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Ischaemic Stroke Due to Schistosoma haematobium (Cerebral Neuroschistosomiasis): A Case Report in a Young Man

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Abstract

Schistosomiasis is a major neglected tropical disease; neurological involvement (neuroschistosomiasis) is uncommon, and cerebral disease is classically associated with *Schistosoma japonicum*, whereas *S. haematobium* more often causes urinary and spinal disease. Cerebral neuroschistosomiasis presenting as ischaemic stroke in *S. haematobium* infection is rare. We report a 22-year-old man, originally from Senegal and resident in the United Kingdom, who presented with sudden-onset left-sided weakness, sensory symptoms, dysarthria, poor balance and headache two days after onset (National Institutes of Health Stroke Scale score 5, improving to 3 at 24 hours). MRI confirmed an acute right thalamic infarct with true restricted diffusion, and CT/MRI intracranial angiography demonstrated a multifocal vasculopathy; a comprehensive autoimmune vasculitis screen was negative. Admission bloods showed a peripheral eosinophilia of 1.4 $\times 10^9$/L (reference 0-0.5 $\times 10^9$/L). Prompted by haematuria and his prior residence in an endemic area, urine microscopy confirmed *S. haematobium* ova, serum schistosomal enzyme-linked immunosorbent assay (ELISA) was positive, and cystoscopy showed a characteristic bladder "sandy patch." Expert neuroradiology review felt the MRI and MRA head were in keeping with cerebral neuroschistosomiasis, and a follow-up contrast MRI head and spine showed a nodular enhancing thalamic lesion with perilesional oedema, also in keeping with cerebral neuroschistosomiasis. This lesion had shown some regression prior to commencement of treatment. Following multidisciplinary input, corticosteroid cover was given before praziquantel, with empirical ivermectin to cover *Strongyloides*; the patient continued to improve clinically, with no further neurological events over 12 months of follow-up. A repeat MRI head and MRA head at six weeks showed stable vasculopathy with maturation of the thalamic infarct. Schistosomiasis should be considered in young patients with stroke from endemic areas, particularly with eosinophilia or a vasculitic angiographic pattern, and corticosteroid cover before praziquantel should be considered when central nervous system involvement is suspected.

Categories: Neurology, Radiology, Infectious Disease
Keywords: neurology, neuroschistosomiasis, praziquantel treatment, stroke, vasculitis

Introduction

Schistosomiasis (bilharziasis) is a chronic parasitic infection caused by blood flukes of the genus *Schistosoma*. It remains one of the most prevalent neglected tropical diseases, affecting more than 200 million people worldwide, with several hundred million more at risk across endemic regions of sub-Saharan Africa, the Middle East, South America, the Caribbean and Asia [1]. The World Health Organisation (WHO) estimates that in 2024, 253.7 million people required preventive treatment [2]. *Schistosoma haematobium* is the principal cause of urinary schistosomiasis and is highly endemic across much of sub-Saharan Africa, including Senegal [3]; it classically produces terminal haematuria and chronic bladder wall changes.

Neurological involvement is a recognised but comparatively uncommon manifestation of schistosomal infection. Cerebral disease is classically attributed to *S. japonicum*, while *S. mansoni* and *S. haematobium* more frequently cause spinal cord disease [4]. Cerebral vasculitis has also been reported in association with schistosomiasis, although a causal relationship has not been firmly established [5,6]. Cerebral neuroschistosomiasis due to *S. haematobium*, with ischaemic stroke as its presenting feature, is unusual. We report a young man from an endemic region in whom an acute ischaemic stroke led to the diagnosis of urinary schistosomiasis with cerebral neuroschistosomiasis and an associated intracranial vasculopathy.

Case Presentation

History

A 22-year-old man, originally from Senegal and resident in the United Kingdom for the preceding few years, was brought in by ambulance with sudden-onset left-sided weakness and numbness that had begun two

days prior, associated with difficulty speaking, headache and poor balance. He was previously well, with no significant medical history and no major cardiovascular risk factors. On direct questioning, he reported intermittent terminal haematuria over the preceding few months, each episode lasting several days before spontaneously settling. While resident in Senegal, the patient reported frequently swimming in freshwater lakes.

