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EC EMERGENCY MEDICINE AND CRITICAL CARE
Case Report

The Great Role of Brain Ultrasound as A Monitoring Tool in the Proper Management of A Case of TB Meningitis

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Abstract

Proposal: To examine and monitor the role of full TCD study in a case of TB meningitis to look for the development of common complications like hydrocephalus and arterial spasm.

Methods: We use a 2MH phase array probe to do Full TCD study including the third and lateral ventricles assessment, full Doppler study of both MCAs, ACAs, and we used high frequency 12MH probe to measure ONSD and internal carotid Doppler to measure Lindegard equation.

Case Report: A 23-year-old male patient presented to our ER because of 15days history of fever, headache and worsening of conscious level before presentation for which he received repeated courses of antibiotics without improvement.

We did full TCD study at ER which revealed marked dilatation of both third and lateral ventricles with a diameter of the third ventricle 1.04 cm, high mean flow velocity of both MCAs with RT MCA MFV 130 cm/sec and lindegaard ratio 4.8, LT MCA MFV 127 cm/sec with normal flow velocity of both ACAs, and LT ONSD 0.65 cm.

These TCD findings which we collect early on admission were alarming, so, urgent MRI brain were done which confirmed severe hydrocephalus, bilateral MCAs narrowing, as well as, RT Thalamic and internal capsule small infarctions plus RT cerebellar wall enhancing small lesion going with tuberculoma.

Patient went urgently for EVD shunt and analysis of CSF which was tuberculous in origin with positive PCR.

We follow the ventricular systems and MCAs Doppler daily while on antituberculous until they normalize.

Conclusion: Full TCD imaging has a great role for early discovering and monitoring for common complications of TB meningitis which are hydrocephalus and cerebral arteries vasculopathy.

Keywords: *TCD; TB Meningitis; Hydrocephalus; Complications of TB Meningitis; Brain Ultrasound*

Abbreviations

TCD: Trans Cranial Doppler; TCCD: Trans Cranial Color Coded Doppler; ONSD: Optic Nerve Sheath Diameter; MCA MFV: Middle Cerebral Artery Mean Flow Velocity; EVD: External Ventricular Drainage; TBM: Tuberculous Meningitis; ACA: Anterior Cerebral Artery

Introduction

Tuberculosis (TB) is a leading cause of morbidity and mortality [1]. It can involve the central nervous system as primary or secondary infection of the meninges (tuberculous meningitis, TBM) or brain parenchyma [World Health Organization. Global Tuberculosis Control. 2015].

The clinical picture may be nonspecific, presenting as chronic meningitis, and may be modified by various complications without the evidence of meningeal irritation. Within the central nervous system (CNS), hydrocephalus, tuberculomas and tuberculous vasculitis are the most important complications [2,3].

Terminal segment of internal carotid artery (ICA) and proximal portions of middle (MCA) and anterior (ACA) cerebral arteries are most frequently involved with inflammatory exudate [4,5].

Cerebral infarction is a recognized complication of TBM, and Vascular complications are often responsible for cerebral infarction and for neurological sequelae among adult survivors of TBM [6].

TBM related vasculopathy is a dynamic disease and hence, it may reflect the natural course of the disease process as well as the response to various treatment strategies. Beyond a certain degree of hemodynamic compromise, TBM-related vasculopathy may lead to cerebral infarcts and permanent disabilities. Thus, early evaluation of cerebral hemodynamic changes can assist in the management of critically ill TBM patients [7].

Previous studies reported cerebral vascular abnormalities in acute bacterial meningitis as short-lived and occurring within the first two to three weeks of infection [7].

Involvement of the intracranial arteries is considered to be one of the more important determinants of functional outcome in TBM [8,9].

Currently, trans cranial Doppler (TCD) is used for Noninvasive detection of changes in blood flow velocities

(BFV) of basal cerebral arteries involved in bacterial meningitis [9,10].

We present a case of TB meningitis which we are assessed in ER early on admission by Brain U/S and discovered the marked dilatation of third and fourth ventricles, distended LT ONSD[0.65], and high MFV of both MCAs by TCCD, these findings which we collected so early and accurately helped us a lot in management of the case, moreover, we monitor the patient by Brain U/S until normalization of the ventricular size and MFV of MCAs.

All of our TCD findings are confirmed by MRI, MRA Brain.

Case Report

A 23-year-old complained of persistent fever for 15 days associated with general weakness, malaise, anorexia and Headache, he asked medical advice and received repeated courses of antibiotics without improvement.

He was presented to our ER because of worsening conscious level, he was emaciated with body mass index 18, BP 160/90, HR 130/min, temperature 39 degree Celsius, O_2 saturation 100% room air.

GCS: 13/15 M5E4V4, no signs of meningeal irritation.

Chest: Clear.

Heart: Sinus tachycardia.

Abdomen: Soft, lax, no organomegaly.

Chemistry

Serum Albumin 32 gm/L, ALP 80 IU/L, ALT 19 IU/L, AST 18 IU/L , Total Bilirubin 5 micM/L , GGT72 IU/L, Serum Protein 74 gm/L, Serum NA 123 mmol/L, K 3 mmol/L, CL 83 mmol/L, BUN 2.7 mmol/L, creatinine 42 micM/L, CK 43 IU/L, Serum Calcium 2.1 mmol/L, Phosphorus 0.81 mmol/L, Magnesium 0.72 mmol/L.

Haematology

- WBCs 13650/cmm, HB 13 gm/dl, Platelets 487000/cmm, RBCs 4000000/cmm.
- TPHA negative, VDRL non-reactive.

