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Bruns-Like Syndrome Heralding Disseminated Intracranial Tuberculomas in a 12-Year-Old Girl: A Rare Pediatric Presentation

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Case Report

Keywords: Bruns syndrome, intracranial tuberculoma, central nervous system tuberculosis, pediatric, seizure, positional vertigo, magnetic resonance spectroscopy

Posted Date: November 19th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11720341/v1

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Additional Declarations: The authors declare no competing interests.

Abstract

Background

Intracranial tuberculomas are a leading cause of seizures and intracranial mass lesions in tuberculosis-endemic countries. Bruns syndrome, defined by sudden headache, vomiting and vertigo on abrupt head movement, is classically attributed to a mobile intraventricular lesion that intermittently obstructs cerebrospinal fluid (CSF) flow. It has been described mainly with intraventricular neurocysticercosis and tumours, is uncommon in children, and has only rarely been linked to tuberculoma.

Case presentation:

A previously healthy 12-year-old girl presented with a first generalised tonic-clonic seizure after 15 days of brief, stereotyped episodes of vertigo and vomiting triggered by abrupt head movement. Neurological examination and fundoscopy were normal. Cranial imaging showed two parenchymal ring-shaped lesions with extensive vasogenic edema, effacement of the right lateral ventricle and mild leptomeningeal enhancement; MR spectroscopy showed a prominent lipid–lactate peak. CSF analysis, sputum CBNAAT and CSF PCR were negative, and the Mantoux test was non-reactive. Intracranial tuberculomas were diagnosed on clinical and radiological grounds. Four-drug antitubercular therapy, dexamethasone and an anticonvulsant resolved the positional episodes within one week, and she remained symptom-free at follow-up.

Conclusions

Bruns-like positional attacks can be the first sign of intracranial tuberculomas in children, even without a mobile intraventricular lesion, probably through edema-related, position-dependent disturbance of CSF dynamics. Recognising this presentation, and considering tuberculoma alongside neurocysticercosis, allows early treatment and avoids unnecessary invasive procedures.

INTRODUCTION

Tuberculosis remains a major global health problem; an estimated quarter of the world's population has been infected with *Mycobacterium tuberculosis* [1]. Involvement of the central nervous system (CNS), as meningitis, intracranial tuberculoma or spinal disease, is its most severe extrapulmonary form. In India, intracranial tuberculomas are an important cause of seizures and space-occupying lesions and, together with neurocysticercosis, account for most granulomatous CNS infections [2, 3]. Because both produce solitary or multiple nodular or ring-enhancing lesions with perilesional edema, they are readily mistaken for one another and for neoplasms [3, 4].

In 1902, Ludwig Bruns described attacks of headache, vomiting and vertigo provoked by head movement in patients with fourth-ventricular cysticercosis [5]. The syndrome is attributed to a mobile, deformable intraventricular lesion that intermittently obstructs CSF flow in a ball-valve fashion, causing transient intracranial hypertension [6]. It has since been reported chiefly with intraventricular neurocysticercosis and, less often, with intraventricular tumours [6, 7]. Reports in children are few and almost all cysticercal [8, 9], and a tuberculoma has been named as the cause only exceptionally [10].

We describe a 12-year-old girl whose Bruns-like positional episodes preceded a first seizure and were caused by disseminated parenchymal tuberculomas, without a mobile intraventricular lesion. The case shows that the syndrome in a child does not necessarily indicate an intraventricular cyst and that tuberculoma belongs in the differential diagnosis.

CASE PRESENTATION

A 12-year-old girl was brought to the pediatric emergency department after a generalised tonic-clonic seizure involving all four limbs, with oro-facial deviation and teeth clenching. There was no urinary or faecal incontinence. She remained confused for about 10 minutes and then recovered fully, with no residual weakness. This was her first seizure. She also had low-grade fever and left arm pain.

For the preceding 15 days her parents had noticed brief episodes, lasting seconds to minutes, of vertigo and vomiting triggered by abrupt head movement. She was well between episodes and had no visual complaints. There was no cough, diarrhoea, abdominal pain, or ear or eye discharge. She was the first child of a term, uncomplicated vaginal delivery, with normal growth and development and complete immunisation. There was no previous medical or surgical history. Her grandmother had been treated for abdominal tuberculosis with six months of antitubercular therapy one year earlier.

On examination she was conscious and well oriented and afebrile, with a pulse of 88/min, blood pressure of 116/84 mmHg and respiratory rate of 14/min. There was mild pallor, but no cyanosis or lymphadenopathy, and mild tenderness over the right temporal scalp. Systemic and neurological examination was normal. Direct fundoscopy showed no papilledema.

