and PCR are specific but not sensitive.

Tuberculomas can present as space-occupying lesions that are indistinguishable from brain neoplasms. Although cancer treatments are frequently toxic, the risk of toxic effects is justified by the potential gains in survival seen when the appropriate treatment is assigned to the right patient. The aim of the study reported here was to find signs to differentiate CNS tuberculomas from brain tumours by reviewing all cases seen at our institute over a 10-year period.

Materials and Methods

We conducted a retrospective chart review of all cases of CNS TB seen at the King Abdulaziz Medical City, Jeddah, between January 2002 and January 2012. A total of 125 patients were identified. While most presented with either tuberculous meningitis or tuberculoma, nine (7.2%) presented with clinical and radiological features suggestive of a brain tumour. A diagnosis was established either intraoperatively from frozen sections (three patients) or postoperatively when the masses were resected (six patients).

Results

No symptoms or signs of pulmonary or systemic TB were found. One patient died, and another patient developed a severe neurological deficit. The remaining patients recovered after receiving anti-TB treatment. None of the cases was tested
for HIV infection, even after the diagnosis was established. Magnetic resonance spectroscopy (MRS) was used for only one patient, and the result suggested a diagnosis of brain tumour rather than CNS TB. Less favourable outcomes were seen in an elderly patient, in cases of posterior fossa TB and in patients who underwent complete resection of the mass. Table 1 shows the clinical and radiological features and outcomes of the nine cases.

**Patient 1**

A 27-year-old male presented with headache of 8 months' duration, which was of moderate severity and felt mostly over the occipital region; the pain responded to over-the-counter medication. He had no history of vomiting, seizures, loss of consciousness, fever, weight loss, cough, weakness or sensory symptoms. He also complained of diminished vision in his both eyes and drowsiness that started 1 month before presentation. He had sought medical attention because of the visual symptoms. He had type 2 diabetes mellitus, which was being treated with metformin and gliclazide. His uncle had been treated for TB a long time previously. On physical examination, the patient was found to be obese, with a blood pressure of 150/70 mm Hg and normal temperature. His higher mental functions were normal. He had decreased visual acuity in both eyes (6/60 bilaterally), with right homonymous hemianopia. Fundoscopy showed bilateral chronic papilloedema. The remaining cranial nerves were normal. Motor, sensory and coordination examinations were normal. There was no sign of meningeal irritation. Computed tomography (CT) of the brain revealed a left occipital hypodense lesion measuring 3.5 x 3.5 cm with surrounding oedema, enhanced after contrast, which was highly suggestive of a meningioma. There was a midline shift of 1.1 cm, with compression of the left lateral ventricle. MRI of the brain confirmed the findings of the CT scan (Figure 1), and MRS was highly suggestive of a tumour (Figure 2). Complete blood count, renal function and electrolyte tests were normal. The erythrocyte sedimentation rate was 55 mm during the first hour, and the C-reactive protein value was 30. A chest X-ray was normal. After his blood sugar was controlled, he received dexamethasone and was prepared for surgery 3 weeks later for a possible biopsy. The lesion was found to be intra-axial with outside attachment to the dura. A frozen section indicated a tuberculoma, which was totally excised. Histopathological examination showed a necrotizing granulomatous lesion (Figure 3), which was positive for acid-fast bacilli. The infectious disease team reviewed the patient and started him on a four-drug anti-TB regimen. He was found to be positive for *M. tuberculosis* and was fully sensitive to the medications. On follow up, the patient was found to have improved markedly, and after 2 months, ethambutol and pyrazamide were discontinued; he was advised to continue with rifampicin and isoniazid to complete the course of 1 year.

**Patient 2**

A 62-year-old male presented with headache, confusion, slurred speech and gait disturbance of 2 weeks’ duration. He had no history of fever, cough or weight loss but was a known case of type 2 diabetes mellitus and hypertension, which were being treated. He had no family history of exposure to TB.