First TCD at ER

1. Marked dilatation of third and lateral ventricles with third ventricle diameter 1.04 cm.
2. RT MCA MFV 130 cm/sec with RT Lindegaard equation 4.8, LT MCA MFV 127 cm/sec, LT ACA PSV 100 cm/sec, EDV 45 cm/sec.
3. LT ONSD 0.65 cm.

First MRI

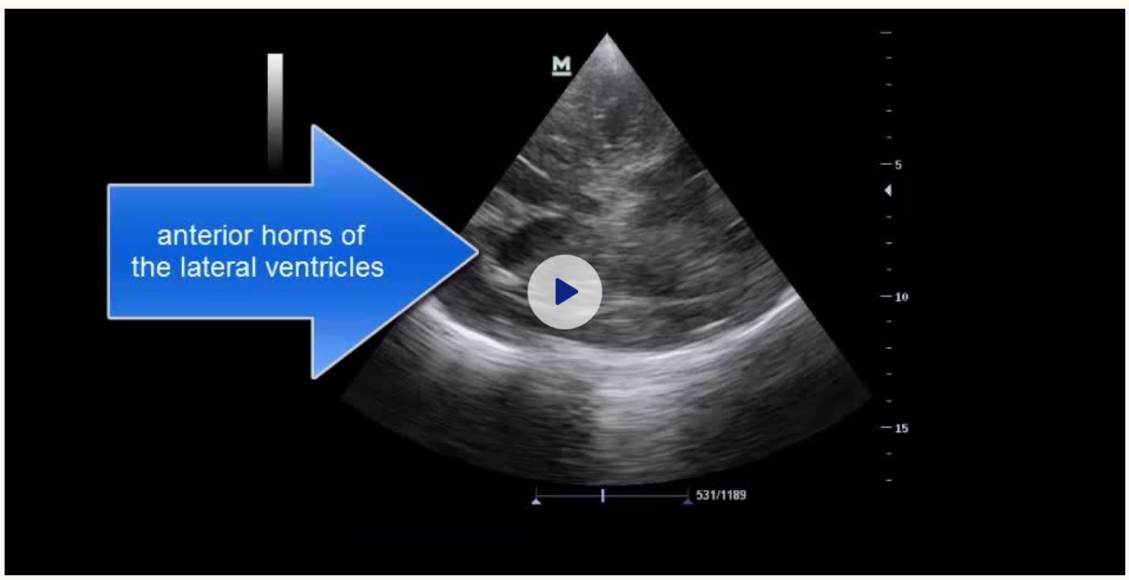
Severe hydrocephalus, bilateral narrowing of MCAs, suspicious tuberculoma RT cerebellum, and small infarctions at RT Thalamic and internal capsule.

Patient went urgently for EVD and analysis of CSF which results came [wcc 80/cmm 80% lymphocytes, glucose 25 mg/dl with CSF Glucose/Blood Glucose = 0.4, Protein 280 mg/dl] and positive TB BCR.

F/U TCD after EVD revealed improvement of hydrocephalus with LT anterior horn of lateral ventricle 0.87 cm, normalization of MCAs Flow velocity RT MCA PSV 122 cm/sec, EDV 63 cm/sec, LT MCA Flow velocity 70/50 cm/sec.

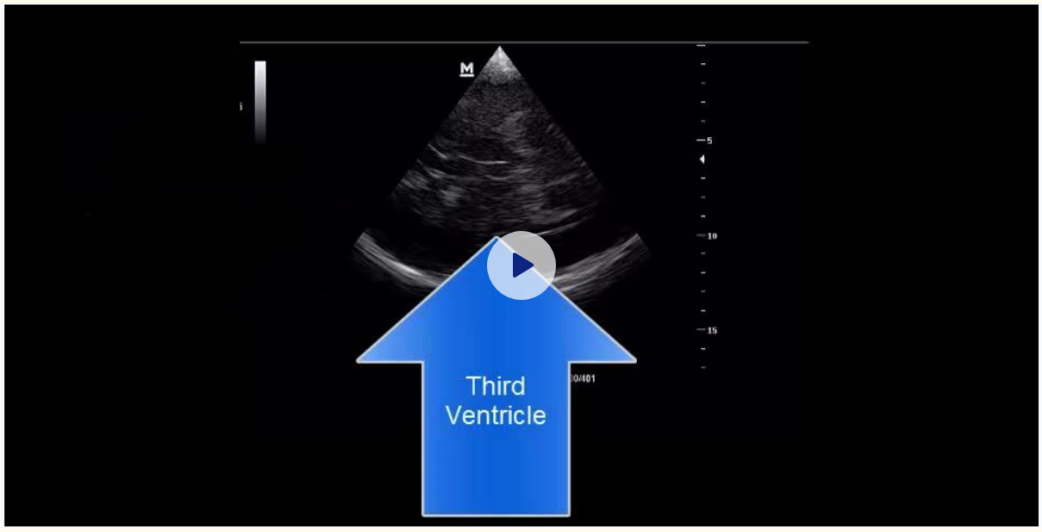
We repeatedly do TCD until normalization of ventricular system [third and lateral ventricles] and our TCD findings were confirmed by repeated CT.