Investigations are summarised in Table 1. Haemoglobin was 10.4 g/dL and the erythrocyte sedimentation rate 55 mm/h. HIV serology was negative, the chest radiograph was normal and the Mantoux test was non-reactive. Cranial imaging MRI – brain showed two ring-shaped lesions, one in the right frontoparietal region and one immediately beside the right lateral ventricle (Figs. 1 and 2). Extensive vasogenic edema surrounded them, producing mass effect with effacement of the right lateral ventricle, and T2-weighted hyperintensity was also seen in the cerebellum and brainstem. Mild leptomeningeal enhancement was present. MR spectroscopy of the lesion showed a prominent lipid–lactate peak (Fig. 3). Lumbar puncture was performed; CSF cell count, protein and glucose were normal, and CBNAAT on sputum and PCR on CSF were negative.

On the basis of the history, family contact with tuberculosis, imaging and spectroscopy, intracranial tuberculomas with mass effect were diagnosed, with neurocysticercosis the main alternative. She was registered under the National Tuberculosis Elimination Programme and started on four-drug antitubercular therapy (isoniazid, rifampicin, pyrazinamide and ethambutol), dexamethasone and anti convulsant Levetiracetam. The vertigo and vomiting on head movement stopped within one week. She is seizure-free and asymptomatic on treatment at 3 months of follow-up.

Table 1
Laboratory and microbiological investigations.

Investigation	Result	Interpretation
Haemoglobin	10.4 g/dL	Mild anaemia
Total leukocyte count	9,000/mm³	Normal
Platelet count	1.9 lakh/mm³ (190 × 10³/μL)	Normal
Erythrocyte sedimentation rate	55 mm/h	Raised
Random blood glucose	69 mg/dL	Normal
Renal and liver function, electrolytes, urinalysis	Within normal limits	Normal
Rheumatological markers and tumour markers	Within normal limits	Normal
HIV serology	Negative	—
Mantoux test	Non-reactive	—
Chest radiograph	No abnormality	Normal
CSF cell count, protein, glucose	Within normal limits	Normal
CBNAAT (sputum)	MTB not detected	Negative
PCR (CSF)	Negative	Negative

CBNAAT, cartridge-based nucleic acid amplification test; CSF, cerebrospinal fluid; MTB, Mycobacterium tuberculosis; PCR, polymerase chain reaction.

DISCUSSION

Significance of the case

Bruns syndrome is rare in children, and the few pediatric reports describe intraventricular neurocysticercosis [8, 9]. A tuberculoma has been named as its cause in only one adult report that we could identify [10]. Our patient is, to our knowledge, the first child in whom Bruns-like attacks were attributed to disseminated parenchymal tuberculomas without an intraventricular lesion. The case matters for two reasons. It widens the recognised causes of the syndrome beyond mobile

intraventricular lesions, and it shows that a paroxysmal, head-movement-triggered presentation can be the first sign of CNS tuberculosis, here preceding the first seizure.

The classical explanation for Bruns syndrome is intermittent ball-valve obstruction of CSF pathways by a mobile intraventricular lesion [6]. Our patient's lesions were parenchymal, but imaging showed marked perilesional edema, effacement of the right lateral ventricle and posterior fossa involvement. We suggest that position-dependent distortion of CSF pathways and reduced intracranial compliance produced transient rises in intracranial pressure on abrupt head movement. Resolution of the attacks within a week of antitubercular therapy and corticosteroids is consistent with an edema-related mechanism.

Tuberculoma has no characteristic clinical signature. Its manifestations depend on the number, site and stage of the lesions and on the host immune response, and include headache, vomiting, seizures, visual disturbance, cranial nerve palsies and focal deficits [11–13]; rarer presentations such as recurrent stroke and intracranial hemorrhage also occur [14]. In our patient the usual supporting evidence was absent: there were no systemic symptoms, the chest radiograph was normal, the Mantoux test was non-reactive and the CSF was normal. The only pointers were a family contact, a raised ESR and the imaging. Normal CSF and a negative tuberculin test do not exclude intracranial tuberculoma, which is frequently mistaken for neurocysticercosis or neoplasm [3, 4, 15]. Diagnosis in our patient was clinicoradiological and presumptive; it was not confirmed microbiologically or histologically, and neurocysticercosis serology was negative. The prompt response to antitubercular therapy supports the diagnosis.

