Open

Citation: J Med Discov (2020); 5(3):jmd20037; DOI:10.24262/jmd.5.3.20037

Case Report

Intracranial Tuberculoma in a Trisomy 21 Patient: A Case Report

Maryam Hannoodee[1,*], Raed Al-Naser[2]

[1]*University of Baghdad, Al-Kindy college of medicine, Baghdad/Iraq.*

[2]*Pulmonary and Critical Care Medicine specialist, Sharp Grossmont Hospital, La mesa, CA 91942.*

Abstract Tuberculosis may involve the central nervous system (CNS), causing a variety of presentations from common meningitis, cerebritis, abscesses, spinal tuberculosis, arachnoiditis to rare tuberculoma. It may also cause lethal CNS complications [1-4]. CNS tuberculosis is more common in children, malnourished, young adults, immunocompromised, alcoholic, and cancer patients [5]. Patients with trisomy 21 are at more risk of developing stroke-like signs and symptoms of focal neurological deficits resulting from cardiovascular defects with or without systemic manifestations [6]. Here, we present a case of intracranial tuberculoma in an 18-year-old female with trisomy 21 who was born in Guatemala and was admitted through the ED with left-sided arm and leg weakness without other systemic manifestations of stroke such as headache, neck pain, vision changes, difficulty speaking or swallowing. After thorough diagnostic tests by MRI brain, CT chest, CT biopsy of the lung, and video-assisted thoracoscopic wedge resection, she was eventually diagnosed with a right inferior basal ganglia tuberculoma presumptive hematogenous dissemination from a tuberculous nodule in the right upper lobe of the lung. The patient responded well to medical management with anti-tuberculous drugs and steroids. We concluded that intracranial tuberculoma needs to be considered in the differential diagnosis of patients with trisomy 21 presented with neurological signs of a stroke, mainly if they live in or have emigrated from areas with a high prevalence of tuberculosis. To the best of our knowledge, this is the second case report of brain tuberculoma described in a child with trisomy 21 [7].

Keywords: Tuberculosis, tuberculoma, dissemination, trisomy 21, case report

How to cite: Maryam Hannoodee et al., Intracranial Tuberculoma in a Trisomy 21 Patient: A Case Report. J Med Discov (2020); 5(3):jmd20037; DOI:10.24262/jmd.5.3.20037; Received May 25th, 2020, Revised July 16th, 2020, Accepted August 10th, 2020, Published August 18th, 2020.

Introduction

Brain tuberculoma is considered one of the rare severe extra-pulmonary complications caused by hematogenous dissemination of tuberculosis [8]. It accounts for about 5-10% of extra-pulmonary TB cases and 1% of all TB cases [9]. Early intervention is required due to the high morbidity and mortality associated with it.

The TB bacilli itself does not cause clinical presentations nor their antigens. The clinical presentations occur because of the space-occupying lesion causing pressure effects [10]. Generally, brain tuberculoma is asymptomatic [11], but the symptoms mostly depend on the anatomical location within the brain [12].

The neuroimaging and histopathological examination results make the diagnosis established. Medical management is preferred over surgery, which is reserved for refractory cases.

Case presentation

An 18-year-old trisomy 21 female who was born in Guatemala and recently emigrated with her family to the United States was brought to the emergency department of San Diego County California local hospital by her parents with a chief complaint of 3 months left-sided upper and lower extremities weakness which was progressively worsened over the past two weeks. The patient was no

* Correspondence: Raed Al-Naser, *Sharp Grossmont Hospital, La mesa, CA 91942.* . Email: maryomadr@gmail.com.

longer able to untie her shoes or walk with a normal gait secondary to the weakness, which warned the family to bring her for further investigation. She denies any history of headache, neck pain, vision changes, difficulty speaking or swallowing, chest pain, or paranesthesia. The patient does not have a past medical or surgical history, and she was not taking any medication. Her vital signs upon admission showed a blood pressure of 108/66 mm Hg and a temperature of 36.6 C (97.9 F), a heart rate of 80 bpm, respiration is 16 breaths/min, spo2 is 99%.

The initial clinical examination of the patient revealed that she had features consistent with trisomy 21, clinically stable with normal general systemic examination. A CNS examination of the left upper and lower extremity revealed weakness, hemiplegic gait, brisk reflexes, and an up-going toe. Otherwise, the patient had intact higher functions and cranial nerves as well as normal fundal examination results. Investigation showed neutropenia and anemia. Coagulation studies, liver, and renal function tests were all within normal limits. The patient's chest x-ray was read as normal.

