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RARE PRESENTATION OF CENTRAL NERVOUS SYSTEM TUBERCULOSIS WITH BILATERAL ANTERIOR CEREBRAL ARTERY ANEURYSM: A CASE REPORT

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ABSTRACT
Central Nervous System tuberculosis (CNS TB) with intracranial aneurysms is rare and needs prompt diagnosis and early management. The presence of un-ruptured or ruptured aneurysms in the presence of CNS infection is difficult to treat in both acute settings and long-term management because of possible complications prevention. A female of 70 years of age presented with a history of fever, headache, vomiting, and altered sensorium for one week with GCS of 13/15, later developing paraparesis. She was diagnosed to have CNS tuberculosis with complicating bilateral anterior communicating artery (ACA) aneurysms. She was managed conservatively in our hospital and was referred for endovascular neurosurgical intervention for the aneurysms to another center because of non-availability in our setup. CNS infection with intracranial aneurysms is difficult to manage and such a combination of vascular complications of tuberculous meningitis must be kept in mind while dealing with such scenarios but carries a good prognosis if diagnosed and managed as early as possible to avoid complications.

KEYWORDS: CNS TB, Anterior communicating artery, Aneurysms

INTRODUCTION
With a global increase in tuberculosis incidence, prompt diagnosis and early treatment are needed. Especially in South Asian countries, the prevalence of this disease has a greater impact on the health care system. [1]Tuberculous meningitis is the most common but worst form of brain infection that needs long-term treatment with many complications. Complications include seizures, stroke, hydrocephalus, hearing loss, and psychiatric disturbances. Being a crippling disease, it has high morbidity and mortality. Untreated infection leads to disastrous complications of obstructive hydrocephalous and obliterative vasculitis.[2-4]

Stroke is the well-recognized complication of tuberculous meningitis but bilateral anterior cerebral infarction is rare.[5] It can be due to anomalous cerebral vessels or space-occupying lesions. Its association with brain infection is rare but few cases were reported. Here we are presenting a case of a female of 70 years of age having a history of pulmonary tuberculosis diagnosed as having CNS tuberculosis with bilateral anterior cerebral artery aneurysm.

CASE PRESENTATION
A female of 70 years of age with a history of pulmonary tuberculosis 50 years back and hypertension presented in emergency with a one-week history of high-grade fever, documented up to 101 degree F, intermittent associated with headache and vomiting, relieved by antipyretics. There was a history of dry cough on and off. Headache was occipital with moderate severity and was continuous, associated with nausea and vomiting, and was not relieved by analgesics. There was a neck pain that aggravated on movement. She developed altered sensorium in the last three days. There was a history of non-specific arthralgias and myalgias but no history of night sweats or weight loss.

On examination in emergency, her vital signs were blood pressure of 144/77 mmHg, pulse rate of 86 beats per minute, respiratory rate of 17 breaths/min and temperature of 100 degree F. Her random blood sugar levels were of 147 mg/dl. Glasgow Coma Scale (GCS) was 13 (E4V4M5). Pupils were bilaterally equal and reactive, the neck was supple, and no facial asymmetry was noted. Dilated fundoscopy showed hypertensive retinopathy. She was moving all four limbs spontaneously and plantar reflex were bilaterally withdrawal. There were no signs of meningeal irritation. Chest examination showed a thoraco-abdominal type of respiration with bilateral harsh vesicular breathing. Cardiovascular and abdominal examinations were unremarkable.

On preliminary investigations, the blood complete picture, serum chemistry including liver function test,

renal function and serum electrolytes, and coagulation profile were done and were normal (Table 1).

Hepatitis B and C screening were negative. Plain computed tomography brain showed left frontal lobe hypo-density. Anti-neutrophilic cytoplasmic antibody (ANA) was negative. The erythrocyte sedimentation rate (ESR) was 25mm/hr. Chest X-ray (CXR) showed bilateral infiltrates.

Table 1: Preliminary investigations

PARAMETERS	PATIENT LABS VALUES	NORMAL RANGE
1. ***BLOOD COMPLETE PICTURE***		
WBC	8410/micro-litre	4000-10,000/microliter
HB	13 g/dl	12-15 g/dl
PLATELET COUNT	220,000/ micro-litre	140000-400000/microliter
2. ***SERUM CHEMISTRY***		
LFTS	Bilirubin-0.27, ALT-33.4, ALP-106	Bilirubin-0.3-1.2mg/dl, ALT-4-42 U/L ALP-35-105 U/L
RFTS	Urea-32.5, Cr-0.5	Urea -13-43mg/dl, Cr-0.5-1.1 mg/dl
Serum electrolytes	Na-136.6, K-3.49	Na-136-146mEq/L, K-3.5-5.1 mEq/L
3. ***COAGULATION PROFILE***		
PT	11.81 sec	10-14 sec
APTT	28.97 sec	28-42 sec
INR	0.90	0-1.1
4.HEPATITIS B &C Screening	Negative	Negative
5.ANA	Negative	Negative
6.ESR	25mm/hr	Less than 30 mm/hr
7. HIV Screening	Negative	Negative

Specific investigations included cerebrospinal fluid studies (CSF) and they are mentioned in Table 2.

