International Journal of Surgery Case Reports 99 (2022) 107619

Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.elsevier.com/locate/ijscr

Case report

Paroxysmal sympathetic hyperactivity syndrome in tuberculous meningitis with paradoxical reaction

Andre Marolop Pangihutan Siahaan [a,*], Steven Tandean [a], Rr Suzy Indharty [a], Bahagia Willibrodus Maria Nainggolan [b], Martin Susanto [c]

[a] Department of Neurosurgery, Faculty of Medicine, Universitas Sumatera Utara, Medan, Indonesia
[b] Student Research Assistant, Department of Neurosurgery, Faculty of Medicine, Universitas Sumatera Utara, Medan, Indonesia
[c] Petang II Public Health Center, Bali, Indonesia

ARTICLE INFO

Keywords:
Tuberculous meningitis
Paradoxical reaction
Paroxysmal sympathetic hyperactivity
Case report

ABSTRACT

Introduction and importance: Paroxysmal sympathetic hyperactivity syndrome is frequently reported in traumatic brain injury. However, it may occur in non-traumatic brain injury, such as tuberculous meningitis with hydrocephalus.

Case presentation: We reported a 38-year-old male who presented with acute decrease of consciousness and hemiparesis that was developed during antitubercular drugs therapy. CT Scan showed hydrocephalus and granuloma lesion. Emergency ventriculoperitoneal shunting were performed. During treatment, the patient developed paroxysmal sympathetic syndrome during treatment that was controlled based on symptom elimination and prevention.

Clinical discussion: Brain tuberculosis remains a difficult problem for clinicians. Even when antitubercular drugs are administered according to protocol, paradoxical reactions can occur. If hydrocephalus develops, ventriculoperitoneal shunting is one of the options for lowering intracranial pressure. Paroxysmal sympathetic hyperactivity may occur in brain tuberculosis and should be detected as soon as possible to avoid serious morbidity.

Conclusion: Paroxysmal sympathetic hyperactivity may be developed in brain tuberculosis. Early identification and treatment are mandatory.

1. Introduction

Paroxysmal sympathetic hyperactivity (PSH) is a phenomenon characterized by episodes of elevated sympathetic activity [1]. Hyperactivity of the sympathetic nervous system can appear as tachycardia, tachypnea, hypertension, diaphoresis, and increased body temperature. Majority of these cases were reported in traumatic brain injury [2]. Reports on PSH in tubercular meningitis are scarce. However, early identification and management of this condition should not be delayed given that PSH relates to problematic clinical outcomes, such as the poorer clinical result, physical handicap, prolonged hospital stay, and high healthcare costs [3].

Tubercular meningitis itself remains challenging problem for clinician due to high morbidity and mortality. Even when antitubercular medications are administered according to protocol, new lesions or new clinical deterioration may develop. This condition is known as paradoxical reaction. This reaction may falsely suggest a drug-resistant state or treatment failure and may even trigger the search for a different diagnosis [4,5].

Hydrocephalus is among the most prevalent consequences of tuberculous meningitis, and it has been demonstrated to negatively impact the prognosis [6]. Surgery is necessary when signs of elevated intracranial pressure are present. There are several surgical treatments for hydrocephalus, such as ventriculoperitoneal shunt, external ventricular drainage, and endoscopic third ventriculostomy [7]. To this date, there is no strong evidence regarding the best choice in hydrocephalus that is caused by tuberculous meningitis.

Here we reported a case of tuberculous meningitis with hydrocephalus and paroxysmal sympathetic hyperactivity as complications. This neurologic deterioration occurred when the patients got antitubercular drugs for two months. This case report is written according to the revised 2020 SCARE guideline [8].

* Corresponding author at: Department of Neurosurgery, Faculty of Medicine, Universitas Sumatera Utara, Jl. Dr. Mansyur No. 66, Medan 20154, Indonesia.
E-mail address: andremarolop@usu.ac.id (A.M.P. Siahaan).

https://doi.org/10.1016/j.ijscr.2022.107619
Received 13 August 2022; Received in revised form 5 September 2022; Accepted 6 September 2022
Available online 8 September 2022

2. Case report

A 38-year-old male presented to the emergency department of our hospital, a secondary community hospital, with a loss of consciousness three day prior to admission with intermittent vomiting. He had been suffering from a progressive headache for two weeks, particularly in the morning. There was also a three-month history of coughing and weight loss. Based on chest X-ray and sputum PCR GeneXpert examination two months prior, he was diagnosed with pulmonary tuberculosis and was given antitubercular drugs in accordance with WHO guidelines (category 1, since resistance to rifampicin was not found). There was no history of trauma. History of BCG vaccine was found. His wife had pulmonary tuberculosis and had completed antitubercular regiment for six months.

