

Psychiatry and organicity interface: A case series

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Summary

An agitated or suicidal patient brought by family or authorities at the A & E can be called a prototypical psychiatric emergency. In these individuals with myriad of psychiatric symptoms, which may have underlying organic etiologies and co-morbidities; final diagnosis should always be made after exclusion of such causes. At presentation identifying underlying medical condition masquerading as a psychiatric disorder can be difficult and challenging in such scenarios. We hereby present three cases where patients were admitted to a tertiary care set up with probable primary psychiatric syndromes; upon investigations were found to have medical-comorbidities; which had pivotal implication on their management.

organicity, psychiatry, co-morbidity, masquerade

Psychiatric emergencies admitted to emergency departments are usually acute onset behavioral disturbances. An organic cause for such a presentation is seen in nearly 9-20% of all such cases [1,2]. The algorithm while arriving at a diagnosis in such an emergency setting, is based on estimating the probability of etiology on the basis of available details and observation. The further management essentially involves looking for the cause in a similar manner. It is therefore essential that all such cases undergo a thorough systemic evaluation for etiology since psychiatric symptoms may have a purely organic etiology [3]. In primary psychiatric presentations too, the management (viz. pharmacotherapy) may be restricted due to organ dysfunctions and other illnesses. We hereby present three cases that presented with varying psychiatric symptoms and after evaluation were found to have a sig-

nificant medical condition. We wish to highlight this concatenation between a psychiatric diagnosis and underlying medical co-morbidity.

CASE A: ACUTE PSYCHOSIS WITH CHRONIC ATRIAL FIBRILLATION

Mrs. SP, 56 years married female was brought by her son to our hospital with an abrupt onset of fearfulness, abnormal behavior in the form of self-harming tendencies, hearing of voices when alone and insomnia. She claimed to have been threatened by someone and that someone had been hiding in her home. She had been found banging her forehead; on being restrained she tried to gag herself and chewed her lower lip injuring herself further. During interview she reported hearing voice of a male and appeared anxious. She was oriented to time, place and person. On examination she had bilateral ecchymosis, a 5 cm hematoma over the frontal region. Due to the biting and gagging she had injuries in and around the oral cavity viz. a 1cm x 0.3cm injury to the lower lip with other multiple bite marks and a vertical in-

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jury (0.7cm) on right lateral aspect of soft palate. Her pulse was irregular and she had a non-specific murmur on cardiac auscultation. She had been operated for Mitral valve replacement 10 years back. No family history or past history of psychiatric disturbances was elicited. Her investigations showed anemia (hemoglobin = 10.2gm/dl), absence of infection (total counts 9000/cu.mm). No electrolyte disturbances were noted and liver and kidney function were normal. Computerized tomography scan of head showed scalp hematoma over frontal region 5cm x 2cm with no injury to the underlying bone. No foreign body was found on otolaryngology evaluation. Her 2D echocardiography showed metallic prosthesis, a dilated left atrium, mitral valve gradient 13/5, chronic atrial fibrillation, with a left ventricular ejection fraction was 45-50%. Her heart rate was 99/min, irregular and electrocardiogram (ECG) was suggestive of chronic atrial fibrillation. She was prescribed Tab. Digoxin at 0.125mg twice a day and warfarin 5mg for the cardiac condition.

In view of her initial presentation a working diagnosis of Brief Psychotic disorder as per Diagnostic and Statistical Manual (DSM 5) [4] was considered. She continued to be agitated in the ward and was noted to be hallucinating; for which pharmacotherapy with anti-psychotics was initially considered [6]. The QT_c in her case was 0.38 which, though normal, an inter rater reliability in QT_c measurement of up to 20msec has been noted even in experts [6]. Evidence has shown that all anti-psychotics are known to cause cardiac conduction effects by increasing the QT_c interval with various risks [7] with only lurasidone and Aripiprazole included as safest while considering cardiac conduction [8,9]. Patient was monitored, managed conservatively with Inj. Lorazepam 2mg P.R.N. She responded to the above medications and was discharged.

CASE B: THYROTOXICOSIS PRESENTING AS MOOD FEATURES WITH STRONG BIPOLAR AFFECTIVE DISORDER PEDIGREE

Mrs. MS, 35 years, married female, with a known family history of bipolar affective disorder was brought with irritability, lability of mood, grandiose delusions and decreased sleep. Patient had a history of mild fever 2 weeks prior to the illness and general examination showed mild swelling over the thyroid. Patient blood indices revealed anemia Hb – 7.9gm/dl. There was no evidence of infection (Total Count – 5900/cu.mm) and peripheral smear for malarial parasite was negative. Considering symptoms and history of similar complaints in mother and elder sister, she was provisionally diagnosed with a manic episode. She was started on Tab. Risperidone 2mg and Tab. Lorazepam 2mg HS. Thyroid function tests were done in view of the fever and thyroid swelling. She had increased free thyroid T₃, free T₄ and Total T₄ (Table 1) that were suggestive of acute thyrotoxicosis. She was started on Tab. Carbimazole 30mg for the same. As seen in Table 1 the improvement correlated with the improvement in her thyroid status. The Brief Psychiatric Rating Scale [10] score decreased and her psychotropic medications were stopped within a week. Patient showed remission in symptoms as her thyroid function started improving. She was discharged after observation for a week and Carbimazole 30mg was continued. She was monitored on outpatient basis. On euthyroid state patient did not display any re-emergence of psychotic symptoms or mood features and has maintained well without any psychotropic medications. She has not relapsed over the last 6 months. Patient is on regular follow up for her thyroid dysfunction.

