CASE REPORT

Right-sided intracranial epidermoid cyst with psychosis: a case report

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ABSTRACT

Background: Epidermoid tumours are rare intracranial benign cystic lesions accounting for 0.2–1.8% of all intracranial tumours.

Case description: We present a case of intracranial epidermoid cyst involving right temporal lobe with psychosis showing resolution of symptoms after resection of tumour.

Discussion: Literature regarding this is sparse. We discuss the rarity of a right sided epidermoid tumour in temporal lobe with psychotic manifestations.

Conclusion: We conclude that organic etiology should always be kept in mind while treating psychosis.

Key words: Intracranial tumours; Epidermoid cysts; Psychosis

INTRODUCTION:

Epidermoid tumours are rare, benign, congenital cysts accounting for 0.2–1.8% of all intracranial tumours. [1] They are believed to arise from sequestered epithelial remnants during neural tube development. Most common location is cerebello-pontine angle [CPA] in the brain representing 4.6-6.3% of all lesions in this site. They have a tendency to envelop neurovascular structures and extend to other regions through foramina. Symptoms arise due to adherence of capsule to neurovascular structures leading to irritation and ischemia. [2] CPA epidermoids usually present in 4th decade with hearing loss, guillotises, dizziness, diplopia, facial paresis and numbness. VIII and V cranial nerves are most commonly affected followed by VII. On CT scan of head they can be hypo dense, hyper dense or iso dense to brain but on T2 weighted MRI imaging they are hyper-intense to brain and cerebro-spinal fluid. [3] Surgical removal is the only treatment in symptomatic patients. Aseptic meningitis, cranial nerve deficits, worsening of cerebellar function and hydrocephalus are the usual post-operative complications. [2, 3] To the best of the authors’ knowledge, there are only two reported cases of intracranial epidermoid tumours manifesting psychosis making this present case extremely rare.

Case history:

A 32 year old male patient was referred by a Magistrate with the complaint that he was wandering on roads. He was admitted in our hospital involuntarily under section 23 of Indian Mental Health Act, 1987. A history of suspiciousness on wife, abusing and assaulting family members without provocation, wandering aimlessly on roads in a disorganised manner and impaired social and occupational functioning of 1 month duration was given by his mother who was the informant. Illness was episodic in course with current presentation being the second episode.

Perusal of past records showed a previous admission 3 years back when he had a history of suspiciousness against mother-in-law, violent and disorganised behavior, impaired biological, social and occupational functioning of 3 months duration with no obvious precipitating factor. On mental status examination(MSE), he had delusions of persecution and second person auditory hallucinations. His general examination and higher cognitive functions were within normal limits. Routine investigations done at that time were within normal range and brain imaging studies were not done. He was diagnosed as having psychosis NOS as per ICD 10[4] and prescribed tablet olanzapine 20mg/day. Patient showed complete remission of symptoms after a period of 1 month with this dosage and was discharged. He had been compliant with this medication and was asymptomatic for a period of 3 years. He reached premorbid level of functioning and pursued a career of cloth merchant.

1 month before the onset of current episode patient developed a seizure with loss of consciousness for the first time, which led the patient to stop anti-psychotic medication. Within a week of stopping medication patient developed current symptoms with no other precipitating factor, and for this reason he was again admitted. During the present admission, he was started on tablet risperidone 4mg/day. He developed another seizure which was generalised tonic clonic type without any preceding aura 10 days after admission. Following this, he was prescribed tablet phenytoin sodium
300mg/day and thoroughly investigated. On probing, patient recollected having difficulty hearing in right ear since 3 years which started along with psychotic symptoms of first episode. He was experiencing early morning headache and dizziness since 1 month. He also developed facial asymmetry, swaying to right side while walking and frequent falls to the same side which progressed over a period of 2 months during hospital stay. There was no history of seizures, head injury, any other significant medical or surgical illness or substance abuse in the past. No family history of any psychiatric illness was present. Premorbidly patient was well-adjusted.

His vitals and general examination findings were within normal limits. On neurological examination, he is right-handed, skull and spine were normal. He had right facial palsy with positive Rinnie’s and Weber’s test on right side indicating right VII and VIII nerve deficits, right palatal palsy indicating involvement of IX, X, XI cranial nerves on right side. His other cranial nerves were functioning normally. His motor and sensory system were grossly normal. He had wide-based gait suggestive of cerebellar ataxia with swaying and falls to right side, positive Rhomberg’s sign with eyes open, abnormal rapid alternating movements, past pointing, intentional tremors and dysdiadochokineses on right side indicating right cerebellar involvement. Direct ophthalmoscopic examination showed blurring of cup margins in fundus of both eyes indicating papilledema due to raised intra-cranial tension. Mini mental status examination was normal with 29/30 score after starting treatment. His mental status examination showed delusions of infidelity with grossly intact cognitive functions except for circumscribed memory loss for the wandering behaviour. This memory loss resolved during hospital stay.