He was admitted with a Glasgow Coma Scale of 11/15. The patient's blood pressure, heart rate, respiratory rate, and temperature were all normal. During a neurological examination, right hemiparesis was discovered. On both lower extremities, there was a normal patellar tendon reflex and a positive Babinski sign. Nuchal rigidity was discovered, as well as increased muscle tone. Pupils were symmetric, with a sluggish reflex on both sides. On funduscopic examination, there was hyperemia and swelling of the disc, as well as a blurring of the disk's margin, which is indicative of papilledema.

On laboratory examination, hemoglobin was 11.6 g/dL (reference 13–18), white blood count was 8800/μL (reference 4000–11,000/μL), platelet was 226.000/mm^3 (reference 140.000–450.000/mm^3), and erythrocyte sedimentation rate was 33 mm/h (reference 0–15 mm/h). There was also mild hyponatremia (130 mmol/L, reference 135–150 mmol/L). Qualitative C-reactive protein was positive. Renal function, liver function, and arterial blood gas analysis were in normal range. Blood HIV ELISA was negative.

Brain computed tomography (CT) indicated hypodensity on left thalamic and midbrain hypodensity, as well as temporal horn enlargement, consistent with communicating hydrocephalus (Fig. 1). The presence of basal cistern enhancement and rim enhancement mass on the quadrigeminal cistern following contrast delivery is suggestive of tuberculous meningitis and intracranial granuloma lesion.

An emergency ventriculoperitoneal shunt was performed by general neurosurgeon. Cerebrospinal fluid (CSF) was clear with high pressure. Cerebrospinal fluid (CSF) analysis revealed lymphocytic pleocytosis with increased CSF protein of 320 mg/dL (reference 15-60 mg/dL) and normal glucose level of 52 mg/dL (reference 50–80 mg/dL). Random blood sugar was in normal range (120 mg/dL), made glucose CSF to blood ratio of 0.43. *Mycobacterium tuberculosis* culture of CSF at six weeks was negative.

The patient was admitted to intensive care unit after procedure.