MRI is the investigation of choice. Appearances vary with the stage of the lesion, from non-caseating granuloma through caseating granuloma with or without central liquefaction to calcified granuloma [4]. Infratentorial tuberculomas are more common than was once believed [16]. MR spectroscopy can help to separate tuberculoma from neurocysticercosis: a large lipid–lactate peak with a raised choline/creatine ratio favours tuberculoma [17]. Our patient showed a prominent lipid–lactate peak (Fig. 3), which supported the diagnosis.

Treatment consists of a four-drug regimen of isoniazid, rifampicin, pyrazinamide and ethambutol, with second-line agents such as aminoglycosides and fluoroquinolones added if drug resistance is suspected. Adjunctive dexamethasone is used to dampen the inflammatory response and may reduce hydrocephalus and infarction; the evidence derives mainly from trials in tuberculous meningitis [18, 19]. If hydrocephalus causes deterioration, repeated lumbar puncture or external ventricular drainage may be needed [11]. An accurate diagnosis spares the child unnecessary shunting or antiparasitic treatment given for a presumed cysticercal cause.

Conclusion

Bruns-like attacks of positional headache, vertigo and vomiting can be the presenting feature of intracranial tuberculomas in children, without a mobile intraventricular lesion. In tuberculosis-endemic regions, clinicians who see this presentation, with or without seizures, should obtain neuroimaging and

keep tuberculoma in the differential diagnosis alongside neurocysticercosis, because early treatment is effective and invasive procedures can be avoided.

Declarations

Written informed consent for participation and publication of this clinical case was obtained from the patient's legal guardian.