Examination

Neurological examination revealed a left hemiparesis with associated sensory disturbance, dysarthria and cerebellar signs of dysdiadochokinesia and past-pointing in the left arm that was out of proportion to the extent of limb weakness. The National Institutes of Health Stroke Scale (NIHSS) score was 5 on admission and improved to 3 at 24 hours [7]. Vital signs were within normal limits, and respiratory, cardiovascular and abdominal examinations were normal.

Investigations

Neuroimaging

Non-contrast CT of the head showed a subtle hypodensity involving the right thalamus and posterior limb of the internal capsule. MRI confirmed an acute infarct of the right posterior thalamus with true diffusion restriction (Figure *1*) and no evidence of haemorrhagic transformation on susceptibility-weighted imaging. CT angiography showed multifocal intracranial luminal narrowing involving the right terminal internal carotid artery, the distal right middle cerebral artery, the right P2 segment, and the proximal right superior cerebellar artery; the findings were atypical for dissection, with vasculitis primarily suspected. MR angiography confirmed a multifocal vasculopathy with arterial wall thickening but no definite enhancement, including narrowing of the right P2 segment (Figure *2A*, *2B*) and of the bilateral A2 segments of the anterior cerebral arteries (Figure *2C*, *2D*); the right P2 stenosis was the segment most likely responsible for the inferolateral thalamic infarct, and atheroma and vasculitis were retained as differentials. The patient was commenced on dual antiplatelet therapy.

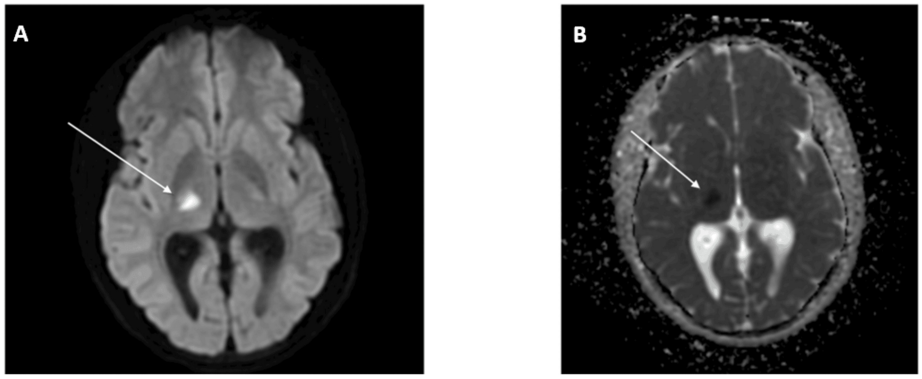


FIGURE 1: Diffusion-weighted MRI of the brain at presentation. (A) Axial diffusion-weighted image showing an acute infarct in the right posterior thalamus with high signal (arrow). (B) The corresponding apparent diffusion coefficient (ADC) map shows low signal at the same site (arrow), confirming true restricted diffusion and excluding T2 shine through.