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (years)/sex</th>
<th>Clinical presentation</th>
<th>Neuroimaging</th>
<th>Tumour location</th>
<th>Type of surgery</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>27/M</td>
<td>Headache, diminished vision, drowsiness</td>
<td>Left occipital region</td>
<td>Left occipital hypodense lesion with surrounding oedema</td>
<td>Frozen section and total excision</td>
<td>Full recovery</td>
</tr>
<tr>
<td>2</td>
<td>62/M</td>
<td>Headache, confusion, dysarthria, gait disturbance, left-side weakness</td>
<td>Left fronto-parietal region</td>
<td>Left fronto-parietal mass with surrounding oedema and midline shift</td>
<td>Total excision</td>
<td>Improved, with residual mild expressive aphasia</td>
</tr>
<tr>
<td>3</td>
<td>75 F</td>
<td>Nausea, gait disturbance, left-side weakness, facial twitch, right-side weakness</td>
<td>Right cerebellar region</td>
<td>Right cerebellar mass with surrounding oedema and hydrocephalus</td>
<td>Biopsy</td>
<td>Full recovery</td>
</tr>
<tr>
<td>4</td>
<td>72 M</td>
<td>Facial twitch, left-side weakness</td>
<td>Right parieto-occipital region</td>
<td>Right parieto-occipital, ring-enhancing nodule with well-defined margins and surrounding oedema</td>
<td>Biopsy</td>
<td>Full recovery</td>
</tr>
<tr>
<td>5</td>
<td>82 M</td>
<td>Vomiting, dizziness, unsteady gait</td>
<td>Right cerebellar region</td>
<td>Right cerebellar mass with enhancement and surrounding oedema</td>
<td>Total excision</td>
<td>Death</td>
</tr>
<tr>
<td>6</td>
<td>41 M</td>
<td>Headache, dizziness, vomiting</td>
<td>Right fronto-parietal region</td>
<td>Right fronto-parietal mass with surrounding oedema</td>
<td>Total excision</td>
<td>Dense left hemiplegia with refractory seizures</td>
</tr>
<tr>
<td>7</td>
<td>14 F</td>
<td>Headache</td>
<td>Right fronto-parietal region</td>
<td>Right fronto-parietal mass</td>
<td>Biopsy</td>
<td>Full recovery</td>
</tr>
<tr>
<td>8</td>
<td>50 F</td>
<td>Facial twitch, seizures, visual impairment</td>
<td>Right fronto-parietal region</td>
<td>Right fronto-parietal mass</td>
<td>Total excision</td>
<td>Full recovery</td>
</tr>
<tr>
<td>9</td>
<td>26 M</td>
<td>Headache, dizziness, vomiting</td>
<td>Right fronto-parietal region</td>
<td>Right fronto-parietal mass</td>
<td>Biopsy</td>
<td>Full recovery</td>
</tr>
</tbody>
</table>

**Table 1: Clinical and radiological features of the nine cases of central nervous system tuberculoma.
Physical examination revealed right hemiparesis and expressive aphasia. MRI of the brain revealed a left frontoparietal mass with surrounding oedema and a midline shift, which was diagnosed as a glioma multiforme. He was treated with steroids.

Figure 1: MRI of the brain of patient 1. T2-weighted (A) and FLAIR (B) images show high signal intensity in the left temporo-parieto-occipital region with surrounding oedema. Post-gadolinium axial (C) and sagittal (D) views show enhancement.

Figure 2: MRS of patient 1 is highly suggestive of a tumour.
After 3 days, he was sent to surgery, and the mass was resected. Histopathological examination revealed a tuberculoma. He was started on anti-TB treatment and phenytoin. He improved clinically and was discharged after 6 weeks. He completed the prescribed course of treatment and was examined clinically 6 months later. He had no neurological deficit, apart from mild expressive aphasia.

Patient 3

A 75-year-old woman presented with a 5-day history of nausea, gait disturbance and left side weakness. She had no history of fever, headache or seizures. The only significant feature of her medical history was hypertension. On physical examination, her consciousness level was found to be depressed, with a Glasgow coma stage score of 7/15, flexing all her limbs to painful stimuli and the left side moving less than the right. Her blood pressure was 180/86 mm Hg, her temperature was normal, and no cranial nerve palsy was apparent. Both plantars were upgoing. CT scan of the brain showed a large hypodense area covering both cerebellar hemispheres and the vermis, with a mass effect on the fourth ventricle and the brainstem, mainly on the right side. MRI of the brain revealed a right cerebellar mass with surrounding oedema and hydrocephalus. A biopsy from the cerebellar mass was consistent with a tuberculoma. The patient was started on anti-TB treatment and made a complete recovery.

