

Year (Yıl) : 2018
Volume (Cilt) : 5
Issue Number (Sayı) : 2
Doi : 10.5455/JNBS.1527282016

Received/Geliş 25.05.2018
Accepted/Kabul 25.06.2018

PSYCHOSIS AND MIDDLE CRANIAL FOSSA ARACHNOID CYST; CASE REPORT AND LITERATURE REVIEW

PSİKOZ VE ORTA KRANYAL FOSSA ARAKNOİD KİSTİ; DURUM RAPORU VE LİTERATÜR İNCELEMESİ

Sharafaldeen Bin Nafisah*

Abstract

Controversies emerged in linking middle cranial fossa-arachnoid cyst to psychosis. Despite the anatomical association between such cyst and psychosocial behaviour, still several features of the cyst itself hamper causation. Here, I report a case of a young female labeled refractory schizophrenia who presented with antipsychotic toxicity overlooking an arachnoid cyst in the middle cranial fossa. Due to the paucity of information in the literature on such association here I discuss further implications from a first medical encounter, from a patient's perspective and from a disposition perspective as well.

Keywords: psychosis, middle cranial fossa, arachnoid cyst

Özet

Orta kranial fossa-araknoid kistin psikozla ilişkisinde bazı uyumsuzluklar bulunmaktadır. Kist ve psikososyal davranışlar arasındaki anatomik ilişkiye rağmen, kistin bazı özellikleri hala nedensizliği engeller. Mevcut raporda, orta kranial fossada bir araknoid kisti olan antipsikotik toksisite ile başvuran genç bir kadının refrakter şizofreni olgusunu sunuyorum. Literatürdeki bu tür bağlantılar hakkındaki bilginin yetersizliği nedeniyle, ilk tıbbi müdahaleden başlayarak hastanın bakış açısından ve eğiliminden bu tartışmayı derinleştirmek istiyorum.

Anahtar Kelimeler: psikoz, orta kranyal fossa, araknoid kisti

*King Fahd Medical City, Riyadh, Saudi Arabia

*Corresponding Author: King Fahd Medical City, Riyadh, 11375 Riyadh Saudi Arabia 395529 E-mail: dr.sharafaldeen@yahoo.com Phone: +966544443187

1. Introduction

Arachnoid cyst is a congenital abnormality that may or may not produce symptoms. As such, it is commonly diagnosed as an incidental middle cranial fossa finding in imaging. Yet, when they produce symptoms, it is usually in early childhood. The patient may present with headache or seizure or focal neurological deficit among other signs and symptoms of an increase in intracranial pressure (ICP) that result from mass effect, hemorrhage or cyst rupture (Greenbreg, 2010). Generally speaking, lesions effecting middle cranial fossa and in particular temporal lobe produces changes in auditory, visual and or perception in addition to distort feelings and emotions. Besides, in its extreme it can lead to a syndrome of Kluver and Bucy (Kiernan, 2012; Hoesen, 1995). Hence the anatomical association between psychosis and middle cranial fossa lesions, especially arachnoid cyst, is established, but its causation needs to be elucidated (Blackshaw & Bowen 1987; da Silva et al., 2007; Mironov et al., 2014; Bahk et al., 2002; Das et al., 2017; Vakis et al., 2006; Maner et al., 2014).

Psychosis in relation to middle cranial fossa arachnoid cyst resembles schizophrenia (Cargaleiro et al., 2013). It was also ascribed as obsessive compulsive behaviour, in addition the manifestation of delusions and hallucinations (Biswas et al., 2012). Such behavioural changes were not related to to increase ICP. In an elaboration for the cause and effect of the cyst; it was noted that even if the middle cranial fossa-arachnoid cyst produced an increase in the ICP, antipsychotic drugs are also required, despite ICP reduction (Das et al., 2017; Vakis et al., 2006). Hence, the cyst-psychosis association should not be attributed to increased ICP. Moreover, psychosis disappeared once the cyst was removed in one patient (Baquero et al., 2014). This aligns with cases that were labelled as refractory psychosis without cyst removal (da Silva et al., 2007; Cargaleiro et al., 2013). On the contrary, those who started on antipsychotic drugs showed controlled symptoms and no resolution (Bahk et al., 2002). It is worth noted that psychosis has not been universally cited as one of the cyst's presentations and hence psychosis as a sole presentation is not an indication for surgical intervention highlighting the gap between the established anatomical association and causation.

Here, I report a case of a young female who presented with lithium toxicity after being diagnosed with refractory schizophrenia, despite overlooking an arachnoid cyst in the middle cranial fossa. Due to the paucity of information in the literature on such association here, I discuss the implications from a first medical encounter, from a patient's perspective and from a disposition perspective as well.