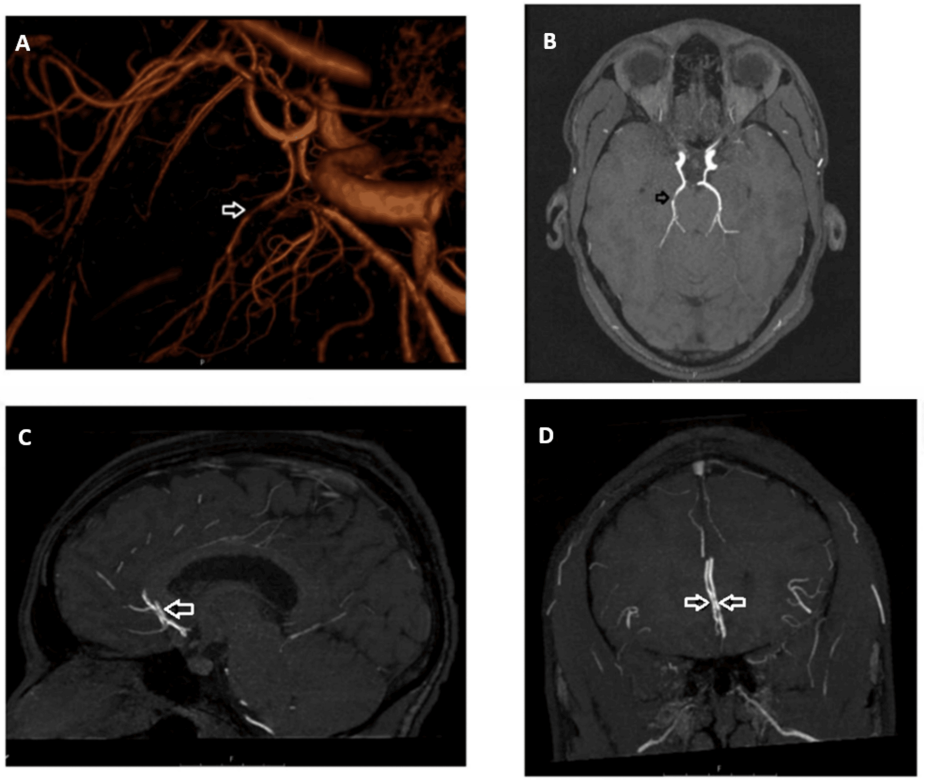


FIGURE 2: Intracranial angiographic imaging demonstrating the multifocal vasculopathy. (A) Three-dimensional volume-rendered CT angiogram showing focal narrowing of the right P2 segment of the posterior cerebral artery (arrow). (B) Axial MR angiogram (maximum intensity projection) confirming narrowing of the right P2 segment (arrow). (C) Sagittal and (D) coronal MR angiograms demonstrating narrowing of the bilateral A2 segments of the anterior cerebral arteries (arrows).

Laboratory

Peripheral eosinophilia was 1.4 ×10^9/L (reference 0-0.5 ×10^9/L). Cholesterol was slightly elevated, but this was felt unlikely to be responsible for the extent of the vasculopathy (Table *1*). Inflammatory markers were normal (Table *1*). A comprehensive autoimmune and vasculitis screen was unremarkable: proteinase 3 (PR3) and myeloperoxidase (MPO) antibodies were negative on two occasions, and antinuclear antibody, complement levels, immunoglobulins, serum protein electrophoresis and an antiphospholipid screen were all negative or within normal limits (Table *1*). The rheumatology team advised that the clinical and serological picture was not in keeping with a systemic vasculitis. The presence of eosinophilia in a patient from an endemic region with haematuria prompted suspicion of underlying schistosomiasis.

Investigation Category	Test	Value	Reference range
Full blood count	White blood cell count (x 10^9/L)	5.1	4-10
	Haemoglobin (g/L)	155	130-170
	Eosinophil count (x 10^9/L)	1.4	0-0.5
Biochemistry	C-reactive protein (mg/L)	<1	0-5
	Sodium (mmol/L)	138	133-146
	Potassium (mmol/L)	4	3.5-5.3
	Haemoglobin A1c (mmol/mol)	36	20-41

	Cholesterol serum (mmol/L)	5.1	0-5
Renal function	Creatinine (umol/L)	96	59-104
	eGFR (mL/minute)	>90	>90
Auto-immune + vasculitis screen	PR3 antibodies (kunits/L)	<0.6, <0.6	0-2
	MPO antibodies (kunits/L)	0.2, 0.2	0-3.5
	ANA antibody (CTD screen)	0.1	0-1
	Anti-cardiolipin antibody	Negative	N/A
	Complement C3 component (g/L)	1.05	0.75-1.65
	Complement C4 component (g/L)	0.26	0.14-0.54
	Immunoglobulin A (g/L)	1.36	0.8-4
	Immunoglobulin G (g/L)	14.7	5.5-16.5
	Immunoglobulin M (g/L)	1.09	0.4-2
	Serum protein electrophoresis	No abnormal bands	N/A
	Lupus anticoagulant screen	Negative	N/A
	Smooth muscle IgG antibody	Negative	N/A
	Mitochondrial antibody	Negative	N/A
	Liver/kidney microsomal antibody	Negative	N/A
	Rheumatoid factor (kunits/L)	<10	0-20
	Haemoglobinopathy screen	Negative	N/A
Microbiology, viral serology and parasitology	Bartonella serology	Negative	N/A
	Cysticercosis antibody	Negative	N/A
	Histoplasma antibody	Negative	N/A
	Schistosomal antibody	**Positive**	N/A
	Strongyloides antibody	Negative	N/A
	Toxoplasma gondii antibody	Negative	N/A
	Syphilis IgG/IgM	Negative	N/A
	CMV IgG/IgM	Negative	N/A
	EBV IgG/IgM	Negative	N/A
	Hepatitis B surface antigen	Negative	N/A
	Hepatitis C IgG	Negative	N/A
	HIV 1 and 2 antibody	Negative	N/A
	VZV IgG (mIU/ml)	>150 (In keeping with immunity)	N/A
	Lyme disease antibody	Negative	N/A
	TB interferon gamma assay	Negative	N/A
Urinary investigations	**Urine microscopy**	**Ova of S. haematobium seen**	N/A
	Urine culture	No growth	N/A

TABLE 1: Blood and Urinary Investigations.