After EVD patient was admitted to our ICU and started antituberculous medication [isoniazid, rifampicin, ethambutol, and pyrazinamide] empirically and continued after confirmation of TB, patient improved and disconnected from mechanical ventilator fully conscious but complained of LT side weakness.



https://www.dropbox.com/s/i9g7s1vq4rifcsb/dilated%20vertricles.mp4?dl=0

Figure 1: *First TCD video, revealing marked dilatation of third and lateral ventricles.*



https://www.dropbox.com/s/5lrwdsx0wpfw8xw/improved%20dilatation%20ventricls.mp4?dl=0

Figure 2: *Second TCD Video, revealing improved hydrocephalus after EVD.*

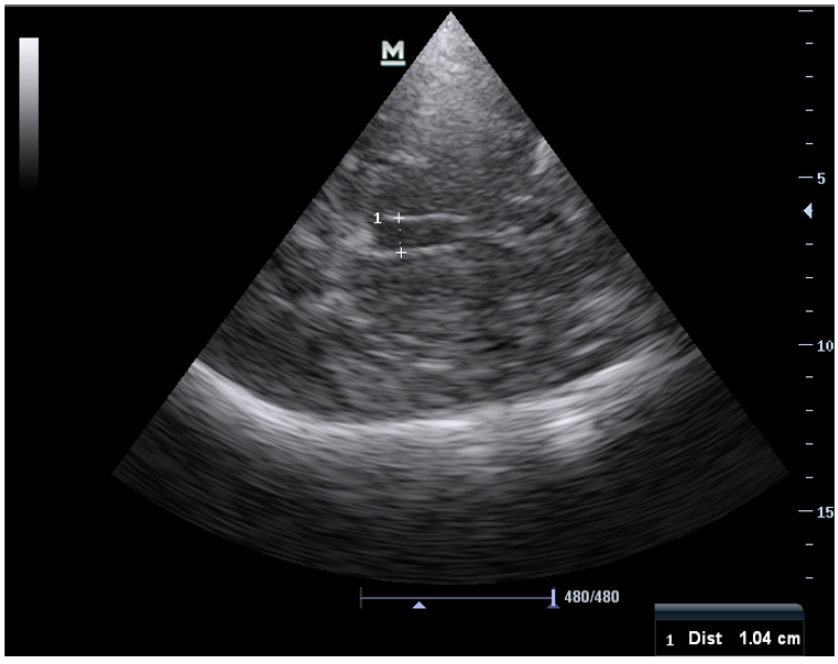


Figure 3: *Dilated third ventricle [1.04] before EVD.*

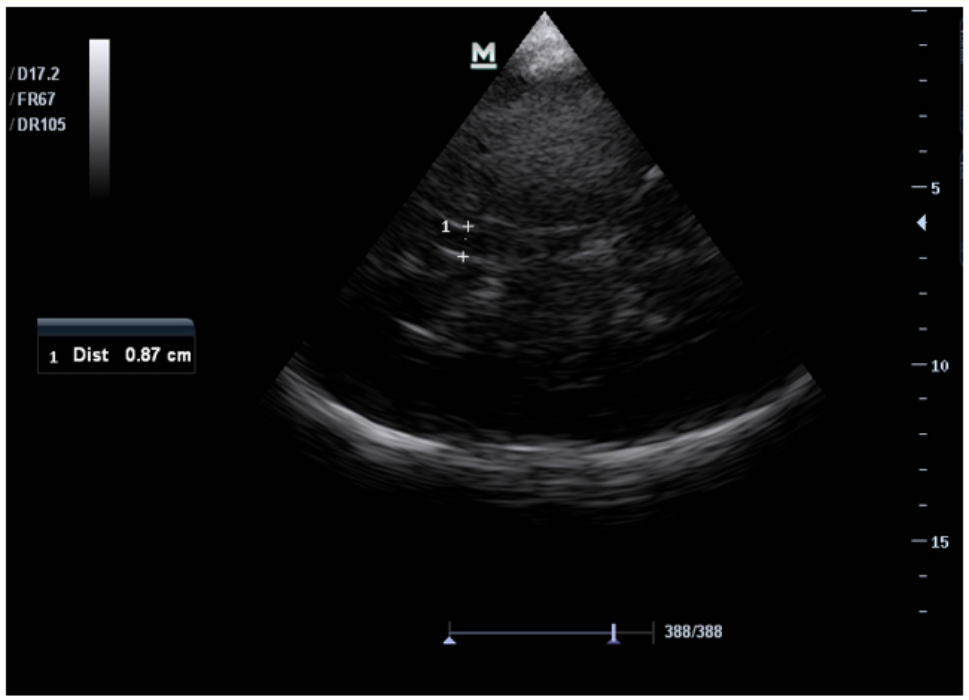


Figure 4: *Improved hydrocephalus after EVD with LT Anterior Horn of Lateral ventricle 0.87 cm.*