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Figures

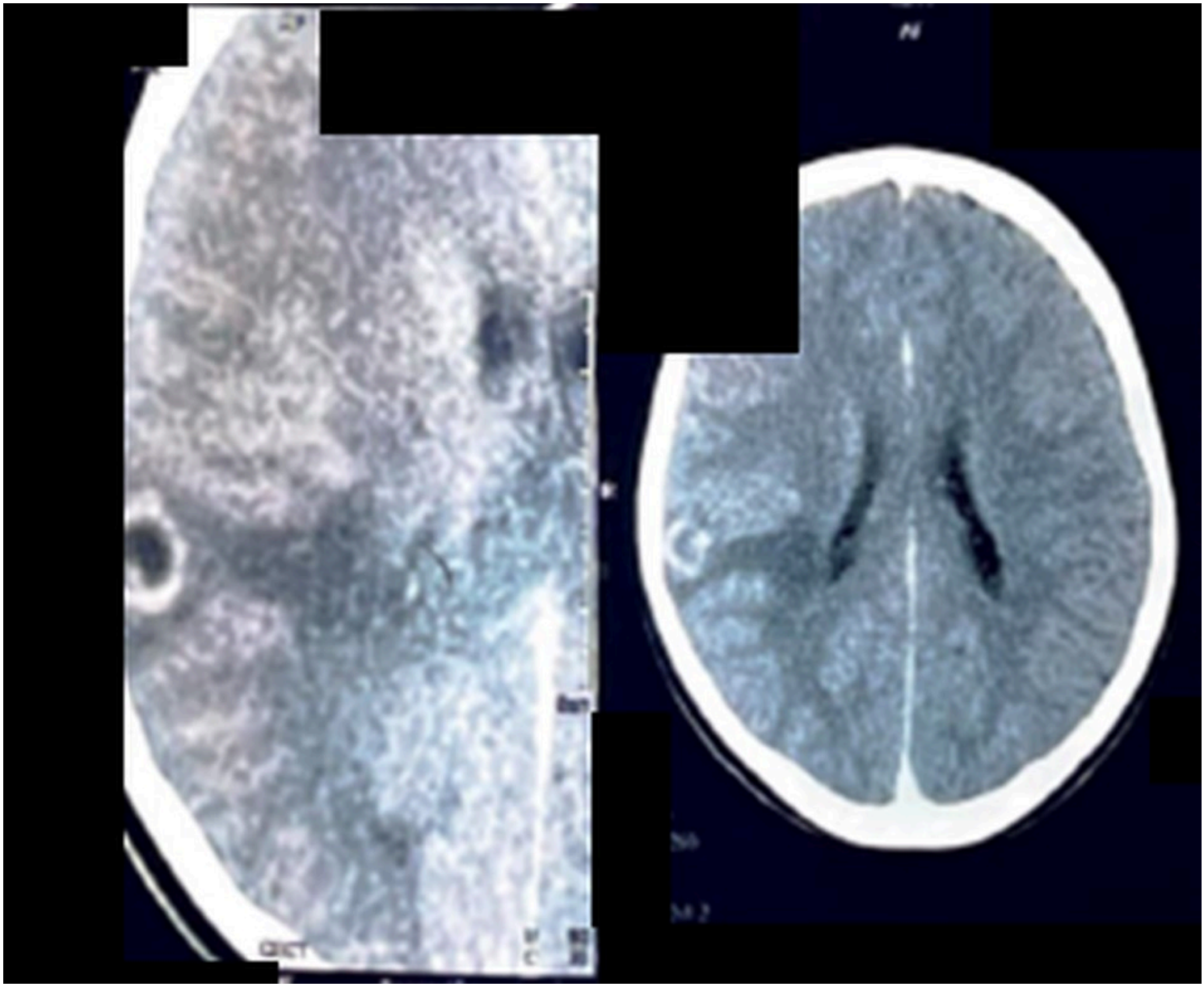


Figure 1

Axial cranial imaging showing a ring-shaped lesion in the right frontoparietal region with surrounding edema (left) and effacement of the right lateral ventricle (right). Patient identifiers have been removed.

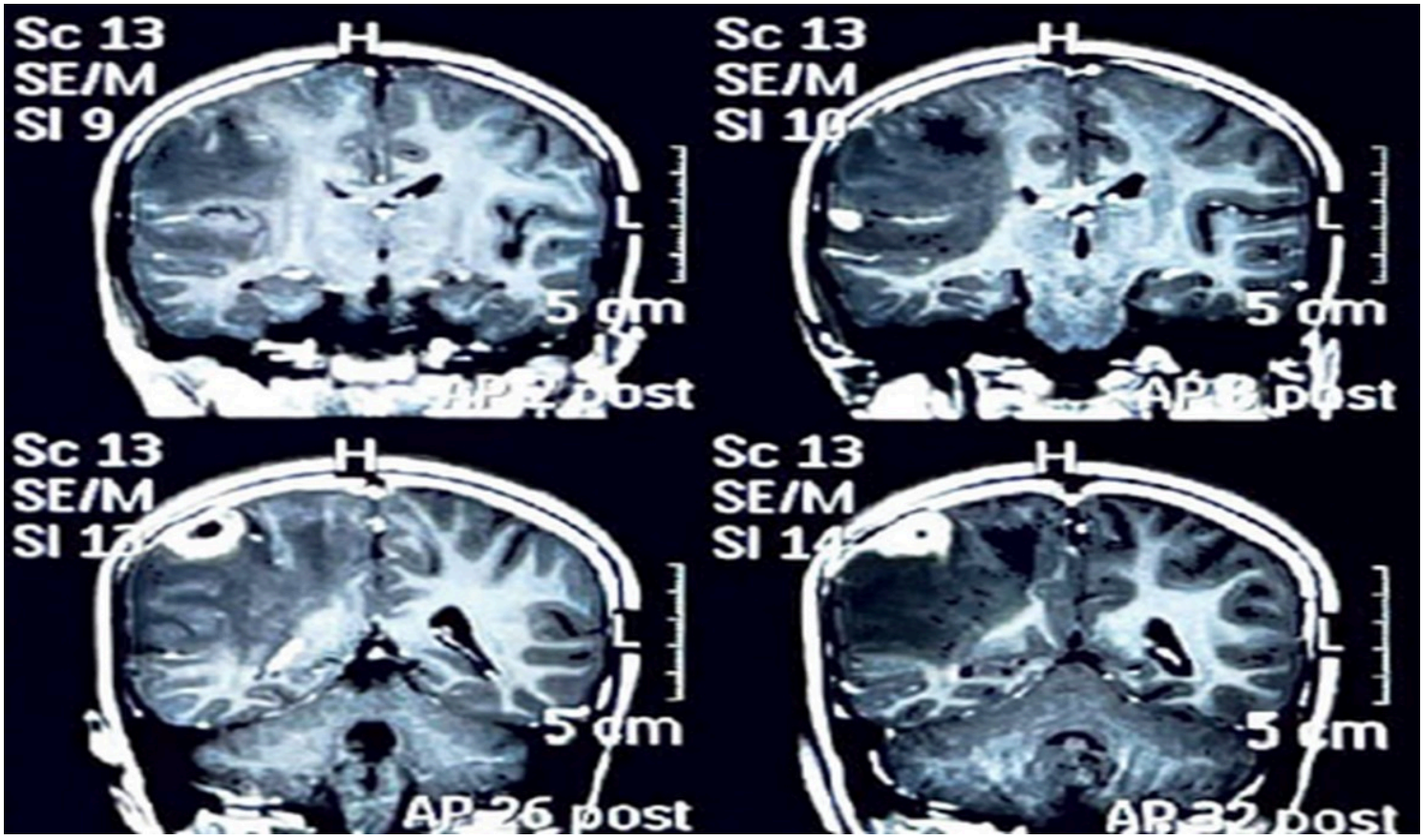


Figure 2

Coronal T1-weighted MR images (four consecutive slices) showing ring-shaped lesions in the right cerebral hemisphere with surrounding edema and mass effect.