Upon admission, a Head CT scan without contrast was done. It showed 1.5x 0.8x 1.0 cm what was described by radiologist as intraparenchymal hemorrhage centered in the posterior right putamen/posterior limb of the right internal capsule with surrounding vasogenic edema (Figure 1).

The patient underwent MRI of the brain with/without contrast for further evaluation. Brain MRI revealed a loculated lesion centered in the inferior basal ganglia with peripheral enhancement and several adjacent foci of nodular enhancement. Radiologist indicated that findings were highly suspicious of neoplasm either primary versus metastatic, including lymphoma or possibly infection (Figure 2). The patient underwent a CT scan of the chest/abdomen/pelvis, Chest CT scan showed two right lung masses. The first was lobular with calcification in the right lower lobe (Figure 3), and the second was measuring 1.5 cm diameter on the right apical lobe with high malignancy suspicion, and biopsy was considered (Figure 4).

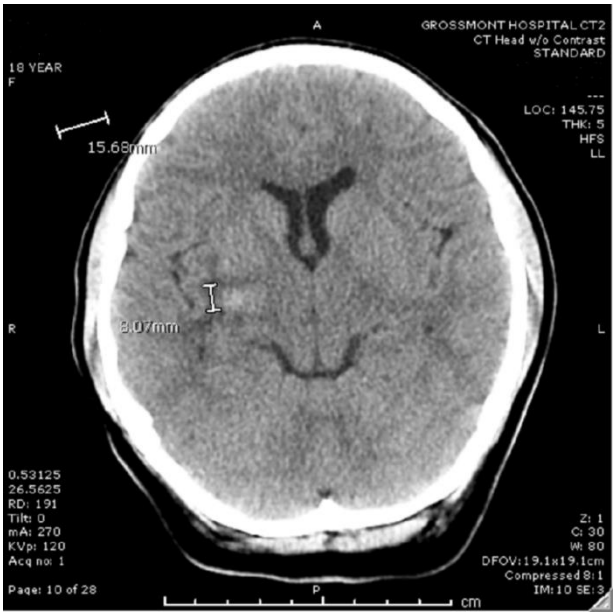

Figure 1: Computed Tomography (CT) of the brain without contrast showing amorphous focus of hyper-density measuring approximately 1.5 x 0.8 x 1 cm consistent with acute hemorrhage centered in the posterior right putamen/posterior limb of the right internal capsule with a rim of hypodensity consistent with vasogenic edema.

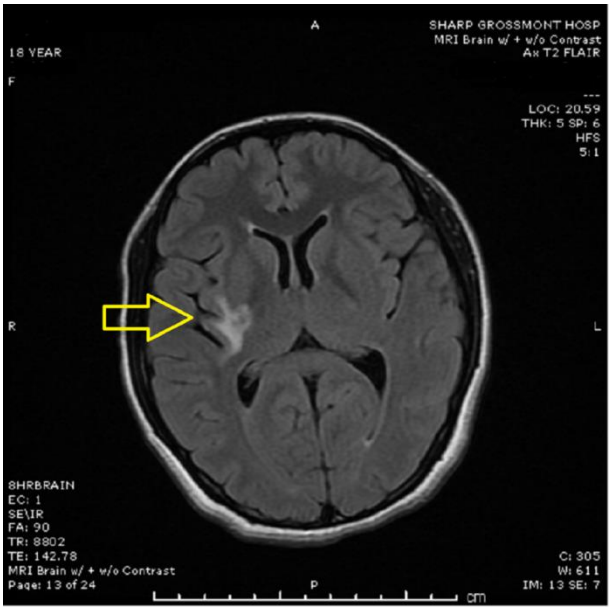

Figure 2: Magnetic resonance imaging (MRI) of the brain with contrast showing a lobulated lesion measuring approximately 1.6 cm in maximal dimension (yellow arrow) centered in the inferior right basal ganglia and with peripheral enhancement

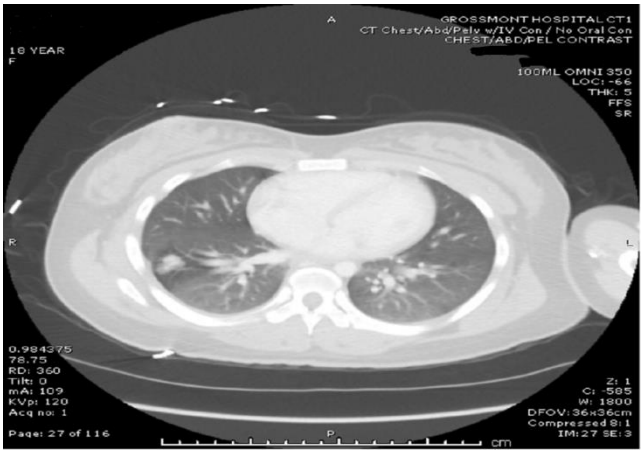

Figure 3: First lung mass. A lobular mass with calcification in the right lower lobe has features suggestive of a hamartoma and a 1.5 cm diameter right apical mass is nonspecific and more suspicious for a neoplasm