ANA: antinuclear antibody; CMV: cytomegalovirus; CTD: connective tissue disease; EBV: Epstein-Barr virus; eGFR: estimated glomerular filtration rate; HIV: human immunodeficiency virus; IgG: immunoglobulin G; IgM: immunoglobulin M; MPO: myeloperoxidase; PR3: proteinase 3; S. Haematobium: Schistosoma haematobium; TB: tuberculosis; VZV: varicella-zoster virus.

Microbiology and Parasitology

Urine microscopy was positive for *Schistosoma haematobium* ova, and serum schistosomal antibody testing (enzyme-linked immunosorbent assay (ELISA)) was positive (Table *1*). A broad infection screen was otherwise negative, including serology for *Strongyloides*, Bartonella, cysticercosis, Lyme disease and toxoplasma, an interferon-gamma release assay for tuberculosis, and testing for HIV, hepatitis B surface antigen, hepatitis C, cytomegalovirus and Epstein-Barr virus (Table *1*).

Renal and Urological

CT intravenous urography demonstrated calcification of the bladder dome, an ectopic left kidney lying in the pelvis, splenomegaly (13 cm) and no radio-opaque stones (Figure *3*). Flexible cystoscopy revealed a classical "sandy patch" lesion (3 × 2 cm) on the posterior bladder wall.