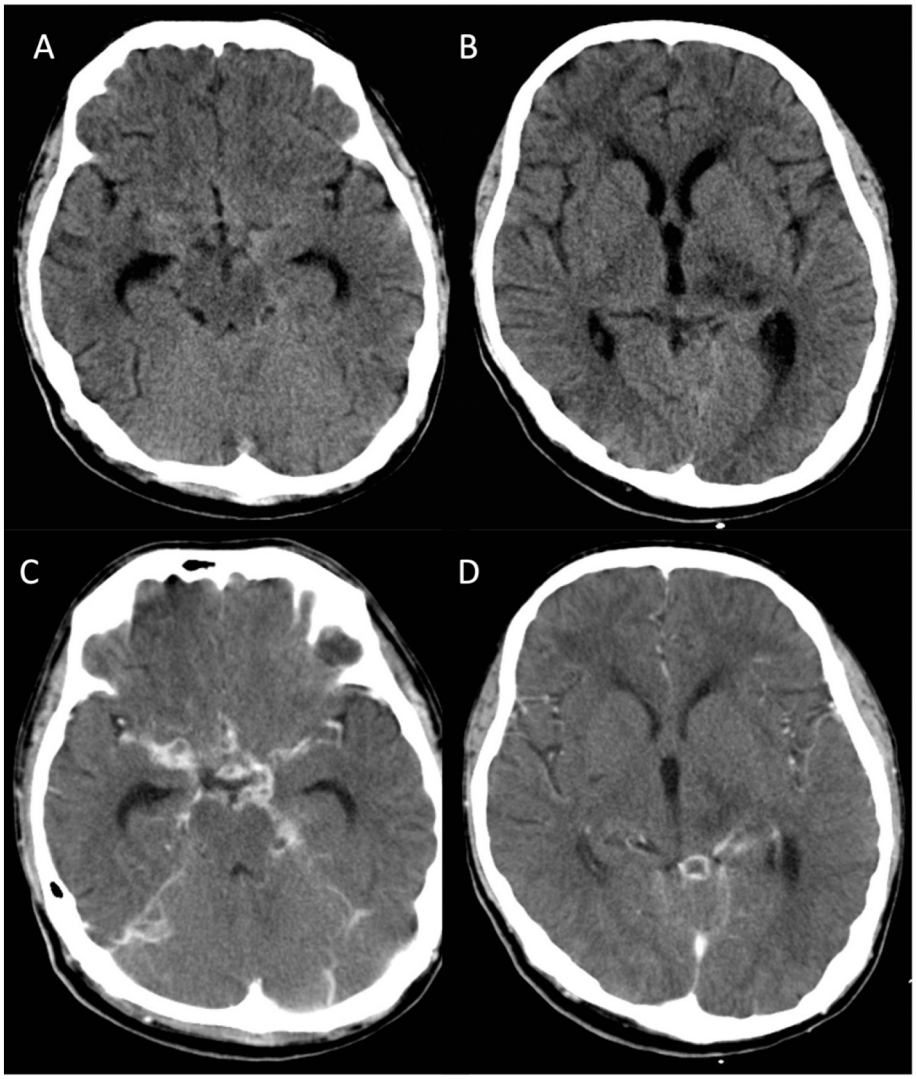

Fig. 1. Head CT scan. Noncontrast CT scan showed enlargement of temporal horn (A) and infarct on the left thalamus (B). Following contrast administration, there was basal enhancement (C) and granuloma lesion on the quadrigeminal cistern (D).

Antitubercular chemotherapies (rifampicin, isoniazid, pyrazinamide, ethambutol, and streptomycin) were given along with mannitol and dexamethasone. He remained in stable condition for three days, but on the fourth day, his conscious state dropped to GCS 8/15. He developed paroxysmal limb stiffness with fever of 40 °C. Heart rate was increased to 150–160 times per minute with increased of respiratory rate to 30–40 times per minute, along with excessive sweating. These symptoms occurred in 5 to 10 min, up to five times a day. Laboratory examination suggested no secondary infections with normal procalcitonin. Electroencephalogram revealed no epilepsy wave. Brain CT Scan showed increased of ventricular size with stable tuberculoma size.

Based on clinical findings, paroxysmal sympathetic hyperactivity was considered a working diagnosis. We gave fentanyl, propranolol, and gabapentin in addition with cooling blanket. Frequency of the symptoms was reduced, but the GCS remained 8/15 until he was discharged from intensive care unit three weeks after. After given antitubercular therapy for three months later, the patient's GCS rose to 10/15 and underwent rehabilitative treatment.

3. Clinical discussion

Tuberculosis remains a significant public health issue around the world, in which Indonesia is a one of the most significant contributors to the global burden [9]. Tuberculosis of brain, such as tuberculous meningitis and tuberculoma, is the most lethal form of tuberculosis with up to 40 % mortality [10,11]. The primary management goals are to reduce intracranial pressure and improve cerebral perfusion, while also to eradicate the bacteria and to lessen intracerebral inflammation [12].

We observed a paradoxical reaction in this patient following antitubercular treatment. The patient had received antitubercular medication for one month but suffered from diminished neurological function. The CNS and respiratory system usually are the most involved sites in paradoxical worsening reported in the literature [13,14]. Even though the pathogenesis is poorly understood, this is commonly regarded as a response of inflammation to dead bacteria [15]. Increased protein level in CSF, that was also observed in our patient, is considered as risk factor [16]. Even though concomitant HIV infection is known as important predictor [17], paradoxical reaction is common in non-HIV patient with incident ranged from 30 % to 50 %, as reported in our case [18,19]. A paradoxical reaction can arise 2–3 months after starting antitubercular drugs [20]. Our patient experienced several CNS symptoms four weeks after treatment. Although paradoxical responses may cause confusion in the care of tuberculosis, especially considering drug resistance, it is not required to alter or discontinue anti-TB treatment when a paradoxical reaction occurs [21]. Even so, drug resistance is a notable consideration, especially in countries with high incident of tuberculosis such as Indonesia [22,23]. Bacterial culture and sensitivity test, despite being the gold standard, is only positive in about 40 % of cases and can take up to 6 weeks to produce a positive result [24]. When compared to culture, the GeneXpert test performs well in detecting rifampicin resistance in significantly less time [25].

The patient presented with enlargement of ventricular system and papilledema, suggestive of increased intracranial pressure. Unfortunately, due to facility limitations, we were unable to monitor intracranial pressure in our patient. The gold standard to measure intracranial pressure is invasive method that can be performed at epidural, intraparenchymal, or intraventricular [26]. However, it is still questionable if intracranial pressure monitoring will direct treatment or improve outcomes in tuberculous meningitis.