Patient 4

A 72-year-old man presented with a 4-month history of recurrent episodes of twitching on the left side of the face. One month previously, he had noted weakness of the left side of the body involving the face, arm and leg. CT of the brain at a local hospital showed a hypodense area in the region of the right middle cerebral artery, suggestive of infarction. MRI of the brain revealed a right parieto-occipital ring-enhancing nodule with well-defined margins and surrounding oedema, suggesting a glioblastoma multiforme. A biopsy from the cerebellar mass was consistent with a tuberculoma. The patient was started on anti-TB treatment and made a complete recovery.

Patient 5

An 82-year-old man was admitted from the emergency room complaining of vomiting, dizziness and unsteady gait of 1 month’s duration. He had been operated on at another hospital 6 months previously to remove a cerebellar mass, which was reported to be a low-grade glioma. On physical examination, he was found to have cerebellar signs and ataxic gait. CT of the brain was repeated and showed a right cerebellar mass with enhancement and surrounding oedema. He underwent craniotomy and excision of the cerebellar mass. Histopathology revealed a tuberculoma, and anti-TB treatment was started. Unfortunately, he developed multiple postoperative complications, including subgial collection at the surgery site, hospital-acquired pneumonia, acute respiratory distress syndrome and multiorgan failure, and died after 52 days.

Patient 6

A 41-year-old man presented with progressive headache of 4 months’ duration, which had increased in severity and was associated with dizziness and vomiting. He was admitted to the local hospital, and CT of the brain revealed a left frontal lesion (4 × 3.7 cm). Although a biopsy was performed, the result was inconclusive, and the patient was referred to our hospital. He had no medical history of significant illness. Physical examination revealed a Glasgow coma scale score of 11/15 and right-side hemiparesis. Biopsy was repeated, and the results were consistent with CNS TB. Treatment with anti-TB drugs markedly improved his neurological status. By the time of discharge, 1 month after starting treatment, he was mobile with support and was independent in his daily activities. He followed the course of anti-TB treatment for 18 months and had apparently complete recovery. A follow-up MRI of the brain showed regression of the lesion to 1.6 × 1.9 cm.

Patient 7

A 14-year-old girl presented with a 4-week history of progressive headache that did not respond to analgesics. She had no history of fever, weight loss or seizures, and her medical history was unremarkable. She had no history of contact with TB. Systemic and neurological examinations and routine blood investigations and chest radiography were normal. CT and MRI of the brain showed a right fronto-parietal mass with...
surrounding oedema, suggesting a brain tumour. Her family elected to take her abroad for a second opinion and further management. She underwent complete resection of the mass, and histopathology showed TB, and she was started on anti-TB treatment. Unfortunately, she developed left-side dense hemiplegia and refractory seizures and is now taking three antiepileptic drugs.

**Patient 8**

A 50-year-old woman presented with a 3-month history of twitching movements on the right side of her face, which had worsened during the past 2 weeks. She had no constitutional symptoms, and her medical history was unremarkable. MRI of the brain was performed at a local hospital, where the diagnosis was a brain tumour. She was started on tegretol and dexamethasone and came to our hospital for further management. General and neurological examinations were normal. MRI of the brain at our hospital revealed a left frontal mass measuring $2 \times 2$ cm with surrounding oedema, suggestive of a high-grade glioma with metastases. Work-up for systemic malignancy and infections, including TB, was negative. Four days later, she underwent craniotomy and total resection of the mass. She had aphasia immediately post-operatively, which subsequently improved. Histopathology and culture were consistent with TB. She completed a course of 12 months of anti-TB treatment and had recovered completely when seen 18 months later.

**Patient 9**

A 26-year-old man with no known chronic illness came to the neurosurgical clinic complaining of progressive headache of 1 year’s duration, associated with two episodes of tonic-clonic seizures and decreased visual acuity within the previous 2 weeks. He had no history of weakness, fever, weight loss or night sweating. Physical examination revealed normal vital signs and higher mental function but decreased visual acuity and bilateral chronic papilloedema. MRI of the brain showed a right tempo-parietal enhancing lesion with surrounding oedema causing a mass effect (Figure 4). He was started on dexamethasone and phenytoin and one week later underwent right tempo-parietal craniotomy and total excision of the mass, with no complications. Frozen section and histopathology reports confirmed a diagnosis of tuberculoma. He was started on anti-TB therapy after assessment and evaluation by the infectious disease team. He improved gradually and was completely normal 6 months later.