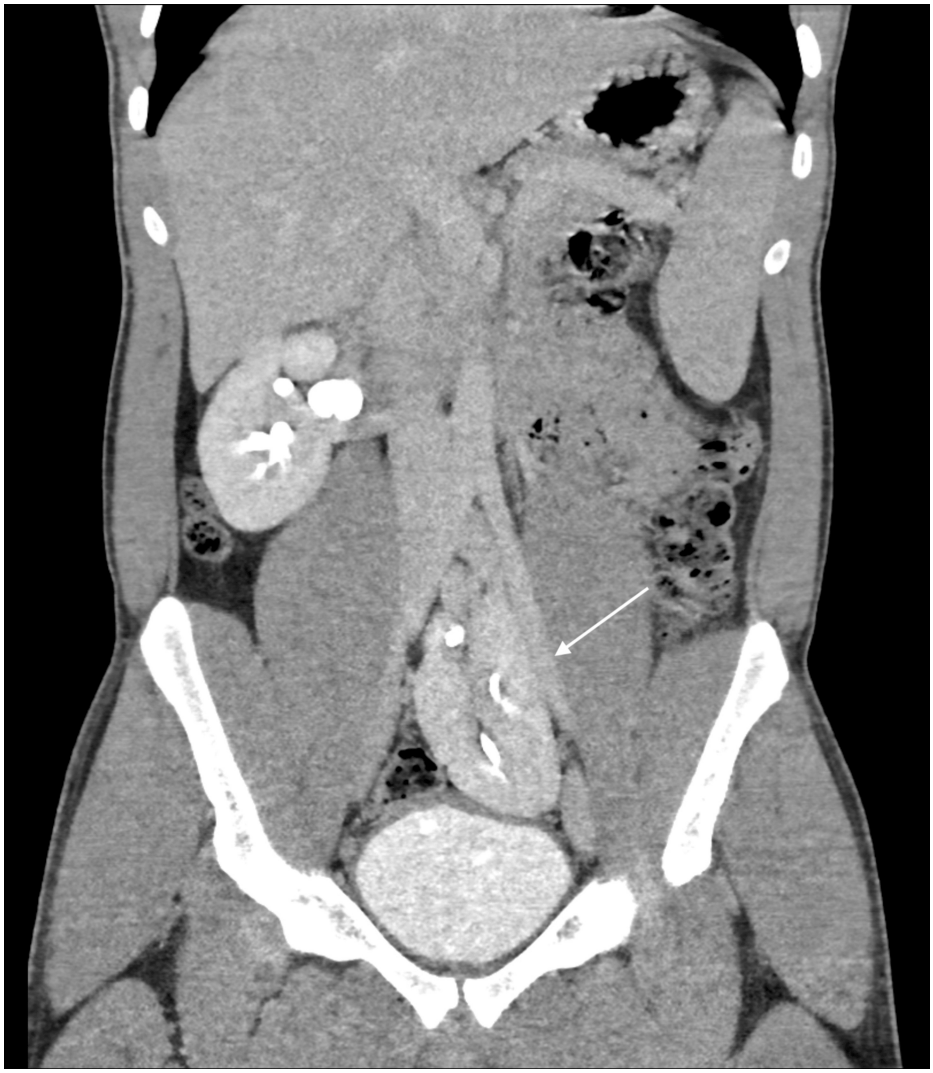


FIGURE 3: CT intravenous urography (IVU) demonstrating an ectopic left kidney lying within the pelvis (arrow).

Cardiac

Transthoracic echocardiography demonstrated a trivial left-to-right interatrial shunt suggestive of a patent foramen ovale, which could not be fully excluded on transthoracic imaging alone; left ventricular size, wall thickness and systolic function were normal (ejection fraction 58%), with no intracardiac thrombus. A subsequent outpatient bubble contrast study was negative. A 72-hour Holter showed no evidence of atrial fibrillation.

Diagnosis

The findings were consistent with confirmed urinary schistosomiasis with probable cerebral neuroschistosomiasis. On review by a neuroradiologist with an infectious diseases interest, the presence of a multifocal vasculopathy with ischaemic stroke on the MRI and MRA head was considered compatible with cerebral neuroschistosomiasis, and correlation with cerebrospinal fluid analysis, serum antibody ELISA, and urine and stool microscopy, together with contrast-enhanced MRI of the head and whole spine, was recommended.

Management

Following multidisciplinary discussion involving the stroke, infectious diseases, urology, rheumatology and neuroradiology teams, corticosteroids were prioritised before antiparasitic therapy. This sequence reflects the recognised risk that praziquantel may precipitate severe, occasionally life-threatening inflammatory deterioration in acute neuroschistosomiasis [4]. The patient received oral prednisolone in a tapering regimen over six weeks (commencing at 60 mg daily and reducing weekly), followed by praziquantel 20 mg/kg twice daily for three days, started during the steroid taper. A single empirical dose of ivermectin was given alongside the corticosteroid to cover possible *Strongyloides* co-infection while awaiting serology, given the risk of hyperinfection with immunosuppression [8].

Outcome and follow-up

A contrast-enhanced MRI of the brain and whole spine performed prior to commencement of treatment demonstrated a nodular enhancing lesion in the right thalamus with surrounding oedema that had reduced in both size and degree of enhancement compared with the earlier MRI studies; the appearances remained consistent with neuroschistosomiasis and were interpreted as showing interval improvement (Figure *4*). There were no new lesions or obstructive hydrocephalus, and the spinal cord appeared normal, with no pathological cord, conus, or cauda equina enhancement. A further MRI head and MRA head performed at six weeks from treatment commencement showed no new acute infarct, maturation of the thalamic infarct, and stable vasculopathy (Figure *5*). Follow-up blood tests at six weeks showed a normalisation of the eosinophilia (0.5 $x10^9$/L) and repeat urinary microscopy post-treatment showed no schistosomal ova. The patient made a good functional recovery and remained well, with no clinical recurrence over 12 months of follow-up.