Increased intracranial pressure leads to the poor prognosis of tuberculous meningitis patients. Due to the existence of hydrocephalus, we inserted a ventriculoperitoneal shunt to lower intracranial pressure in this patient. However, pathology in ganglia basal, diencephalon, and midbrain, such as ischemia or inflammation, may also contribute to decrease of consciousness [27]. As in our instance, granulation lesions were suspected in the midbrain and thalamus. It may have been the reason why there was no increase in our degree of consciousness. The patient was in grade 3 based on modified Vellor grading (Table 1) [28]. External ventricular drainage (EVD) is another alternative for cerebrospinal fluid diversion. The decision to undertake shunt surgery is determined by the patient's response to EVD in a duration of time. Those who advocate for the use of EVD contend that global shunting in this patient is unsuitable due to the patient's high mortality and morbidity rates [29]. EVD is nevertheless impractical and susceptible to infection [30]. In addition, a prospective investigation revealed that the response of EVD did not correlate with the outcome of Vellore grade 3 patients [31].

Another alternative of CSF diversion is endoscopic third ventriculostomy (ETV). ETV is an endoscopic treatment that connects blocked CSF to the pre-pontine cistern via a stoma, allowing access to parts of the brain that may have normal CSF absorption [32]. However, ETV may be challenging in tuberculous meningitis because to the thicker third ventricular floor and larger basal exudates [33]. Both ventriculoperitoneal shunting and ETV pose a high risk of infection and bleeding [34]. Since there is limited high quality data, the choice between medical treatment for hydrocephalus, EVD, ETV, and ventriculoperitoneal shunting remains complex and debatable.

Four days following surgery, the patient developed episodic hyperactivity of sympathetic system. Paroxysmal sympathetic hyperactivity (PSH) is an episodic of sympathetic and/or motor hyperactivity in patients with an acquired brain injury. Increased respiratory rate, fever, diaphoresis, tachycardia, and dystonic posturing are some of the symptoms observed [35]. Although PSH has been found in various condition related to acquired brain injury such as stroke, infection, and anoxic injury, most reports regarding this condition are in clinical context of traumatic brain injury [1].

The precise pathogenic process is still unknown. There is no association between PSH and the severity of brain injury. Currently, the disconnection theory and the excitatory: inhibitory ratio (EIR) model are the most frequently recognized theories [35,36]. According to disconnection hypotheses, PSH followed the release of one or more excitatory centers from control by a higher center. The EIR model proposes that PSH developed from disconnection and/or imbalance between the sympathetic and parasympathetic nervous systems, resulting in sympathetic hyperactivity. However, PSH may result in secondary brain injury, extend hospitalization, and impose a large expense on the medical system. During an episode, hyperventilation and elevated blood pressure can cause brain tissue hypoxia, worsen cerebral oedema, and increase intracranial pressure [3,37].

Diagnosing PSH may be difficult, even with the available diagnostic criteria [38]. PSH must be differentiated from sepsis, central high fever, seizure, and withdrawal syndrome. If a patient's conscious status deteriorated severely, PSH could be mistaken for an epileptic seizure. Therefore, EEG is mandatory. In our case, the EEG did not reveal any epileptic changes. In addition, because it is difficult to differentiate PSH episodes from sepsis or infection, antibiotics misuse is common. Regular close monitoring of the infection marker should be conducted [2]. Malignant hyperthermia, thyroid storm, and narcotic withdrawal syndrome are among the differential diagnoses for patient with such condition like PSH.

Symptom elimination and symptom prevention, along with definitive treatment, are the main treatment approaches. As a result of their

Table 1
Modified Vellore grading [28].