Investigations

Complete blood picture, Erythrocyte Sedimentation Rate, Complete Urine Examination, Random Blood Sugar, chest X-ray PA view and Electrocardiogram were within normal limits. CT scan brain plain showed large hypodense lesion measuring 6.0cmX 6.8cm X 8.0cm involving supratentorial and infratentorial right sided cisterns and right temporal lobe suggestive of epidermoid tumour (figure 1, 2).

He was provisionally diagnosed as having organic delusional (schizophrenia-like) disorder (F06.2) as per ICD-10.19 By this time 2 months have lapsed since patient’s admission and his symptoms have remitted. He was referred to Neurosurgery Department, where they diagnosed him to have right cerebello-pontine angle epidermoid tumour with supratentorial extension. He was advised to discontinue antipsychotics prior to surgery. He underwent craniotomy and excision of CP angle tumour and its extention through retro-sigmoid, retro-mastoid, sub-occipital approach. Histopathology of sample confirmed it to be an epidermoid cyst. Post surgery, patient’s recovery was uneventful. Patient continued to be free of psychotic symptoms after surgical resection without any antipsychotic medication till date.

Figure 1: A non-contrast Computed tomography image of brain in a horizontal plane showing large ill-defined lobulated hypo dense mass lesion in CPA cistern compressing right cerebellar hemisphere and mild effacement and destruction of 4th ventricle

Figure 2: A non-contrast Computed tomography image of brain in horizontal plane showing supratentorial extension into right temporal lobe suggestive of epidermoid
DISCUSSION:

Literature is sparse regarding epidermoid tumours manifesting with psychosis. Previously reported case series show that psychosis is associated with temporal lobe tumours and they commonly manifest as schizophrenia-like symptoms [6, 7, 8]. On the contrary, finding brain tumours in chronic schizophrenia patients is very rare [9]. There are many case reports of temporal lobe tumours other than epidermoid tumours like astrocytoma associated with psychotic symptoms [10, 11, 12], but we noticed a definite left preponderance except in one case report, that of a dyssembryoplastic neuroepithelial tumour which showed involvement of right temporal lobe [13]. Psychosis can also be manifested in tumours of cerebellar region due to disruption of cerebellar efferents to mesial dopaminergic areas, locus coeruleus and raphe nuclei; the centres for major neurotransmitters associated with psychosis. A cerebellar lesion could also cause deafferentation of thalamo-limbic circuits leading to behavioral changes [14]. Our search could only find two case reports of epidermoid tumours associated with psychotic symptoms. The first case report was of a simultaneous manifestation of paranoid psychosis, epilepsy and epidermoid tumour in left temporal lobe where in epilepsy got resolved following surgical resection of tumour but psychosis persisted. Thus they could not establish causal relationship between tumour and psychosis [15]. The second report was of an epidermoid tumour displacing left temporal lobe presenting with mood and perceptual abnormalities. Here also the causal relationship could not be established [16].

In our case,

- The epidermoid tumour originated from cerebello-pontine angle and grew to involve right temporal lobe. Both these locations are known to manifest with psychosis.
- There was simultaneous manifestation of deafness and non-specific psychotic features in the first episode, with later development of other neurological deficits and seizures in the second episode all of which are known to occur in CPA angle epidermoid tumour
- Both psychotic and neurological symptoms completely remitted following resection of tumour
- This remission was maintained without any antipsychotic medication from post surgery till date unlike first episode wherein the patient relapsed immediately following discontinuation of antipsychotics.
- There is no family history of psychosis and no precipitating stressor to both episodes.

All this evidence favours a causal relationship between the epidermoid tumour and psychosis in both episodes rather than the tumour being a mere incidental finding. Thus a diagnosis of organic delusional disorder can be entertained in both episodes. But evidence to link first episode with tumour is retrospective and circumstantial. Due to lack of imaging studies during first episode, a definite relationship of tumour and psychosis of first episode can not be established. This brings us to our second diagnosis of psychosis NOS in first episode and organic delusional disorder in second episode. One other interesting finding in our case is the remission of symptoms to anti-psychotic medication. There are precedents in literature that psychosis secondary to tumours can respond to low dose medications symptomatically but definite treatment is only resection [17, 18, 19]. The unique features of our case report are:

- Presence of intracranial epidermoid tumour is rare and its association with psychosis also is a rarity.
- Right sided tumour associated with psychosis is rarely encountered in literature.
- Complete resolution of symptoms following resection of tumour while off medication lends credential to causal relationship.

CONCLUSION:

The above case is an example of neuropsychiatric interface with clinical implications that psychosis can have an organic etiology. Psychiatrists rarely encounter such cases in routine clinical practice and when encountered rarely attribute causal relationship. Our case suggests that cerebello-pontine angle epidermoid tumour can present with psychosis and even right sided (non-dominant) tumours can manifest with psychosis.

Acknowledgements:

We thank Prof V Pramod kumar, Superintendent and HOD, Department of Psychiatry, Institute of Mental Health, Hyderabad, India for his support and encouragement. We thank the department of general surgery, department of pathology, Osmania General Hospital, Hyderabad, India for their contribution in providing treatment details and reports. We also wish to express our gratitude to the department of radiology, MNJ Institute of Oncology, Osmania Medical College, Hyderabad, India for their contribution in providing CT scan report and film.

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