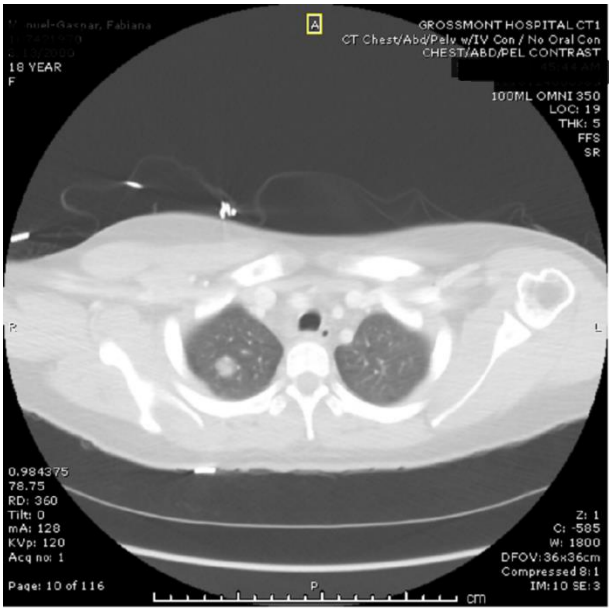

Figure 4: Second lung mass was measuring 1.5 cm diameter on the right apical lobe with high malignancy suspicion.

CT scan of the abdomen/pelvis showed focal thickening of the anterior bladder wall, potentially related to a urachal anomaly versus neoplasm. Cystoscopy was considered for correlation.

CT guided biopsy of the right upper lobe lesion has been done, which showed necrotizing granulomatous inflammation with multinucleated giant cells and extensive necrosis. No evidence of malignancy identified. GMS stain for fungus was negative, and the fluorescent auramine stain for the acid-fast organism was also negative.

A spinal tap was performed after that and cytology exam was negative for malignant cells. CSF stains for AFB and fungal organisms were negative.

Video-assisted thoracoscopic right upper lobe and right lower lobe segmentectomy were done. The biopsy confirmed granulomatous inflammation with central necrosis. Stains for acid-fast and fungal organisms were negative on both right upper and right lower lobe resected segments. The right lung upper lobe segmentectomy MTB complex PCR, paraffin block assay was positive (Mycobacterium tuberculosis organism detected).

Tissue culture from the right upper lobe segment eventually confirmed the growth of Mycobacterium tuberculosis. The organism was sensitive to INH, rifampin, ethambutol, and pyrazinamide.

The specimens eventually confirmed Mycobacterium tuberculosis based on MTB PCR and final cultures. The diagnosis of tuberculosis with brain tuberculoma as extra-pulmonary complication through hematogenous spread has been made. Subsequent lab results showed that the patient has a positive QuantiFERON test.

Discussion

Tuberculosis infection is caused by the inhalation of the respiratory droplets containing mycobacterium, leading to the activation of the helper T-cell within the lung's alveoli, leading to granuloma formation. Some bacilli can be disseminated through the blood to different parts of the body that are highly oxygenated like the brain [13]. Patients may present with non-specific findings like headaches, fever and

weight loss, nausea, photophobia, vomiting, sleep disturbance, and seizures [14-17]. Altered mental status and focal neurological signs were other common clinical manifestations [18-21] caused by the compression effects of the tuberculoma that may produce these unusual neurological symptoms. These were the findings in our patient, who presented to the emergency department with left-sided upper and lower extremities weakness. Clinical presentations of intracranial tuberculoma usually are not due to tubercle bacilli or its antigens but due to pressure effects of a space-occupying lesion. Neurological symptoms have evolved over the past three months of presentation in the absence of fever, headache, nausea/vomiting, or any other constitutional manifestations.

Diagnostic evaluation of the intracranial tuberculoma started with proper history taking, physical exam with a thorough neurological exam, sputum acid-fast stain, QuantiFERON TB test, CT scan of the brain and chest, and MRI of the brain [22,23]. A definitive diagnosis is made by tissue biopsy, which demonstrates TB bacilli within the biopsied tissue [24].