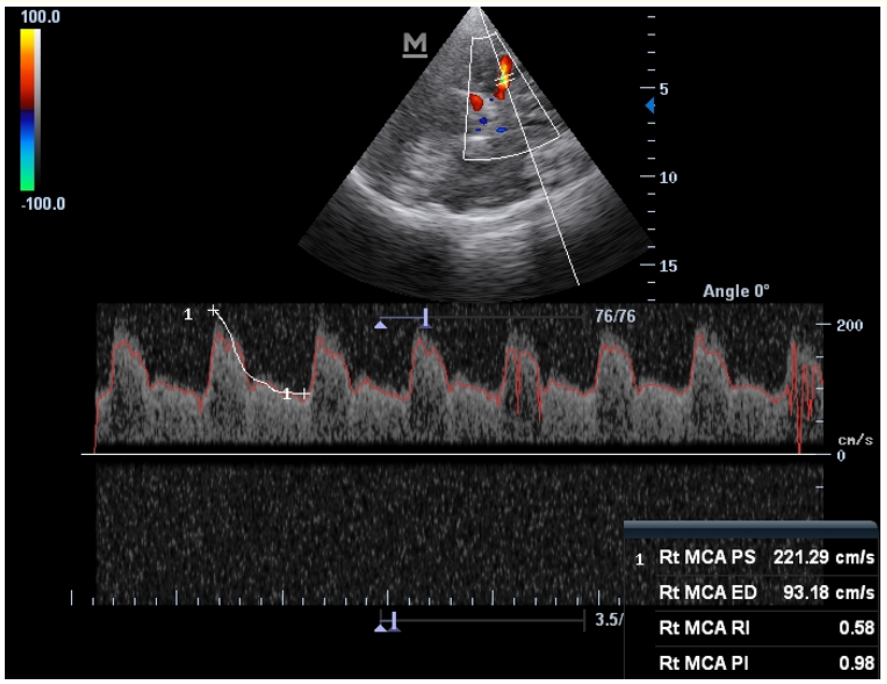


Figure 5: *RT MCA PSV 221 cm/sec, EDV 93 cm/sec, MFV 130 cm/sec.*

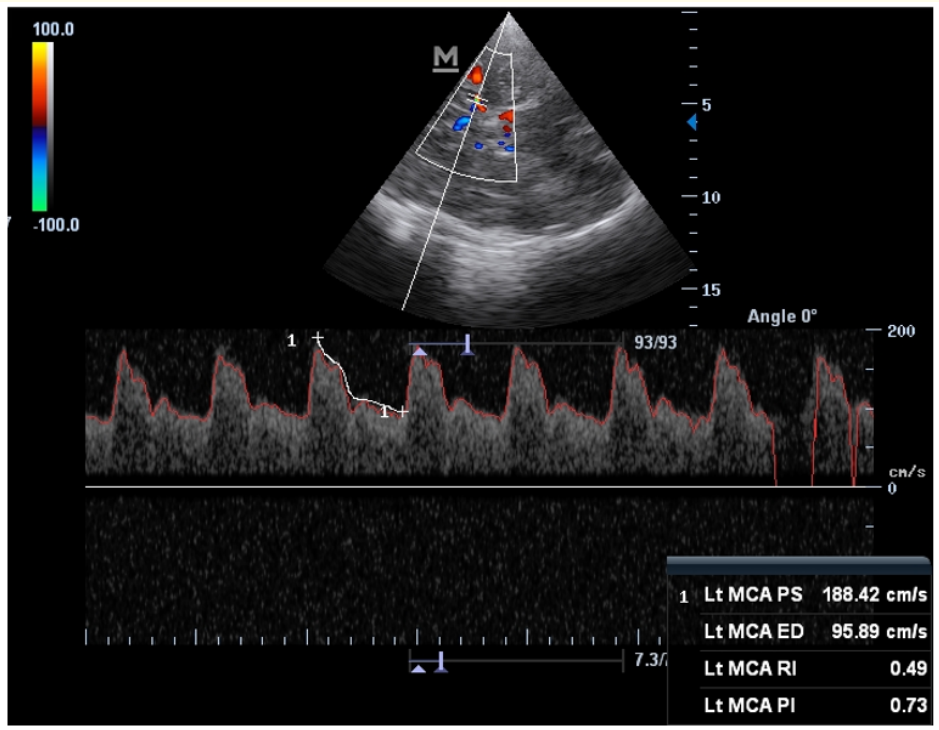


Figure 6: *LT MCA PSV 188 cm/sec, EDV 95 cm/sec, MFV 127 cm/sec.*

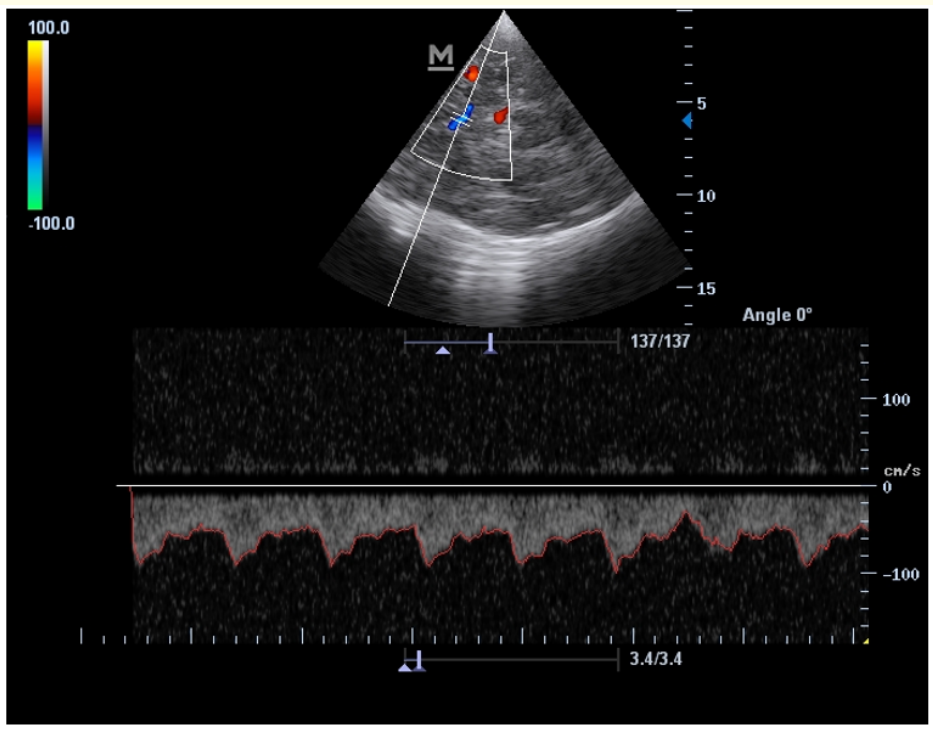


Figure 7: *LT ACA PSV 100 cm/sec, EDV 50 cm/sec.*

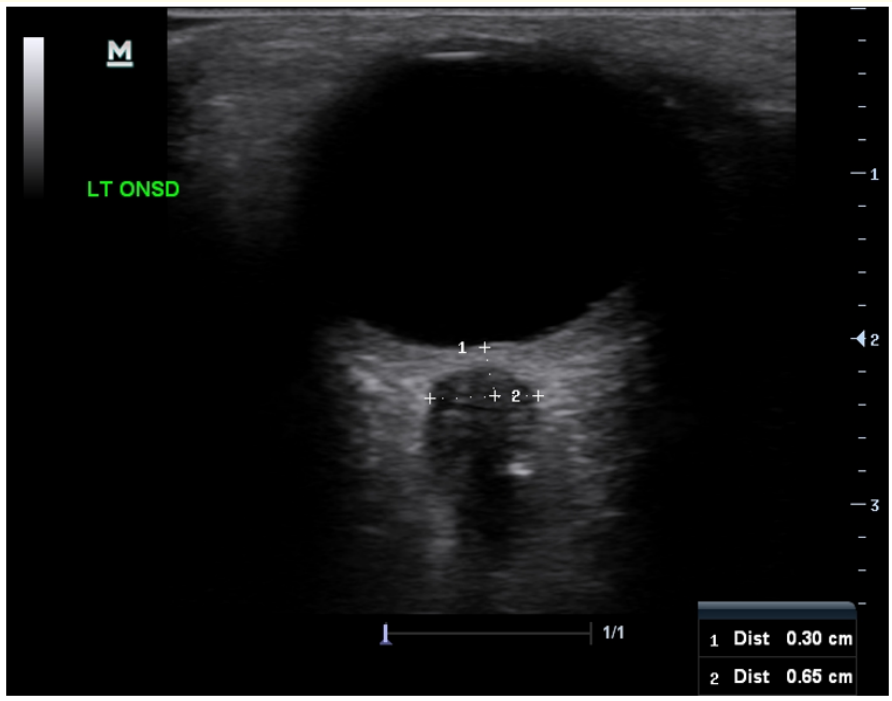


Figure 8: *LT ONSD 0.65 cm.*

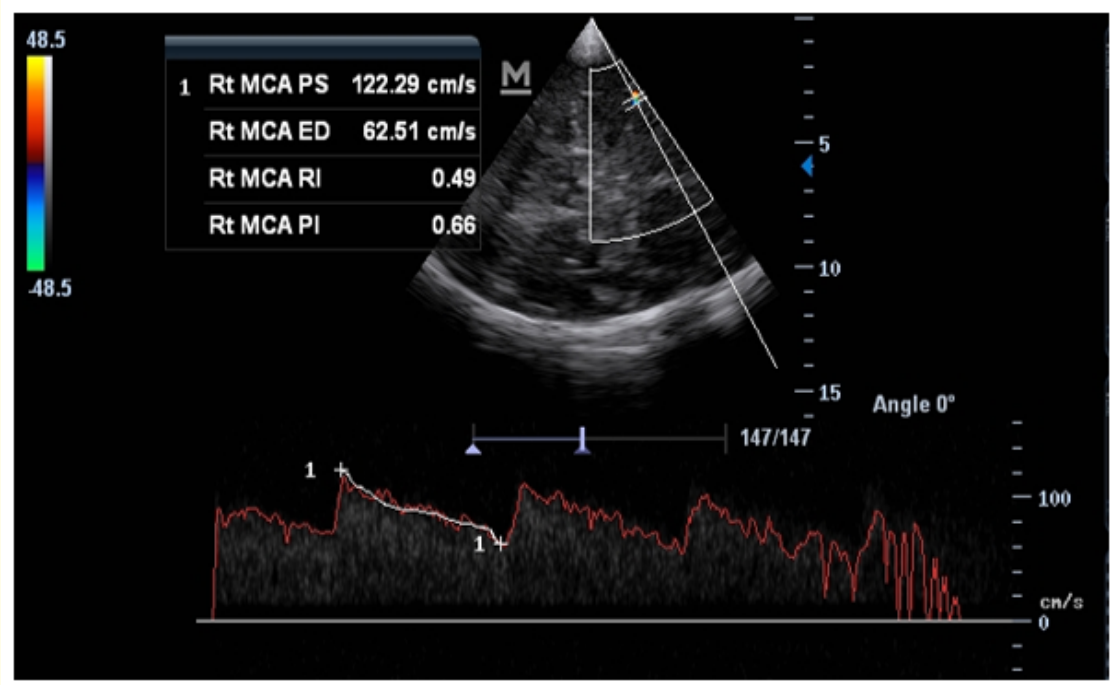


Figure 9: *Normalise RT MCA Flow Velocity after EVD and antituberculous.*

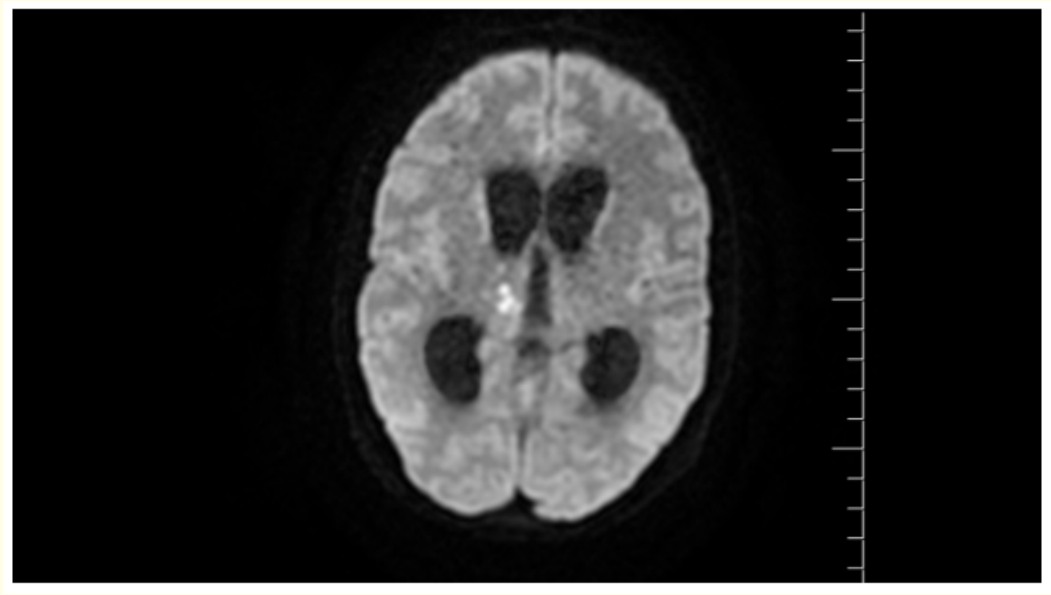


Figure 10: *MRI Brain with severe hydrocephalus, RT thalamic infarction.*

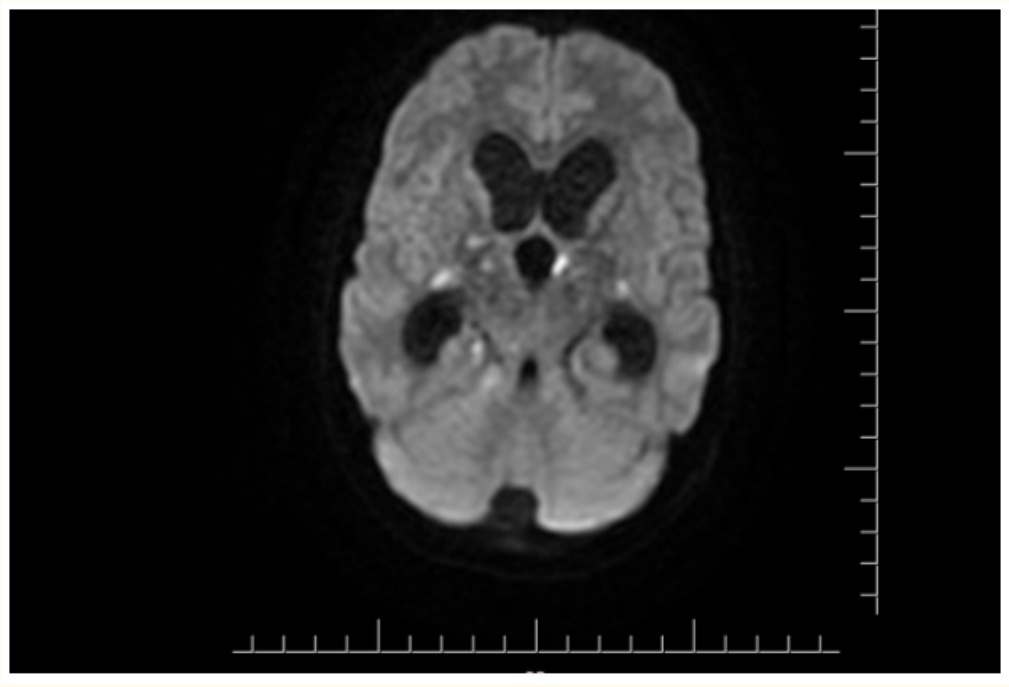


Figure 11: *MRI Brain with RT internal capsule small infarction.*

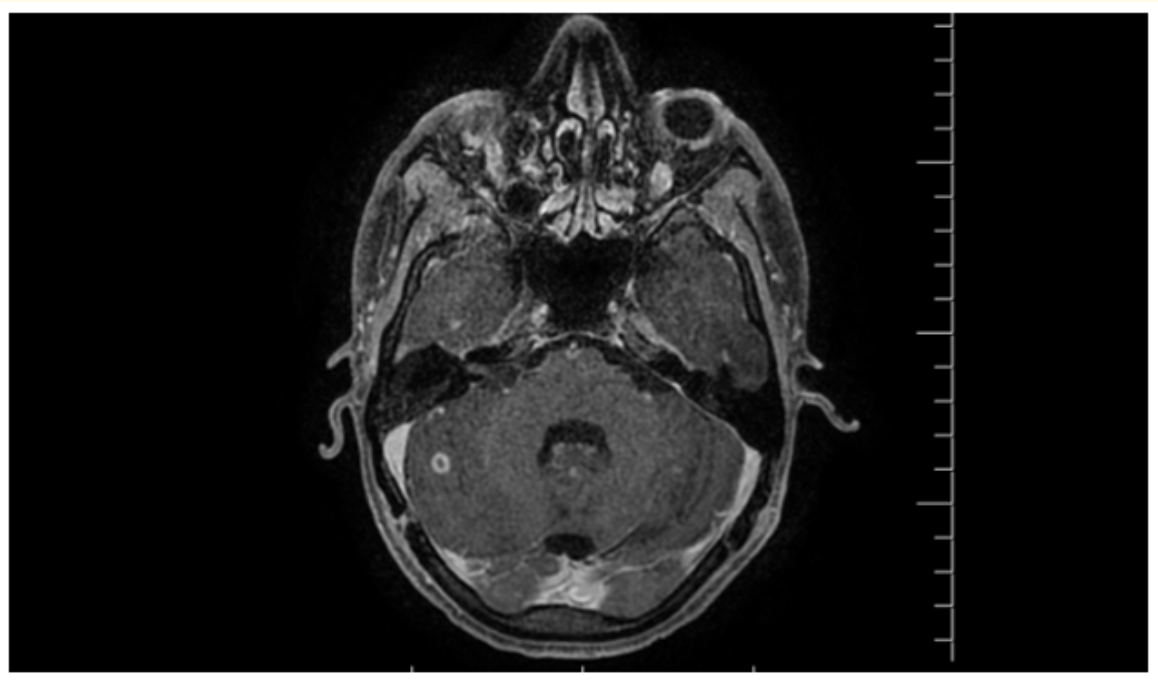


Figure 12: *Brain MRI suspicious RT cerebellar tuberculoma.*

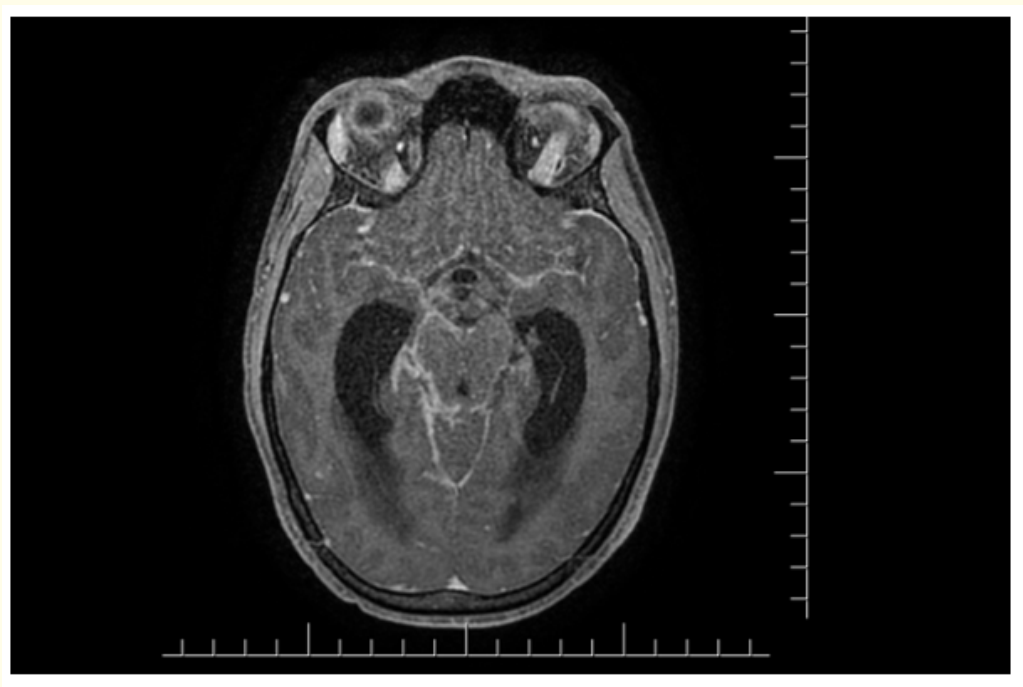


Figure 13: *MRI Brain, narrowing both MCAs.*

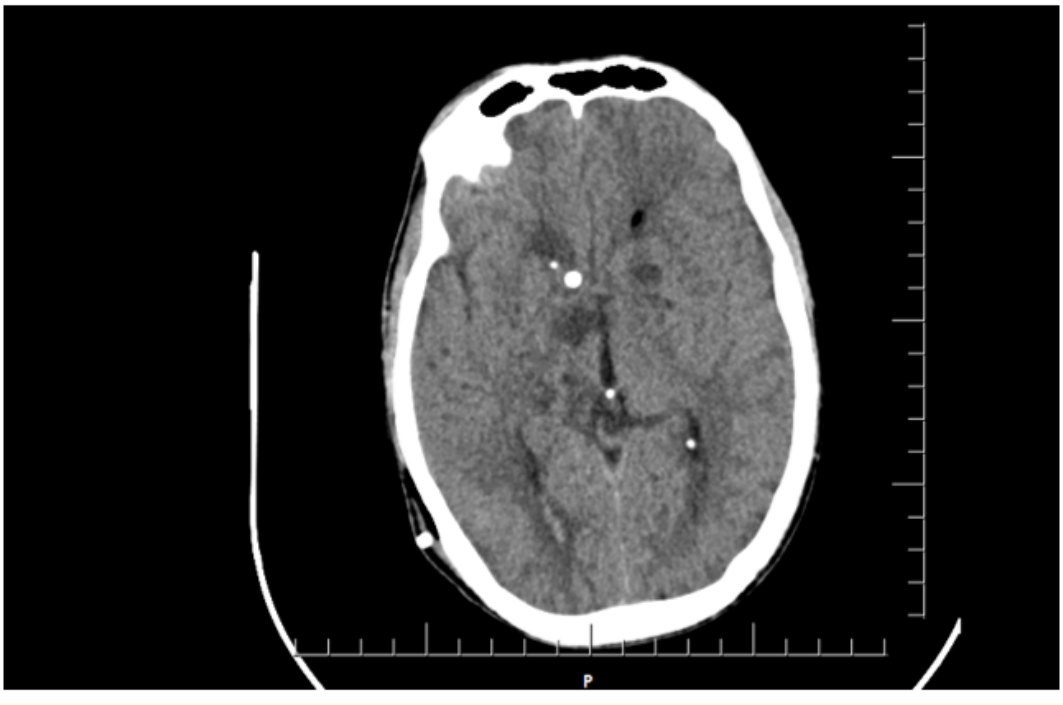


Figure 14: *CT Brain F/U no hydrocephalus after EVD with RT Thalamic and LT internal capsule infarction.*

Discussion

Tuberculosis (TB) is a leading cause of morbidity and mortality [1], It can involve the central nervous system as primary or secondary infection of the meninges (tuberculous meningitis, TBM) or brain parenchyma.