2. Case Report

2.1. Patient information

A 27-year-old female diagnosed with refractory schizophrenia was found unconscious at home; she then developed a tonic clonic seizure that lasted for less than

2 minutes in the ambulance. She arrived at the emergency department in the post-ictal state.

One year prior to presentation, her first medical encounter was a suicidal attempt by a corrosive material preceded by agitation and delusion. During her admission for an oropharyngeal and ocular burn, she was diagnosed with schizophrenia based on her delusions, paranoid ideation and hallucination in addition to uncontrolled agitation. Lithium and clozapine were started, and the patient was discharged. During the follow-up, the patient underwent several blood tests for monitoring side effects of those two drugs for which she developed agranulocytosis once.

One day to her current presentation, she developed ataxia and dysmetria triggered by nutritional fasting. The medications were administered by the patient's mother, and the patient had no access to those drugs.

2.2. Clinical findings

Her physical examination after the post-ictal revealed a Glasgow Coma Scale of 15/15, dysmetria to finger-nose-test and truncal ataxia in addition to myoclonus. She also demonstrated hyper-reflexia all over and clonus. There was no weakness, and no sensory deficit was noted.

2.3. Diagnostic assessment

Her complete blood count revealed a normal leukocyte count, and the other basic panel including electrolytes renal and liver profile was all within the normal range. Just upon arrival, she showed metabolic acidosis with high lactate that soon normalized from her recent seizure. The pregnancy test was negative.

2.4. Therapeutic intervention

She was prone to develop seizures from the use of clozapine. However, her constellation of symptoms suggested lithium toxicity given her fasting status and her ataxia. A brain computed tomography (CT) scan was ordered as she had undergone no previous brain CT scans. Her CT brain scan revealed an extra-axial well-defined CSF density-area involving the left middle cranial fossa measuring 1.7 × 2.8 cm, representing an arachnoid cyst without an evidence of increased ICP.

Figure (1) illustrates the CT brain scan images in different axial cuts.



Figure (1): CT brain, three Axial cuts A-C showing an arachnoid cyst of 1.7X 2.8 cm in the middle cranial fossa.

2.5. Follow-up and outcomes

She was treated with fluid and sodium polystyrene sulfonate (kexalyate) for the management of lithium toxicity. The patient offered no surgical intervention for cyst removal due to the dilemma of association between the cyst and her psychosis. She was admitted for a new-onset seizure from a metabolic cause, and for the treatment of lithium toxicity.

3. Discussion

The implication for the lack of established causation appears in two situations. First, an argument is noted against CT brain scans for a young with an acute psychosis. It was reported that structural causes were not common and that those incidental findings that ranged from 17.6 to 37% including arachnoid cysts not related to psychosis (Coentre et al., 2016; Strahl et al., 2010; Khandanpour et al., 2013). This may be explained in part by the congenital nature of the arachnoid cyst, and its asymptomatic phase. However, it is hard to overlook such association without monitoring for the size of the cyst before and after the psychosis manifested. This is difficult and require a baseline imaging showing an arachnoid cyst in a brain imaging done for another reason and without any other explanation for the psychosis in such patient. In addition, the manifestation of auditory hallucination in those patients is another reason that impeded causation. The reason is that it represents a functional-type of psychosis and therefore hint toward a psychiatric origin and not organic. On the whole a functional-type of psychosis when coupled with the juvenile age of presentation; two factors further contributing to the dilemma of cyst producing psychosis. Although this may hold true as a general rule, nonetheless it may also hint toward our lack of knowledge on how the cyst behaves beyond rupture or bleed.

Moreover, the relationship between the arachnoid cyst and seizure was another controversy. There is a possibility that arachnoid cyst produces epilepsy and afterwards causes psychiatric abnormalities secondary to epilepsy (Murthy, 2013). This epileptogenesis attributed to the ICP exerted by the cyst rather than its anatomical location (Koch, 1995). In addition, only a minority of those without an increase in ICP had their seizure foci near the cyst (Arroyo & Santamaria, 1997; Mackle & Wile, 2017). Consequently, such associational controversy would have its repercussion in hampering further investigation toward the cyst-seizure association whether an electroencephalogram (EEG) and or a Magnetic resonance imaging (MRI). It's worth to note that here in our case, the patient did not develop seizure prior to this presentation and hence her psychosis attributed to the cyst itself. Furthermore, the patient's seizure assumed secondary to lithium toxicity provoked by dehydration given her fasting status. She did not develop any seizure after hydration and after the start of lithium toxicity management.

Second, this case reveals that the rarity of some diseases impedes the establishment of well-designed studies

and, hence, hampers the movement from association to causation which in this case would hamper any surgical removal of the cyst.