Grade	
1	GCS 15 with no neurological deficit
2	GCS 15 with neurological deficit
3	GCS 9–14, with or without neurological deficit
4	GCS 3–8, with or without neurological deficit

GCS: Glasgow Coma Scale.

rapid onset and short half-life, symptom-relieving medicines can immediately terminate paroxysmal episodes. In general, these drugs aim to reduce fever in hyperthermia, control heart rate in tachycardia, dynamically manage blood pressure, provide timely and appropriate sedation, and relieve spasticity or decrease muscular tone. There are numerous pharmacological classes, such as non-selective β-blockers, α2-agonists, gabapentin, baclofen, bromocriptine, and long-acting benzodiazepines, that have demonstrated considerable clinical success. If symptoms persist, a continuous infusion of propofol, benzodiazepines, opioids, or dexmedetomidine may be administered until the symptoms decrease [39,40]. Importantly, physicians should be aware that combination therapy may be required to avoid persistent episodes, and that prophylactic drugs should be evaluated first for difficult-to-control persistent and chronic symptoms [2]. In our cases, episodes were successfully managed using fentanyl, propranolol, and gabapentin. We also used cooling blanket to prevent the symptoms. For hyperthermia patients, it is beneficial to regulate the temperature of the room to give a less stimulating setting and to provide daily care [41]. A recent pilot study demonstrated that a lower room temperature is correlated with PSH, suggesting that environmental treatments, e.g., application of cooling blanket, may supplement medicinal therapies [42].

We reported an unfavorable outcome in our patient. Although PSH is usually associated with brain injury, it is problematic to declare that poor clinical results or higher morbidity are independently driven by these dysautonomia in TBI patients [1]. However, in an effort to reduce long-term disability in individuals with severe TBI, early detection of PSH may be beneficial [3,35].

4. Conclusion

Paradoxical reaction following antitubercular drugs may lead to confusion in management. Drug resistance test should always be performed. Ventriculoperitoneal shunting, external ventricular drainage, and endoscopic third ventriculostomy are surgical approaches available in treatment of hydrocephalus following tuberculous meningitis. Since there is no strong evidence, patient's clinical condition is the main consideration. Early identification of paroxysmal sympathetic hyperactivity is mandatory to reduce long term disability in individuals with brain tuberculosis.

Funding

None.

Ethical approval

None declared.

Consent

Written informed consent was obtained from the patient's family for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

CRediT authorship contribution statement

AMPS, ST, SI = Patient management (including surgery), study concept, data collection.

AMPS = Writing- original draft preparation.

AMPS, BWMN, MS = Editing and writing.

AMPS, ST, SI = senior author and manuscript reviewer.

Registration of research studies

None.

Guarantor

Andre Marolop Pangihutan Siahaan.

Provenance and peer review

Not commissioned, externally peer reviewed.

Declaration of competing interest

There is no conflict of interest.

References

[1] G. Meyfroidt, I.J. Baguley, D.K. Menon, Paroxysmal sympathetic hyperactivity: the storm after acute brain injury, Lancet Neurol. 16 (2017) 721–729, https://doi.org/10.1016/S1474-4422(17)30259-4.

[2] R.Z. Zheng, Z.Q. Lei, R.Z. Yang, G.H. Huang, G.M. Zhang, Identification and management of paroxysmal sympathetic hyperactivity after traumatic brain injury, Front. Neurol. 11 (2020) 81, https://doi.org/10.3389/FNEUR.2020.00081/BIBTEX.

[3] J.F. Fernandez-Ortega, M.A. Prieto-Palomino, M. Garcia-Caballero, J.L. Galeas-Lopez, G. Quesada-Garcia, I.J. Baguley, Paroxysmal sympathetic hyperactivity after traumatic brain injury: clinical and prognostic implications, J. Neurotrauma 29 (2012) 1364–1370, https://doi.org/10.1089/NEU.2011.2033.

[4] D.J. Nicolls, M. King, D. Holland, J. Bala, C. del Rio, Intracranial tuberculomas developing while on therapy for pulmonary tuberculosis, Lancet Infect. Dis. 5 (2005) 795–801, https://doi.org/10.1016/S1473-3099(05)70299-1.

[5] A.K. Singh, H.S. Malhotra, R.K. Garg, A. Jain, N. Kumar, N. Kohli, R. Verma, P. K. Sharma, Paradoxical reaction in tuberculous meningitis: presentation, predictors and impact on prognosis, BMC Infect. Dis. 16 (2016) 1–11, https://doi.org/10.1186/S12879-016-1625-9/FIGURES/3.

[6] T. Raut, R.K. Garg, A. Jain, R. Verma, M.K. Singh, H.S. Malhotra, N. Kohli, A. Parihar, Hydrocephalus in tuberculous meningitis: incidence, its predictive factors and impact on the prognosis, J. Infect. 66 (2013) 330–337, https://doi.org/10.1016/J.JINF.2012.12.009.

[7] V. Rajshekhar, Management of hydrocephalus in patients with tuberculous meningitis, Neurol. India 57 (2009) 368, https://doi.org/10.4103/0028-3886.55572.