Within the central nervous system (CNS), hydrocephalus, tuberculomas and tuberculous vasculitis are the most important [2,3].

Terminal segment of internal carotid artery (ICA) and proximal portions of middle (MCA) and anterior (ACA) cerebral arteries are most frequently involved with inflammatory exudate [4].

TCD is a noninvasive quick way to evaluate the ventricular enlargement and flow velocity of the basal cerebral arteries so it can be very useful in monitoring of TB meningitis complications.

TCD can evaluate and monitor the ventricular system [11,12].

TCD is an accurate way of measuring Flow velocity of basal cerebral arteries [13].

We present a case who admitted to our ER Department with a history of suspicious CNS infection as evidenced by 2 weeks history of fever malaise, weakness, fever, anorexia with recent deterioration of conscious level, there was no signs of meningeal irritation, patient did not respond to repeated courses of antibiotics.

We used to assess any patient with DCL by Transcranial U/S as well as Brain imaging and full analysis for metabolic causes of coma [serum lytes, glucose, renal and liver function, ABG, Toxicology once indicated].

We do full trans cranial U/S including third and fourth ventricles diameter to diagnose hydrocephalus, midline shift, any hyper echoic lesions denoting cerebral hemorrhage, Trans cranial color coded Doppler for basal cerebral arteries, as well as ONSD.

Brain Ultrasound is noninvasive, radiation free, accessible, Cheap, repeatable and a very quick modality to gather a very valuable informations in a golden minutes.

In our patient and while we are waiting the results of biochemistry and CT to be ready, we did the full TC U/S.

First, we discover very clearly and so early the marked dilatation of third and fourth ventricle, together with the distended LT ONSD [0.65], this shift the diagnosis satisfactory towards the increase ICP as a central cause for confusion.

Second, by TCCD we discover so early the marked increase of both MCA MFV [RT MCA MFV 130 cm/sec, and LT MCA MFV 127cm/sec] and the RT side Lindegaard 4.8 which highly support the diagnosis of cerebral vasculopathy whatever spasm or vasculitis.

Considering the high suspicious of CNS, infection these findings alert us about the occurrence of complications of CNS infection which are hydrocephalus and basal cerebral vasculopathy.