Table 2: CSF studies

CSF PARAMETERS	PATIENT'S VALUE	REFERENCE RANGE
CSF SAMPLE		
TLC (Lymphocyte, Neutrophil)	40/mm^3(95%lympho,5%neutrophil)	Less than 5/mm^3
RBC	6000/mm^3	None
Xanthochromia	Present	Absent
Protein	38.2mg/dl	Less than 45 mg/dl
Glucose	67mg/dl (Serum glucose-130mg/dl)	40-70 mg/dl
Gene Xpert	MTB-detected very low	None
HSV I &II BY PCR	NEGATIVE	Negative
CSF Opening Pressure	20 cm of H_2O	10-25 cm of H_2O
CSF Cytology	Few bacteria seen, no malignant cells	None

Magnetic Resonance Imaging of the brain showed infarcts in the bilateral frontal region (parasagittal location) with no abnormal enhancement noted (Figure 1). Computed tomography angiography (CTA) showed aneurysmal dilatation of both anterior cerebral arteries with right ACA midline aneurysmal dilatation in midline measuring 3.9x4.7mm APXTR whereas left one measuring 3.6x4.2 mm APXTR (Figure 2). Computed tomography venography was normal.

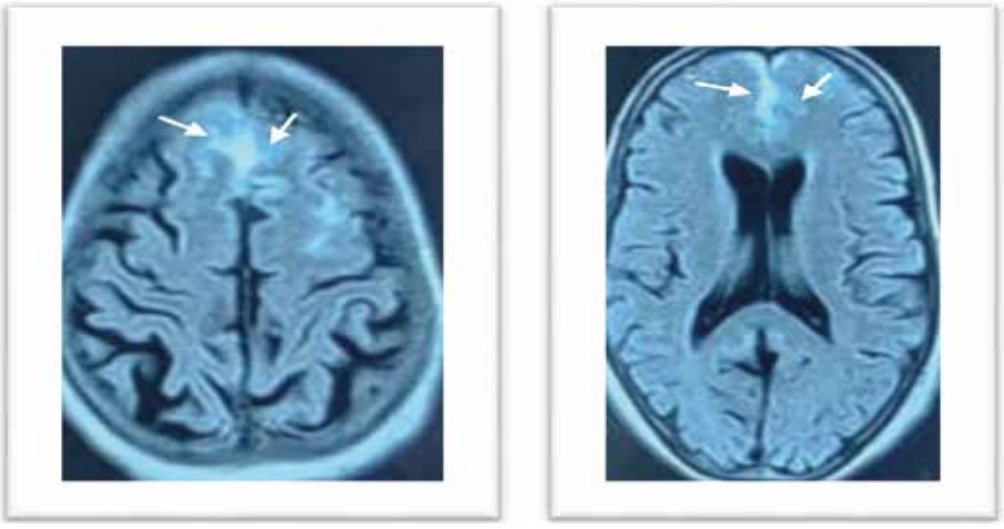

Figure 1: Magnetic Resonance Imaging of the brain showing infarcts in the bilateral frontal region

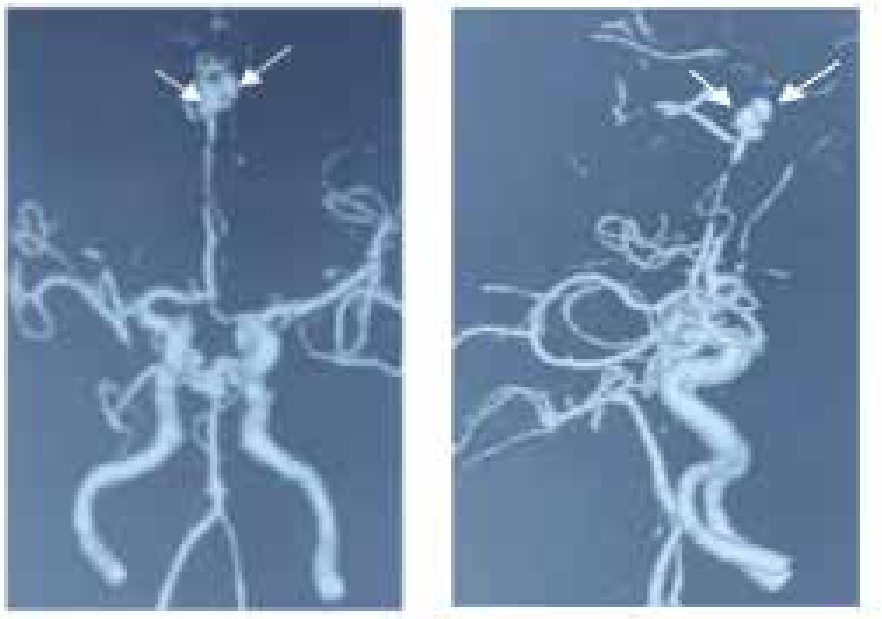

Figure 2: Computed tomography angiography (CTA) showing aneurysmal dilatation of both anterior cerebral arteries