[8] R.A. Agha, T. Franchi, C. Sohrabi, G. Mathew, A. Kerwan, A. Thoma, A.J. Beamish, A. Noureldin, A. Rao, B. Vasudevan, B. Challacombe, B. Perakath, B. Kirshtein, B. Ekser, C.S. Pramesh, D.M. Laskin, D. Machado-Aranda, D. Miguel, D. Pagano, F. H. Millham, G. Roy, H. Kadioglu, I.J. Nixon, I. Mukhejree, J.A. McCaul, J. Chi-Yong Ngu, J. Albrecht, J.G. Rivas, K. Raveendran, L. Derbyshire, M.H. Ather, M. A. Thorat, M. Valmasoni, M. Bashashati, M. Chalkoo, N.Z. Teo, N. Raison, O. J. Muensterer, P.J. Bradley, P. Goel, P.S. Pai, R.Y. Afifi, R.D. Rosin, R. Coppola, R. Klappenbach, R. Wynn, R.L. de Wilde, S. Surani, S. Giordano, S. Massarut, S. G. Raja, S. Basu, S.A. Enam, T.G. Manning, T. Cross, V.K. Karanth, V. Kasivisvanathan, Z. Mei, S.C.A.R.E. The, Guideline: updating consensus surgical CAse REport (SCARE) guidelines, Int. J. Surg. 84 (2020) (2020) 226–230, https://doi.org/10.1016/J.IJSU.2020.10.034.

[9] J. Chakaya, M. Khan, F. Ntoumi, E. Aklillu, R. Fatima, P. Mwaba, N. Kapata, S. Mfinanga, S.E. Hasnain, P.D.M.C. Katoto, A.N.H. Bulabula, N.A. Sam-Agudu, J. B. Nachega, S. Tiberi, T.D. McHugh, I. Abubakar, A. Zumla, Global Tuberculosis Report, – reflections on the global TB burden, treatment and prevention efforts, Int. J. Infect. Dis. 113 (2021) (2020) S7–S12, https://doi.org/10.1016/J.IJID.2021.02.107.

[10] P.J. Dodd, M. Osman, A.M. Stadelman, N.Huu Lan, N.Thuy Thuong Thuong, M. Muzyamba, L. Glaser, S.S. Dlamini, J.A. Seddon, F.v. Cresswell, P.Ngoc Thach Hospital, H.Chi Minh City, The global burden of tuberculous meningitis in adults: a modelling study, PLoS Global Public Health 1 (2021), e0000069, https://doi.org/10.1371/JOURNAL.PGPH.0000069.

[11] A. Navarro-Flores, J.E. Fernandez-Chinguel, N. Pacheco-Barrios, D.R. Soriano-Moreno, K. Pacheco-Barrios, Global morbidity and mortality of central nervous system tuberculosis: a systematic review and meta-analysis, J. Neurol. 269 (2022) 3482–3494, https://doi.org/10.1007/S00415-022-11052-8/TABLES/4.

[12] J. Donovan, A. Figaji, D. Imran, N.H. Phu, U. Rohlwink, G.E. Thwaites, The neurocritical care of tuberculous meningitis, Lancet Neurol. 18 (2019) 771–783, https://doi.org/10.1016/S1474-4422(19)30154-1.

[13] R.K. Garg, H.S. Malhotra, N. Kumar, Paradoxical reaction in HIV negative tuberculous meningitis, J. Neurol. Sci. 340 (2014) 26–36, https://doi.org/10.1016/J.JNS.2014.03.025.

[14] R.A.M. Breen, C.J. Smith, H. Bettinson, S. Dart, B. Bannister, M.A. Johnson, M.C. I. Lipman, Paradoxical reactions during tuberculosis treatment in patients with and without HIV co-infection, Thorax 59 (2004) 704–707, https://doi.org/10.1136/THX.2003.019224.

[15] N.T.T. Thuong, G.E. Thwaites, Treatment-associated inflammatory deterioration in tuberculous meningitis: unpicking the paradox, J. Infect. Dis. 215 (2017) 665, https://doi.org/10.1093/INFDIS/JIW565.