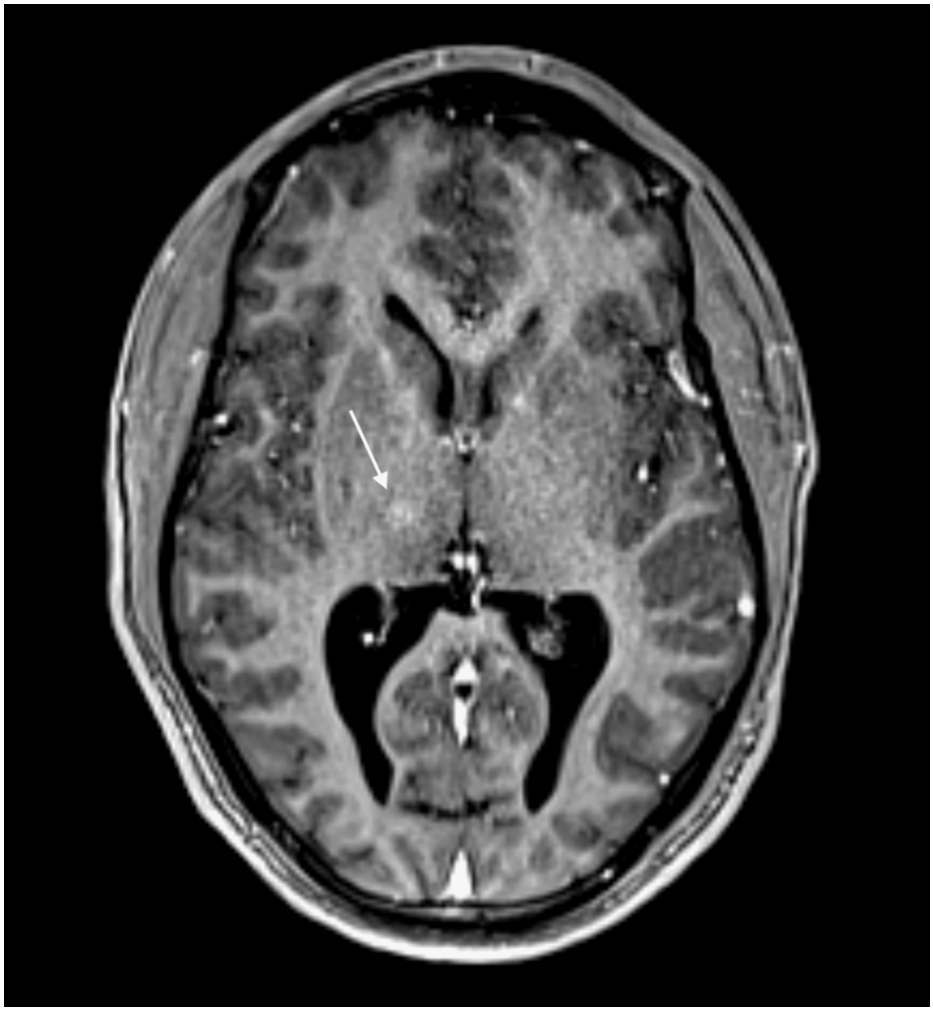


FIGURE 4: Intracranial MRI head with gadolinium performed prior to commencement of treatment. Axial view post-contrast demonstrating the nodular enhancing lesion in the right thalamus (arrow).

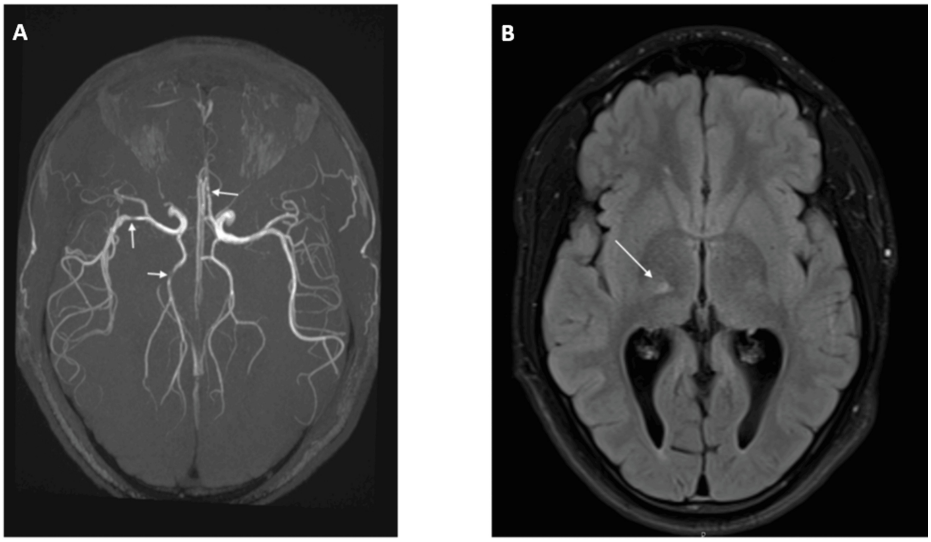


FIGURE 5: Intracranial MRI and MRA Head performed six weeks following treatment commencement. (A) Axial view of MR angiogram showing the stable multifocal vasculopathy affecting the right posterior cerebral artery, right middle cerebral artery and bilateral anterior cerebral arteries (arrows). (B) Axial view of MRI head in fluid-attenuated inversion recovery sequence (FLAIR) demonstrating maturation of the right thalamic infarct (arrow).