At the time of this writing, this case, reports the smallest middle cranial fossa-arachnoid cyst that produces symptoms without any signs of increasing ICP. Such finding aligns with previously reported cases that have argued for the anatomical association between arachnoid middle cranial lobe cyst with psychosis rather than from increasing ICP (Das et al., 2017; Vakis et al., 2006). Overall, the quality of the patient's life urges the need to use such association in the decision to operate, and the patient or the guardian should have the choice in such decision. A need for further studies to investigate the psychosocial behaviour of those patients after surgical removal of their cysts.

References

- Arroyo S, Santamaria J. (1997). What is the relationship between arachnoid cysts and seizure foci? *Epilepsia*; 38(10):1098-102.
- Bahk W, Pae C, Chae J, Jun T, Kim K. (2002). A case of brief psychosis associated with an arachnoid cyst. *Psychiatry And Clinical Neurosciences*; 56:203-205.
- Baquero G, Molero P, Pla J, Ortuño F. (2014). A schizophrenia-like psychotic disorder secondary to an arachnoid cyst remitted with neurosurgical treatment of the cyst. *The Open Neuroimaging Journal*; 8:1-4.
- Biswas PS, Sen D, Chaudhary S. (2012). Scientific Letter: Middle cranial fossa arachnoid cyst presenting with obsessive compulsive behaviour associated with psychosis – two cases. *African Journal of Psychiatry*; 15:59-60.
- Blackshaw S, Bowen R. (1987). A case of atypical psychosis associated with alexithymia and a left fronto-temporal lesion: possible correlations. *Canadian Journal of Psychiatry. Revue Canadienne De Psychiatrie*; 32:688-692.
- Cargaleiro I, Oliveira-Maia A, Duarte P, Barahona-Corrêa B. (2013). Arachnoid cyst and psychosis: a case report. *European Psychiatry*; 28:1.
- Coentre R, Silva-Dos-Santos A, Talina M. (2016). Retrospective study on structural neuroimaging in first-episode psychosis. *Peerj*; 4:e2069.
- da Silva J, Alves A, Talina M, Carreiro S, Guimarães J, Xavier M. (2007). Arachnoid cyst in a patient with psychosis: Case report. *Annals of General Psychiatry*; 6:16.
- Das S, Kartha A, Thoppil Purushothaman S, Rajan V. (2017) Arachnoid Cyst and Psychosis: The Troublemaker or Innocent Bystander. *Indian Journal of Psychological Medicine*; 39:194-195.
- Greenbreg M. (2010). Developmental Anomalies-Arachnoid Cyst In Greenbreg M. 7th Eds: *Handbook Of Neurosurgery*. New York: Thieme press.
- Hoesen G, (1995). Anatomy of the medial temporal lobe. *Magnetic Resonance Imaging*, 13: 1047-1055
- Khandanpour N, Hoggard N, Connolly D. (2013). The role of MRI and CT of the brain in first episodes of psychosis. *Clinical Radiology*; 68:245-250.
- Kiernan, J. A. (2012). Anatomy of the Temporal Lobe. *Epilepsy Research and Treatment*, 176157. <http://doi.org/10.1155/2012/176157>
- Koch CA, Voth D, Kraemer G, Schwarz M. (1995). Arachnoid cysts: does surgery improve epileptic seizures and headaches? *Neurosurg Rev*. 18:173-81.
- Mackle, T., & Wile, D. (2017). Arachnoid Cysts And Adult Onset Epilepsy. *CMAJ : Canadian Medical Association Journal*, 189(7), E280. <http://doi.org/10.1503/cmaj.160423>
- Maner F, Babalioglu M, Cetinkaya O, Ipekcioglu D, Ergen N, Yesil R, Üstün I, Bas O, Ulukaya S, Yerebakan M and Alici S. (2014). The Coexistence of Arachnoid Cyst with First Episode Psychosis: Four Cases. *J Neurol Disord*, 2:6
- Mironov A, John S, Auerbach J, Jamaledine G. (2014). Acute onset of psychosis in a patient with a left temporal lobe arachnoid cyst. *Case Reports In Medicine*; 2014:204025.
- Murthy J. (2013). Intracranial arachnoid cysts: Epileptic seizures. *Neurology India*; 61:343-4

Strahl B, Cheung Y, Stuckey S. (2010). Diagnostic yield of computed tomography of the brain in first episode psychosis. *Journal of Medical Imaging and Radiation Oncology*; 54:431-434.

Vakis A, Koutentakis D, Karabetsos D, Kalostos G. (2006). Psychosis-like syndrome associated with intermittent intracranial hypertension caused by a large arachnoid cyst of the left temporal lobe. *British Journal of Neurosurgery*; 20:156-159.