[16] R. Gupta, R.K. Garg, A. Jain, H.S. Malhotra, R. Verma, P.K. Sharma, Spinal cord and spinal nerve root involvement (myeloradiculopathy) in tuberculous

meningitis, Medicine 94 (2015), e404, https://doi.org/10.1097/MD.0000000000000404.
[17] A.K. Singh, H.S. Malhotra, R.K. Garg, A. Jain, N. Kumar, N. Kohli, R. Verma, P. K. Sharma, Paradoxical reaction in tuberculous meningitis: presentation, predictors and impact on prognosis, BMC Infect. Dis. 16 (2016) 1–11, https://doi.org/10.1186/S12879-016-1625-9/FIGURES/3.
[18] M.L.S. Tai, H.M. Nor, K.A.A. Kadir, S. Viswanathan, K. Rahmat, N.R.M. Zain, K. G. Ong, M.H. Rafia, C.T. Tan, Paradoxical manifestation is common in HIV-negative tuberculous meningitis, Medicine 95 (2016), https://doi.org/10.1097/MD.0000000000001997.
[19] P.N. Sütlas, A. Ünal, H. Forta, S. Şenol, D. Kirbaş, Tuberculous meningitis in adults: review of 61 cases, Infection 31 (2003) 387–391, https://doi.org/10.1007/S15010-003-3179-1.
[20] R.K. Garg, H.S. Malhotra, N. Kumar, Paradoxical reaction in HIV negative tuberculous meningitis, J. Neurol. Sci. 340 (2014) 26–36, https://doi.org/10.1016/J.JNS.2014.03.025.
[21] R. Domínguez-Moreno, M. García-Grimshaw, D. Medina-Julio, C. Cantú-Brito, A. González-Duarte, Paradoxical manifestations during tuberculous meningitis treatment among HIV-negative patients: a retrospective descriptive study and literature review, Neurological Sciences 43 (4) (2021) 2699–2708, https://doi.org/10.1007/S10072-021-05693-2.
[22] S.C. van Kampen, N.H. Susanto, S. Simon, S.D. Astiti, R. Chandra, E. Burhan, M. N. Farid, K. Chittenden, D.E. Mustikawati, B. Alisjahbana, Effects of introducing xpert MTB/RIF on diagnosis and treatment of drug-resistant tuberculosis patients in Indonesia: a pre-post intervention study, PLoS ONE 10 (2015), e0123536, https://doi.org/10.1371/JOURNAL.PONE.0123536.
[23] L. Chaidir, C. Ruesen, B.E. Dutilh, A.R. Ganiem, A. Andryani, L. Apriani, M. A. Huynen, R. Ruslami, P.C. Hill, R. van Crevel, B. Alisjahbana, Use of whole-genome sequencing to predict mycobacterium tuberculosis drug resistance in Indonesia, J. Glob. Antimicrob. Resist. 16 (2019) 170–177, https://doi.org/10.1016/J.JGAR.2018.08.018.
[24] A.I. Bhigjee, R. Padayachee, H. Paruk, K.D. Hallwirth-Pillay, S. Marais, C. Connoly, Diagnosis of tuberculous meningitis: clinical and laboratory parameters, Int. J. Infect. Dis. 11 (2007) 348–354, https://doi.org/10.1016/J.IJID.2006.07.007.
[25] N.T.Q. Nhu, D. Heemskerk, D.D.A. Thu, T.T.H. Chau, N.T.H. Mai, H.D.T. Nghia, P. P. Loc, D.T.M. Ha, L. Merson, T.T. van Thinh, J. Day, N. van Vinh Chau, M. Wolbers, J. Farrar, M. Caws, Evaluation of GeneXpert MTB/RIF for diagnosis of tuberculous meningitis, J. Clin. Microbiol. 52 (2014) 226–233, https://doi.org/10.1128/JCM.01834-13.
[26] X. Zhang, J.E. Medow, B.J. Iskandar, F. Wang, M. Shokoueinejad, J. Koueik, J. G. Webster, Invasive and noninvasive means of measuring intracranial pressure: a review, Physiol. Meas. 38 (2017) R143–R182, https://doi.org/10.1088/1361-6579/AA7256.
[27] B.M. McClenathan, N.v. Thakor, R.E. Hoesch, Pathophysiology of acute coma and disorders of consciousness: Considerations for diagnosis and management, Seminars in Neurology. 33 (2013) 91–109, https://doi.org/10.1055/S-0033-1348964/ID/JR00857-34.
[28] V. Rajshekhar, Three decades of Vellore grading for tuberculous meningitis with hydrocephalus: a reappraisal, Neurol. India 69 (2021) 569, https://doi.org/10.4103/0028-3886.332251.