Discussion

This case illustrates ischaemic stroke as the presenting feature of *S. haematobium* infection in a young adult from an endemic region, with subsequent diagnosis of cerebral neuroschistosomiasis. Two overlapping processes appear to be present: a granulomatous cerebral lesion - the nodular thalamic enhancement with perilesional oedema was felt by an infectious disease neuroradiologist to be in keeping with a neuroschistosomiasis granuloma rather than a bland infarct - and a multifocal intracranial vasculopathy, with the right P2 stenosis the segment most plausibly responsible for the thalamic infarct.

Several features support this association: the patient's origin in an endemic area, a history of freshwater swimming in Senegal, the absence of major stroke risk factors, peripheral eosinophilia, and an angiographic pattern consistent with vasculopathy, with active *S. haematobium* infection on urine microscopy and serology. The comprehensively negative autoimmune and vasculitis screen, together with the rheumatology assessment, argues against a primary systemic or CNS vasculitis and supports a parasitic aetiology.

Several mechanisms have been proposed for schistosomiasis-associated cerebral ischaemia, including haemodynamic compromise, direct injury to the arterial wall, obliterative endarteritis of the vasa vasorum, contiguous spread from adjacent inflamed tissue, and inflammatory intimal damage; embolisation of ova with a surrounding granulomatous reaction may also contribute [4]. The eosinophilia observed in this case is a recognised, often early, clue to the diagnosis. In a review of eight published cases of schistosomiasis-associated stroke, eosinophilia was the most frequent finding, with watershed infarction or cerebral vasculitis typical, mirroring the present case [9]. Notably, cerebral neuroschistosomiasis is classically associated with *S. japonicum* [4], so its occurrence here with *S. haematobium* is unusual.

Praziquantel is a broad-spectrum schistosomicidal agent active against adult worms and achieves parasitological cure in most treated patients [1]. In suspected acute neuroschistosomiasis, however, it may precipitate a paradoxical inflammatory reaction with clinical deterioration; corticosteroid cover before and during treatment is therefore advised, although the optimal regimen and timing are not firmly established [4]. Suggested steroid regimens include prednisolone 1 mg/kg/day with a slow taper over approximately one month [4]. *Strongyloides* co-infection should be excluded, or empirically covered, before corticosteroids are started, as immunosuppression can precipitate hyperinfection - the rationale for the empirical ivermectin given here [8].

The principal teaching point is that schistosomiasis should be included in the differential diagnosis of stroke in young patients who live in, or originate from, endemic areas - particularly when eosinophilia or a

vasculitic angiographic pattern is present. A history of haematuria, as in this case, may be the clue that unlocks the diagnosis.

Cerebrospinal fluid examination, although recommended to further support the diagnosis, was not undertaken. Furthermore, there was evidence of lesion regression and neurological improvement prior to treatment commencement, which may represent the natural history of a subacute infarct. The diagnosis of cerebral neuroschistosomiasis therefore rests on the imaging appearances, expert neuroradiology review, positive serum and urine studies and the comprehensive exclusion of other causes. As with any single case report, the mechanistic association between infection and stroke, while well supported, remains inferential.

Conclusions

Schistosomiasis is a prevalent tropical disease but a rare and treatable cause of ischaemic stroke and cerebral disease in young people from endemic regions. Haematuria, previous residence in an endemic country, and peripheral eosinophilia were key to the diagnosis in this case. Clinicians should maintain a low threshold for considering schistosomiasis, especially in the presence of vasculitic imaging, and should recognise that cerebral involvement can occur with *S. haematobium*. Excluding vasculitic and conventional cardiovascular causes remains an important part of the workup. If central nervous system involvement is suspected, consider corticosteroid cover before praziquantel, with empirical treatment or screening for *Strongyloides* co-infection, to reduce the risk of treatment-related deterioration.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Thomas H. Fluck, Mahim Verma, Farhad Huwez, Bader Mohamed

Drafting of the manuscript: Thomas H. Fluck, Mahim Verma

Critical review of the manuscript for important intellectual content: Mahim Verma, Ken Wong, Farhad Huwez, Bader Mohamed

Acquisition, analysis, or interpretation of data: Ken Wong

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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