All these valuable TCD Data helped us a lot in the next steps of management which are:

1. we keep BP 160/100 because of high MFV of both MCAs.
2. Urgent MRI, MRA Brain which confirmed the TCD findings of severe hydrocephalus, Brain Edema and bilateral narrowing of MCAs.
3. Early neurosurgical consultation and EVD insertion before admission to ICU.

Moreover, we monitor our patient by Full Brain U/S in ICU after EVD and we confirmed first, improvement and later cure of hydrocephalus, as well as we monitor the Blood flow velocity of MCAs which normalize with antituberculous treatment.

We believe that Full Brain U/S has a great value for the first assessment of comatose patient in ER and in case of CNS TB infection can help a lot in monitoring for the most common complications which are hydrocephalus and cerebral arterial vasculopathy.

Mei-Ling Sharon Tai and Vijay K Sharma recruited a total of 36 TB meningitis patients. Focally elevated flow velocities in the middle cerebral artery (MCA) were observed in 11 (30.6%) patients, bilaterally in 6 of them. The Lindegaard ratio was elevated (>3) in 10 (90.9%) of them. Eighty percent of patients with TBM vasculopathy by TCD criteria, also had narrowing on CTA or MRA [14].

Evguenia Vassileva, M.D., Ph.D., University Department of Neurology, Medical University of Sofia, Queen Joanna University Hospitaa presented a case study of 44-year-old female patient diagnosed with TB meningitis by histological study and they did TCD for the patient which revealed A RT MCA MFV 133 cm/sec and LT MCA MFV 97cm/sec.

Otherwise, studies of TCD with meningitis especially TB meningitis are scare.

Conclusion

We believe than total Brain U/S including assessment of ventricular system. Midline shift, presence of hyper echoic areas in brain denoting hemorrhage, full Blood Flow Doppler study of the basal cerebral arteries as well as ONSD should be one of the first investigation to be done in a comatose patient in ER with suspected central origin of coma and it has a great value in diagnosing and monitoring TB meningitis common complications which are hydrocephalus and basal cerebral arteries vasculopathy.

We believe in Brain U/S Coma protocol like BLUE protocol of acute respiratory failure and RUSH protocol of shock.

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