Medical therapy is currently the recommended treatment for CNS tuberculosis. The regimen of isoniazid, ethambutol, pyrazinamide, rifampicin, and steroids diminishes tuberculoma size and complete resolution within three months [25]. Our patient showed gradual improvement in her power and gait with medical therapy. Surgery is usually reserved for patients who do not respond to medical treatment, mid-line shift, compression of the brainstem and spinal cord or presents with obstructive hydrocephalus [26,27].

Conclusion

Trisomy 21 patients are at increased risk for stroke. Our patient had focal neurological manifestations of stroke due to intracranial tuberculoma. Intracranial tuberculoma should be considered in patients present with neurological deficits and increased intracranial pressure signs with no symptoms of systemic illness, especially if they come from a high prevalence area of tuberculosis. Surrogate diagnosis is made by a CT/MRI brain along with other radiological imaging of the chest, abdomen, and pelvis. Tissue biopsy or body fluid sampling is considered the definitive diagnostic test revealing the TB bacilli from an extracranial site. Invasive brain biopsy should be generally avoided since it is a high-risk procedure. When there are no radiologic findings of extracranial tuberculosis, stereotactic brain biopsy is preferred over open craniotomy, and this might be a reasonable approach to establish the diagnosis. In areas where tuberculosis is endemic, an empirical treatment trial might be a feasible alternative [28]. In our patient who emigrated from a developing country where neurocysticercosis is also prevalent, establishing diagnosis was essential. Neurocysticercosis is a benign and self-limiting condition, whereas brain tuberculoma is an active infection requiring prolonged antituberculosis therapy [29].

To the best of our knowledge, this is the second case report of brain tuberculoma described in a child with trisomy 21 [7].

Medical therapy with anti-tuberculous medication and steroids is the mainstay of treatment with the surgical drainage reserved for non-responsive cases and is rarely needed [30].

