Blurred boundaries of the brain-1 (Aide-memoire for patient narrative)

Dutta A[1], Poudel R[2], Thapa L[3], Bhattarai S[4], Shrestha MA[5], Thapa M[6]

1. MD Psychiatry, Nepal Police Hospital/National Neuro Center,
2. DM Neurology Grande International hospital/National Neuro Center,
3. DM Neurology, National Neuro Center,
4. MD Neurology National Neuro Center,
5. MD Internal Medicine, National Neuro Center,
6. MBBS, National Neuro Center

Abstract

Background:

This is a case series of clinical cases presenting to our Neuropsychiatry Center, who were either referred by physicians or approached to seek treatment on their own. They were receiving treatment since few years. No improvement in symptom were primary reason for referral and consultation with our team. Re-evaluation in realms of Neurology and Psychiatry depicted the missed link in diagnostic and therapeutic approaches exercised previously. The intent to reflect upon novel findings is not to undermine the effort applied in clinical evaluation by prior physicians, but to reflect few observations which may have been inadvertently missed during routine assessment. This serves as as an aide-memoire to history taking and suggestions to remain well informed and updated in our approach to service seekers.

Abstract:

Symptom overlap and comorbidities are commonly encountered in clinical practice of Psychiatry or Neurology. Many neurologic disorders have psychiatric symptoms (e.g., depression in patients following a stroke or with multiple sclerosis or Parkinson's disease), and many psychiatric disorders have been associated with neurologic symptoms (e.g., movement disorders in schizophrenia).[1] This overlap occurs mostly because brain is the source common for both neurological and psychiatric illness and on multiple occasion it becomes difficult to delineate the boundary, leading to diagnostic dilemmas, followed by inappropriate management strategies. Patients and family members suffer for long periods of time in trying to understand the disease phenomenon and prognosis.

In this case series, we present 3 cases of clinical interest across different framework of clinical presentation and assessment for e.g, neurological illness being managed as psychiatric illness or scenarios where both neurological and psychiatric illness coexisted but only one aspect was addressed. Re-assessment and work up by a team of neurologists and psychiatrist lead to clarification of the boundaries (at least in these cases who underwent prolonged sufferings owing to lack in clarity of pathology) with precise diagnosis generating improved outcomes in functionality and satisfaction.

The diagnosis of neuropsychiatric disorders is performed by psychiatrists through diagnostic interviews, and categorization of patients based on diagnostic and classification manual (ICD-10, DSM-5) which are syndrome/symptom based. Although this has been a standard practice, often error may occur in diagnosing illnesses falling under the wider realm of neurology and psychiatry. Some presenting symptoms may be assumed being part of neurological illness or of functional origin, or mixed states and many may be overlooked during routine clinical assessment.

Keywords:

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***Corresponding Author**

Dr. Ashish Dutta

MD Psychiatry,
Nepal Police Hospital/National Neuro Center
Email: dutta.doc@gmail.com

INTRODUCTION

Neuroscience describes the brain mechanisms that gather information from the external and internal world, analyze the information, and execute the best response. Neuropsychiatry represents a field of medicine at the crossroads of neurology and psychiatry, and deals with the interface of behavioral phenomenon driven by brain dysfunction.[2] Our brain is responsible for all cognitive processes, emotions, and behaviors—that is, everything we think, feel and do[1].

Psychiatric disorders are the result of problems with these mechanisms. The first major classification of mental illness was based on the distinction between disorders arising from disease of the brain and those with no such obvious basis, i.e. organic versus functional states. These terms are still used, but as knowledge of the neurobiological processes associated with psychiatric disorders has increased, their original meaning has been lost.[3] After 1950s, recognition of the effectiveness of medications in treating mental illness and the psychological effects of illicit drugs resumed a biologic perspective of mental illness. This concept had already been introduced by the use of electroconvulsive therapy (ECT) and James Papez's description of the limbic circuit in the 1930s. This biologic view has been reinforced further by the development of brain imaging techniques that have helped reveal how the brain performs in normal and abnormal conditions. Brain is the center for mental illnesses as well as neurological disorders. With ongoing research and advances in neurosciences a better understanding of brain structure and function prevails.