[29] R. Harrichandparsad, S.S. Nadvi, M.Y.Suleman Moosa, J.Rikus van Dellen, Outcome of ventriculoperitoneal shunt surgery in human immunodeficiency virus-positive patients on combination antiretroviral therapy with tuberculosis meningitis and hydrocephalus, World Neurosurg. 123 (2019) e574–e580, https://doi.org/10.1016/J.WNEU.2018.11.221.
[30] K.R.I.S. Dorresteijn, M.C. Brouwer, K. Jellema, D. van de Beek, in: Bacterial External Ventricular Catheter-associated Infection 18, 2020, pp. 219–229, https://doi.org/10.1080/14787210.2020.1717949.
[31] M. JM, R. V, C. MJ, Shunt surgery in poor grade patients with tuberculous meningitis and hydrocephalus: effects of response to external ventricular drainage and other variables on long term outcome, J. Neurol. Neurosurg. Psychiatry 65 (1998) 115–118, https://doi.org/10.1136/JNNP.65.1.115.
[32] P. Goyal, C. Srivastava, B.K. Ojha, S.K. Singh, A. Chandra, R.K. Garg, S. Srivastava, A randomized study of ventriculoperitoneal shunt versus endoscopic third ventriculostomy for the management of tubercular meningitis with hydrocephalus, Childs Nerv. Syst. 30 (2014) 851–857, https://doi.org/10.1007/S00381-014-2371-1.
[33] Y.R. Yadav, V. Parihar, M. Agrawal, P.R. Bhatele, Endoscopic third ventriculostomy in tubercular meningitis with hydrocephalus, Neurol. India 59 (2011) 855, https://doi.org/10.4103/0028-3886.91365.
[34] A. Aranha, A. Choudhary, S. Bhaskar, L. Gupta, A randomized study comparing endoscopic third ventriculostomy versus ventriculoperitoneal shunt in the Management of Hydrocephalus due to tuberculous meningitis, Asian J. Neurosurg. 13 (2018) 1140, https://doi.org/10.4103/AJNS.AJNS_107_18.
[35] I. Perkes, I.J. Baguley, M.T. Nott, D.K. Menon, A review of paroxysmal sympathetic hyperactivity after acquired brain injury, Ann. Neurol. 68 (2010) 126–135, https://doi.org/10.1002/ANA.22066.
[36] I.J. Baguley, R.E. Heriseanu, I.D. Cameron, M.T. Nott, S. Slewa-Younan, A critical review of the pathophysiology of dysautonomia following traumatic brain injury, Neurocrit. Care 8 (2008) 293–300, https://doi.org/10.1007/S12028-007-9021-3.
[37] H.A. Choi, S.B. Jeon, S. Samuel, T. Allison, K. Lee, Paroxysmal sympathetic hyperactivity after acute brain injury, Curr. Neurol. Neurosci. Rep. 13 (2013), https://doi.org/10.1007/S11910-013-0370-3.
[38] I.J. Baguley, I.E. Perkes, J.F. Fernandez-Ortega, A.A. Rabinstein, G. Dolce, H. T. Hendricks, Paroxysmal sympathetic hyperactivity after acquired brain injury: consensus on conceptual definition, nomenclature, and diagnostic criteria, J. Neurotrauma 31 (2014) 1515–1520, https://doi.org/10.1089/NEU.2013.3301.
[39] S. Samuel, T.A. Allison, K. Lee, H.A. Choi, Pharmacologic Management of Paroxysmal Sympathetic Hyperactivity after Brain Injury, J Neurosci Nurs. 48 (2016) 82–89, https://doi.org/10.1097/JNN.0000000000000207.
[40] A.A. Rabinstein, Paroxysmal sympathetic hyperactivity in the neurological intensive care unit, Neurol. Res. 29 (2007) 680–682, https://doi.org/10.1179/016164107X240071.
[41] K.A. Kirk, M. Shoykhet, J.H. Jeong, E.C. Tyler-Kabara, M.J. Henderson, M.J. Bell, E.L. Fink, Dysautonomia after pediatric brain injury, developmental medicine and child, Neurology 54 (2012) 759, https://doi.org/10.1111/J.1469-8749.2012.04322.X.
[42] L. Letzkus, N. Addison, L. Turner, M. Conaway, B. Quatrara, Paroxysmal sympathetic hyperactivity and environmental factors: a pilot study, J Neurosci Nurs. 50 (2018) 88–92, https://doi.org/10.1097/JNN.0000000000000349.