Her treatment was started on the lines of subarachnoid hemorrhage (SAH). She was started on calcium channel antagonist to prevent vasospasm with good hydration and advice to avoid constipation through dietary modifications. Along with this, she was started on anti-tuberculous treatment (ATT) with oral steroids as per body weight. She improved clinically, and her GCS became 15/15. But she showed para-paresis with a power of 4/5 in both lower limbs. Neurosurgical consultation was sought and they advised an endovascular surgeon's opinion for any possible intervention. She was discharged on oral ATT with steroids along with a calcium channel antagonist. Pros/Cons/risks and prognosis were explained in detail along with advice of regular follow-up.

DISCUSSION

Being a catastrophic disease of the central nervous system, TBM may present as an acute infection or chronic infection with marked variability in presentation.[6] Dysregulated inflammatory response results in secondary vasculitis and ultimately cerebrovascular accident.[7] Our patient's diagnosis was compatible with probable TBM. She had altered mentation, focal neurologic signs, acute onset of illness, and an MTB detected on Gene X-pert, and the clinical improvement with anti-TBM treatment, was also supportive of TBM. Vascular complications of TBM include cerebral infarction and hydrocephalus. SAH is thought to be due to vasculitis or rupture of a mycotic aneurysm.[8,9]

Several mechanisms may result in the formation of aneurysms, such as endovascular expansion beginning with the septic embolization from an infective origin resulting in the dissemination of tubercle bacilli into the arterial wall via the vasa-vasorum causing tuberculous embolic vasculopathy remote from primary tuberculous lesions. Secondly, there could be hematogenous spread with an autoimmune response to tuberculosis.[10] Thirdly meningeal inflammatory exudate encroaches on the adventitia leading to enfeebling the vessel wall resulting in infective aneurysm formation.[11] Fourthly, the direct extension from a contiguous focus such as tuberculoma.[4] Fifthly, pre-existing congenital or degenerative intracranial aneurysms can be ascertained coincidentally with tuberculous meningitis.

Infectious intracranial aneurysms arising from diverse primary infection foci, such as infective endocarditis, bacterial meningitis, or cavernous sinus thrombophlebitis were reported.[10] One case was reported of bilateral ACA territory infarction in a patient with bacterial meningitis and miliary tuberculosis in 2019 in India.[12]

Few authors have reported such cases that showed extreme rarity of the combination of these conditions. In 1972, a female of 22 years of age reported having an anterior cerebral artery, middle cerebral artery, and posterior cerebral artery aneurysm.[13] Similarly in 1981 and 1988 Whelan and Leiguarda reported MCA aneurysm.[14,15] Cases were reported of MCA, posterior inferior cerebellar artery, and internal carotid artery

aneurysms with hemorrhages in TBM by Gupta, Griffiths, Tsuboi , and Jee-Hoon Roh respectively.[16-19] Recently a case was reported in 2023 with a young male diagnosed with tubercular meningitis who developed third cranial nerve palsy during course of illness and was found to have aneurysm of left ICA-MCA junction.[20]

CNS TB has a high mortality and morbidity. As it is rare and lacks data, the outcome of infectious intracranial aneurysms in TBM is uncertain and there is no widely accepted management or guidelines to follow in such scenarios. The management decision of an individual patient should probably include the location of aneurysms, surgical accessibility, number of lesions, and whether a bleed has occurred, with the latter carrying significant mortality. In such patients with or without intracranial aneurysm, anti-tuberculous therapy should be initiated as quickly as possible. The anti-tuberculous therapy should be continued with successful treatment of aneurysms.[21-23] The limitation in our case was that endovascular neurosurgical intervention facilities and expertise are not available in our center so we have to refer the patient where such facilities are available.

CONCLUSION

CNS infection with intracranial aneurysms is difficult to manage and such a combination of vascular complications of tuberculous meningitis must be kept in mind while dealing with such scenarios. The prognosis is likely to be good if diagnosed and managed as early as possible to avoid complications.

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Maryam Khalil; concept, case management, data collection, data analysis, manuscript writing
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Samer Naik; case management, data collection, data analysis, manuscript writing
Ayma Khan; data collection, data analysis, manuscript review
Zakir Jan; case management, data analysis, manuscript review
Mazhar Badshah; case management, data analysis, manuscript review
All the authors have approved the final version of the article and agree to be accountable for all aspects of the work.