Table 1. Change in Thyroid Profile on Carbimazole 30mg

No	Normal Levels	At Admission		1 week		2 weeks	
		Thyroid	BPRS	Thyroid	BPRS	Thyroid	BPRS
1.		Thyroid	BPRS	Thyroid	BPRS	Thyroid	BPRS
2.	T3 (0.58 – 1.59ng/mL)	5.15	48	2.50	26	1.39	11
3.	T4 (4.87 – 11.72 ug/dl)	22.08		16.85		11.99	
4.	ft3 (1.71 – 3.71)	>30		7.34		4.04	
5.	ft4 (0.70 – 1.48)	4.65		2.42		1.69	
6.	TSH (0.35 – 4.95uIU)	0.0035		0.00025		0.0025	

In our patient the initial presentation and family history supported the presence of a primary psychiatric diagnosis as a manic episode, since the substantial role of genes in the susceptibility to mood disorders has long been supported by family, twin, and adoption studies [5]. The deranged thyroid profile made the picture clear. While we considered that the coexistence of psychosis and thyrotoxicosis could have been due to the above, we diagnosed our patient with psychosis due to thyroiditis for following reasons. First, the appearance of psychiatric symptoms was chronologically related to the onset of thyrotoxicosis and the resolution of the psychosis was temporally related to the improving thyroiditis. This suggested that the psychotic symptoms were directly related to the thyrotoxicosis. Second, the clinical symptoms of a sore throat and anterior neck tenderness, the laboratory results of an increased Free T₃ and Free T₄ levels with low Thyroid stimulating hormone (TSH), clearly indicated a thyroid dysfunction. Third, anti-psychotics were discontinued soon enough and confounding was therefore avoided. Patient was only treated with Lorazepam as per need basis.

CASE C: ALTERED SENSORIUM IN AN UNATTENDED MALE WITH INCONSISTENT ALCOHOL USE

Mr. FV, 60 year old unattended male was admitted to our hospital as a case of probable alcohol withdrawal as an involuntary admission. He gave inconsistent history of alcohol use and was bruised and disheveled. He had been initially assessed by the departments of medicine, neurology and neurosurgery in view of his confused behavior. He was referred to us in view of his alcohol use, mild tremors and overall presentation with provisional diagnosis of delirium tremens. All opined it to be a case of alcohol withdrawal delirium.

Patient was poorly nourished and anemic (hemoglobin 9 g/dl) while liver function, renal function and hematology were normal. He was unable to walk without support. He was given prophylactic injection thiamine 100mg intramuscular once a day. During his stay patient did not have features of alcohol withdrawal, the CIWA – Ar [12] score was 4 and thus benzodiazepines

were not administered immediately. On the second day of indoor stay he developed restricted neck movements and fluctuating levels of consciousness. For this he was referred to the department of neurosurgery; considering trauma as a probable cause given his presentation. His computer tomography scan of brain showed hydrocephalus, and an immediate ventriculoperitoneal shunt was done by the neurosurgery department. Patient's relatives were traced with help of authorities. The last known use of alcohol had been more than 3 weeks making alcohol withdrawal unlikely. As per details available from them, he had irregular use of alcohol; no dependence pattern was elicited.

In this case, considering the representative male patient with alcohol use and confused state, the patient was labelled as delirium tremens. While delirium tremens is a known complication in alcohol dependent patients, an altered sensorium in elderly requires adequate individual clinical experience, a high degree of suspicion, and repeated cognitive testing of at-risk individuals to arrive at a diagnosis [13]. Similarly even with history of alcohol use in a case with altered sensorium doesn't preclude it to be the only aetiological cause, as delirium itself has a large number of possible causes. Delirium due to any cause has an increased morbidity and mortality and a delay in diagnosis may worsen the prognosis [14]. Improving consultation – liaison and sensitization may be the way forward in managing such presentations.

CONCLUSION

Overall in clinical practice, a psychiatric cause assumes a common denominator for all 'behavioural disturbances'. It warrants reasoning that this may not be true in all such scenarios. Psychiatry has a dearth of pathognomonic signs or symptoms and even lesser 'confirmatory' or 'gold standard' laboratory tests to diagnose even the most common disorder like anxiety and depression. We therefore wish to highlight the importance of medical causes and co-morbidities; which in our experience operated as a treatment adjudicator in the first scenario, an etiological cause in the second one and a masquerade in the third. It also reiterates the principle that non-or-

ganic psychiatric syndromes must remain a diagnosis of exclusion in acute psychiatric presentation to the emergency department.

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