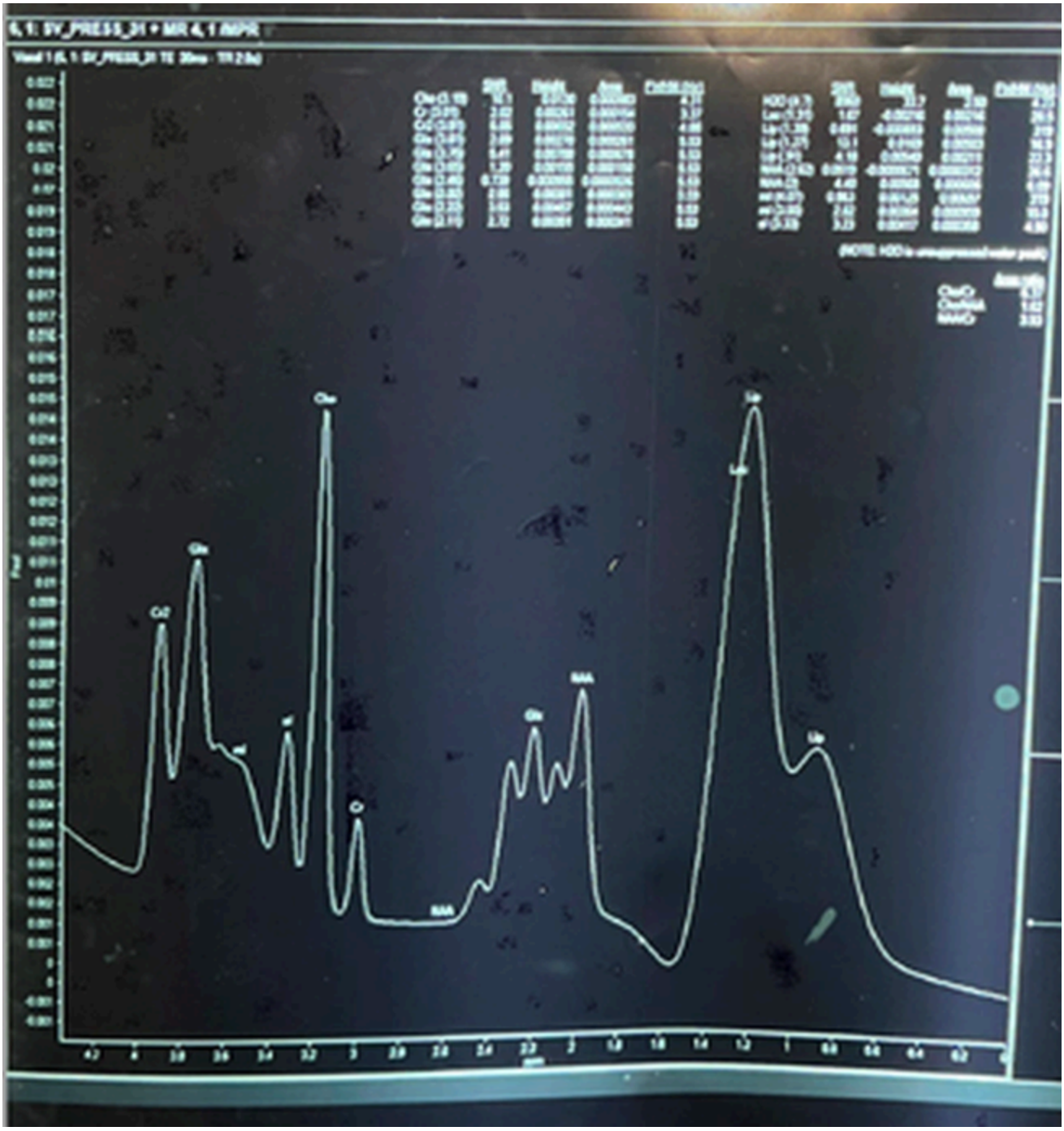


Figure 3

MR spectroscopy of the lesion showing a prominent lipid–lactate peak at approximately 1.3 ppm together with a choline peak.