**Discussion**

On CT, tuberculomas are either round or lobulated, with a variable appearance, and may appear hypo- or hypodense to the brain parenchyma. Caseating granulomas show homogeneous contrast enhancement enhanced peripherally.8,9 Moderate-to-marked peri-lesional oedema is frequent. In rare cases, a ring-enhancing lesion with a hypodense centre may reveal central calcification. This is known as the ‘target sign’ and, although considered characteristic of tuberculoma by some authors, others consider it to be nonspecific and therefore leading to erroneous diagnoses.13

MRI findings of a tuberculoma vary according to the stage and location of the lesion. They appear as isointense to grey matter on T1-weighted images and may have a slightly hyperintense rim. Noncaseating lesions are bright on T2-weighted images, with nodular enhancement. Caseating tuberculomas vary from isointense to hypointense on T2-weighted images and also exhibit rim enhancement. They may have a variable degree of mass effect and perilesional oedema, which is usually more prominent in the early stages.8,9,14

Although MRS may be helpful in differentiating tuberculomas from other space-occupying lesions, they are commonly indistinguishable from brain tumours or infections such as neurocysticercosis. MRS shows a prominent decrease in the ratio of $N$-acetylaspartate to creatine and a slight decrease in that of $N$-acetylaspartate to choline. The choline:creatinine ratio is $>1$ in tuberculomas,15,16 with a high peak of lipids, more choline and less $N$-acetylaspartate and creatine.

In 2011, 8.7 million people developed TB worldwide. Of those, 1.1 million (13%) were HIV-positive, and 79% were in the African Region.12 In 2004, WHO issued recommendations on the interventions required to prevent, diagnose and treat TB in people living with HIV, including testing TB patients for HIV, providing antiretroviral therapy and co-trimoxazole preventive therapy to TB/HIV patients and

![Figure 4](image_url)

**Figure 4:** MRI of the brain of patient 9. FLAIR images (A) show high signal intensity in the right parieto-occipital region with surrounding oedema. Post-gadolinium axial (B) and sagittal (C) views show enhancement.
providing HIV prevention services for TB patients.\textsuperscript{12} It also included intensifying TB case-finding among people living with HIV, offering isoniazid preventive therapy to people living with HIV who do not have active TB, and controlling the spread of TB infection. Unfortunately, none of our patients were tested for HIV during the initial work-up.

Biopsy remains the gold standard for diagnosing CNS tuberculomas. In managing patients at high risk for other infectious processes or malignancies or those infected with HIV, there should be a low threshold for brain biopsy for a definitive diagnosis. In patients who live in or have emigrated from areas where TB is endemic, early stereotactic needle brain biopsy may be unnecessary, and empirical therapy may be a reasonable option. In developed countries, where TB is less common, a more aggressive approach is probably appropriate, unless clinical suspicion is high. Because of the reduced surgical risk, stereotactic brain biopsy is preferred over craniotomy. The overall complication rate of stereotactic biopsy is 0.6–6.3%. When stereotactic biopsy is coupled with paraffin sectioning, the diagnostic efficacy reaches 85%.\textsuperscript{17}

Surgical treatment of patients with tuberculous meningitis is directed to the hydrocephalus and tuberculobrain abscesses. Ventrículo-peritoneal or ventriculo-atrial shunts are permanent measures, which relieve the signs and symptoms of hydrocephalus and significantly improve the consciousness level and neurological deficits.\textsuperscript{18} However, these shunts may require replacement because of blockage by the high protein content of the cerebrospinal fluid. Early shunting in combination with drug therapy offers the best therapeutic outcome.

A single intracranial tuberculoma causing a midline shift and increased intracranial pressure that fails to respond to chemotherapy should be removed surgically.\textsuperscript{19} In chronic adhesive spinal arachnoiditis, the cerebrospinal fluid loculations may cause cord compression, necessitating surgical intervention.

Before the development of effective anti-TB therapy, surgical treatment was the only modality available for treating patients with intracranial tuberculomas. It was associated with 10% operative mortality and 40% additional mortality due to postoperative meningitis.\textsuperscript{20} There have been no randomized controlled trials to determine which anti-TB regimen is the most effective in treating CNS tuberculomas.\textsuperscript{21} In the USA, the recommended initial therapy is isoniazid, rifampicin, ethambutol and pyrazinamide until susceptibility to these drugs is known. If the tubercle bacillus is sensitive, three-drug therapy with isoniazid, rifampicin and pyrazinamide should be given for 2 months. Pyrazinamide is then stopped, and isoniazid and rifampicin are continued for the rest of the treatment. It is recommended that CNS tuberculomas be treated for a minimum of 1 year, or longer if they have developed or progressed despite treatment.\textsuperscript{22}