Conflicts of Interest

None

Acknowledgments

None

References

1. B. Rock, M. Olin, C.A. Baker, T.W. Molitor, P.K. Peterson Central nervous system tuberculosis: pathogenesis and clinical aspects Clin Microbiol Rev, 21 (2) (2008), pp. 243-261
2. M. Phypers, T. Harris, C. Power CNS tuberculosis: a longitudinal analysis of epidemiological and clinical features Int J Tuberc Lung Dis, 10 (1) (2006), pp. 99-103
3. R. Monteiro, J.C. Carneiro, C. Costa, R. Duarte Cerebral tuberculomas - a clinical challenge
4. Respir Med Case Rep, 9 (2013), pp. 34-37 F. Venter, A. Heidari, K. Galang, M. ViehwegAn atypical presentation of tuberculomas in an immunocompetent host J Investig Med High Impact Case Rep, 6 (2018), Article 2324709618798407
5. R.B. Rock, M. Olin, C.A. Baker, T.W. Molitor, P.K. PetersonCe ntral nervous system tuberculosis: pathogenesis and clinical aspects Clinical Microbiology Reviews, 21 (2) (2008), pp. 243-261
6. Sobey CG, Judkins CP, Sundararajan V, Phan TG, Drummond GR, Srikanth VK (2015) Risk of Major Cardiovascular Events in People with Down Syndrome. PLoS ONE 10(9): e0137093. https://doi.org/10.1371/journal.pone.0137093
7. Brain tuberculoma, an unusual cause of stroke in a child with trisomy 21: A case report (Abdelmoneim E.M. Kheir, Salah A. Ibrahim,[…] Farouk A. Hamid (www.casereports.biomedcentral.com).
8. Kim TK, Chang KH, Kim CJ, Goo JM, Kook MC, Han MH: Intracranial tuberculoma: comparison of MR with pathologic findings. Am J Neuroradiol. 1995, 16:1903-8.
9. WHO Global Tuberculosis Report. (2019). Accessed: February 2,2019:https://www.who.int/tb/publications/global_report/tb19_Exec_Sum_12Nov2019.pdf?ua=1.
10. Mukherjee S, Das R, Begum S. Tuberculoma of the brain - a diagnostic dilemma: magnetic resonance spectroscopy a new ray of hope. J Assoc Chest Physicians. 2015;3:3–8.
11. Thwaites GE, Macmullen-Price J, Tran TH, Pham PM, Nguyen TD, Simmons CP, et al. Serial MRI to determine the effect of dexamethasone on the cerebral pathology of tuberculous meningitis: an observational study. Lancet Neurol. 2007;6(3):230–6.
12. Nicolls DJ, King M, Holland D, Bala J, del Rio C. Intracranial tuberculomas developing while on therapy for pulmonary tuberculosis. Lancet Infect Dis. 2005;5(12):795–801
13. Meegada S, Gyamfi R, Muppidi V, et al. (March 12, 2020) Multiple Intracranial Tuberculomas with an Intramedullary Spinal Cord Tuberculoma in a Pediatric Patient. Cureus 12(3): e7248. DOI 10.7759/cureus.7248
14. Gasparetto EL, Tazoniero P, Neto A, de C. Disseminated tuberculosis in a pregnant woman presenting with numerous brain tuberculomas: case report. Arq Neuropsiquiatr 2003.
15. Wasay M, Farooq S, Khowaja ZA, Bawa ZA, Ali SM, Awan S, et al. Cerebral infarction and tuberculoma in central nervous system tuberculosis: frequency and prognostic implications. J Neurol Neurosurg Psychiatry 2014, doi:http:// dx.doi.org/10.1136/jnnp-2013-307178.
16. Aloreidi K, Dodin J, Berg J, Hoffman W. Brain tuberculoma: a case report and literature review. South Dakota Med 2017;70:.
17. Bayindir C, Mete Ö, Bilgic B. Retrospective study of 23 pathologically proven cases of central nervous system tuberculomas. Clin Neurol Neurosurg 2006, doi:http://dx.doi.org/10.1016/j.clineuro.2005.03.001.
18. Unal A, Sutlas PN. Clinical and radiological features of symptomatic central nervous system tuberculomas. Eur J Neurol2005,doi:http://dx.doi.org/10.1111/j.1468-1331.2005.01067.x.
19. Kelly JD, Teeter LD, Graviss EA, Tweardy DJ. Intracranial tuberculomas in adults: A report of twelve consecutive patients in Houston. Texas. Scand J Infect Dis 2011, doi:http://dx.doi.org/10.3109/00365548.2011.586367.
20. Sahaiu-Srivastava S, Jones B. Brainstem tuberculoma in the immunocompetent: case report and literature review. Clin Neurol Neurosurg 2008, doi:http://dx.doi.org/10.1016/j.clineuro.2007.11.002.
21. Venter F, Heidari A, Galang K, Viehweg M. An atypical presentation of Tuberculomas in an immunocompetent host. J Investig Med High Impact Case Reports 2018, doi:http://dx.doi.org/10.1177/2324709618798407.
22. Bernaerts A, Vanhoenacker FM, Parizel PM, etal: Tuberculosis of the central nervous system: overview of neuroradiological findings. Eur Radiol. 2003, 13:1876-90. 10.1007/s00330-002-1608-7
23. Food and Drug Administration, Center for Devices and

24. Radiological Health . (2002). Accessed: February 2, 2019: http://www.fda.gov/cdrh/pdf/P010033b.pdf..
25. Bouchama A, Al-Kawi MZ, Kanaan I, Coates R, Jallu A, Rahm B, Siqueira EB: Brain biopsy in tuberculoma: the risks and benefits. Neurosurgery. 1991, 28:405-9. 10.1227/00006123-199103000-00011
26. Jacobs RF, Sunakorn P, Chotpitayasunonah T, Pope S, Kelleher K. Intensive short course chemotherapy for tuberculous meningitis. Pediatr Infect Dis J.1992;11(3):194–8.
27. Cárdenas G, Soto-Hernández JL, Orozco RV, Silva EG, Revuelta R, Amador JL: Tuberculous brain abscesses in immunocompetent patients: management and outcome. Neurosurgery.2010,67:1081-7.10.1227/NEU.0b013e3181eda396
28. Rajshekhar V: Management of hydrocephalus in patients with tuberculous meningitis . Neurol India. 2009, 57:368-74. 10.4103/0028-3886.55572
29. A. Mohanty, V. Santosh, B. Anadh, et al.Diagnostic efficacy of stereotactic biopsies in intracranial tuberculomas Surg Neurol, 52 (1999), pp. 252-258
30. Shetty G, Avabratha KS, Rai BS. Ring-enhancing lesions in the brain: a diagnostic dilemma. Iran J Child Neurol. 2014;8(3):61-64.
31. A. Rashid Choudhury (1989) Non-surgical Treatment of Tuberculomas of the Brain, British Journal of Neurosurgery, 3:6, 643-653, DOI: 10.3109/02688698908992687