The only study in which surgery was compared with drug therapy for intracranial tuberculomas is that of Harder and colleagues.\textsuperscript{23} In their nonrandomized, uncontrolled trial, 20 cases of intracranial tuberculoma were reviewed. Half of the patients had been treated with isoniazid, ethambutol and rifampicin, while the other half had undergone excision of the tuberculoma, followed by the same drug regimen. All the patients treated with drugs showed resolution of their tuberculomas by 1 year of treatment, and all patients who underwent excision were stable by 6 months; however, patients given drugs were more likely to show improvement or a return to baseline on a semi-quantitative disability score. The study has several limitations, including the small size of the sample and lack of randomization; however, others have supported these findings. Most experts in the field of CNS TB management therefore continue to recommend medical management as the preferred treatment for intracranial tuberculomas, reserving surgical intervention for patients who require decompression or biopsy for definitive diagnosis.

Paradoxical enlargement of a pre-existing tuberculoma or the appearance of new intracranial or spinal tuberculoma in patients receiving effective anti-TB therapy is a well-known phenomenon.\textsuperscript{24} This immune reconstitution inflammatory syndrome is thought to be the result of an immunological reaction, which has variably been attributed to a combination of the release of new antigen targets during mycobacterial killing, hypersensitivity to such antigens and exaggerated immune restoration (after TB-induced immunosuppression). The lesions are usually found on routine follow-up scans or when new neurological signs develop during medical therapy. In addition, this phenomenon may develop in patients with undiagnosed resistance to rifampicin. Drug susceptibility testing (preferably a rapid diagnostic assay) should therefore be performed in all patients presenting with a paradoxical reaction. Simultaneous steroid administration probably prevents these focal lesions, although tuberculomas usually resolve with continuation of anti-TB therapy. In case of unresponsiveness to medical therapy, surgery is recommended. Fortunately, none of our patients developed this complication of therapy.

**Table 2: Factors that suggest a non-neoplastic diagnosis in a patient with a space-occupying lesion.**

| Disease onset in adolescents and young adults (AIDS and other infectious or inflammatory diseases) |
| History of contact with febrile patients or patients with active tuberculosis |
| History of travelling to endemic areas (cysticercosis, hydatidosis, amoebiosis) |
| Intravenous drug addiction or sexual risk behaviour (AIDS, syphilis, brain abscess) |
| Personal or family history of autoimmune or inflammatory diseases (multiple sclerosis, Behçet disease, sarcoidosis) |
| Recent dental procedure, dental abscess or ear, nose and throat infections (brain abscess) |
| Immunosuppression, including diabetes (opportunistic infection) |
| History of recurrent or transient neurological deficits affecting multiple areas in the neuroaxis (multiple sclerosis) |
| History of oral and genital ulcers and uveites (Behçet disease, syphilis) |
| Presence of oral candidiasis (AIDS, immunosuppression) |
| Presence of skin rashes (Behçet disease, sarcoidosis, AIDS) |
| Abnormalities on extra-cranial imaging (sarcoidosis, tuberculosis, fungal infections, cysticercosis) |
| Good control of systemic cancer and absence of lung metastases argues against brain metastases in patients with a history of cancer |
In developing countries in which TB is endemic, CNS mass lesions caused by TB account for 1.4–8% of all space-occupying lesions,\textsuperscript{25,26} while they account for only 0.2% of all such lesions in industrialized regions. In the absence of extra-cranial disease, the diagnosis of intracranial TB is difficult and challenging. Table 2 lists clinical factors that raise the possibility of a non-neoplastic diagnosis.

\section*{Conclusion}

CNS TB is a great mimicker of brain tumours, especially in endemic areas, where there should be a high index of suspicion. The diagnosis is based on clinical presentation and the results of investigations. In patients with CNS TB who present with a mass lesion, a biopsy should be taken to avoid the morbidity and mortality associated with unnecessary surgical interventions.

MRS should be used more widely in evaluating brain-space occupying lesions, and CNS TB should be included in the differential diagnosis of such lesions in all endemic areas, including the Kingdom of Saudi Arabia. The absence of constitutional or pulmonary symptoms of TB is deceptive, and HIV testing should be done routinely in all cases of suspected CNS TB. More nationwide epidemiological studies are required to establish guidelines for the early detection and successful outcome of this condition.

\section*{Conflict of interest}

None declared.

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