Although difficult to demarcate the line between neurological and psychiatric disorders with respect to origin in the brain, a thoughtful approach and engagement in active listening to the history and presenting symptoms with systematic clinical examination will help in a justified diagnostic approach and focused treatment to patients presenting with symptoms in neuropsychiatry domain.

CASE 1: DRIVER DRIVEN DIZZY

37 years old male, 10th grade education, from mid-socioeconomic status was under treatment for panic disorder for 3 years. He was compliant to Escitalopram 20 mg and propranolol 40 mg, but no improvement acknowledged. Re-evaluation revealed a right ear discomfort and dizziness and some gait imbalance since the same time he experienced anxiety, Patient also recalled that his panic attacks occurred in the context of his dizziness and ear discomfort. As he is a cab driver, he was experiencing difficulty in driving to an extent that his professional life was jeopardized. Fukuda test (Simple Bedside test for evaluation of vestibular dysfunction) revealed positive result for Right Ear Vestibular Neuritis. Balance exercises along with short term use of Betahistine ensued significant improvement in dizziness, achievement of a good sense of control and balance, leading to significant improvement in anxiety symptoms and overall quality of life.

Persistent dizziness causing occupational dysfunction with no relief of symptoms despite use of SSRI for anxiety in an adequate dose over a substantial period of time, prompted a re-evaluation in this case.

Patients suffering from anxiety or panic go through various physical symptoms but all cannot be attributed to "anxiety experience". Dizziness is often experienced which is disabling. Its assessment is usually missed usually while dealing with patients suffering from anxiety. Among differential diagnosis for dizziness, Of special mention here, we emphasize on being aware of two conditions, Vestibular neuritis, Persistent Postural Perceptual Dizziness (Different from Benign Paroxysmal Positional Vertigo).

Other physical symptoms of anxiety should also be thoroughly assessed to verify whether they are a part of anxiety experience or an independent occurrence relating to specific organ pathology (Co- morbidity).

CASE 2 : DEREALIZED DOCTOR

47 years male, medical doctor by profession presented with chief complaints of "derealization". He elaborated derealization as things "not looking real" in the environment and he felt "detached" on and off since approximately 2 years after a road traffic accident. The accident was not associated with any major injury (external or internal) and no loss of consciousness or vomiting. Radiological Investigations were normal. Attending physician's note revealed patient "might" have suffered concussion with diffuse axonal injury (although there was no evidence for the same). He attributed this feeling of derealization to the accident and "organic" brain lesion. He expressed that he had experienced panic attacks in the past and was anxious by personality. He became obsessed with what the attending physician had told him about developing DAI (Diffuse Axonal Injury) in the brain and grew more anxious thereafter and experienced varieties of symptoms some of which he claimed to be "like" diplopia and visual hallucinations. There was no history of substance use.

Serial investigations over long period of time by specialists did not reveal any CNS pathology and his theme of obsession "why" he was having derealization remained unanswered and exhausted him of financial resources and time in getting evaluated, all in vain.

During this pursuit an MRI head with MRS(Magnetic Resonance Spectroscopy) was done. MRI head was normal. MRS revealed normal metabolite concentration of NAA (N-acetyl aspartate), creatinine and choline but raised

lipid/lactate peak in both grey and white matter, not correlated clinically with his symptoms.

A trial with amitriptyline 25 mg was taken but required discontinuation due to excessive drowsiness. A second trial with Venlafaxine upto 75 mg also failed owing to discomfort and inner sense of restlessness experienced. Finally, Fluoxetine monotherapy at 40 mg resulted in a significant and sustained improvement after 4 weeks of treatment, helping us conclude that his "derealization" was actually a subjective experience of his anxiety disorder which was further enhanced by a component of obsession related to the experience. He is maintaining well now.

Depersonalization and derealization are subjective experiences of misinterpretation owing to its etiology. The actual subjective experience vs the actual meaning can lead to substantial confusion among treating doctors leading to long term treatment trials and wastage of time/money and resources.

This case in particular gave rise to complexities in extricating the dilemma whether the derealization was due to organic phenomenon existing in the brain (In the context of RTA) or was it a part of experience of anxiety and panic attacks. Noticeable features in this case were, undue emphasis on technical words to describe symptoms, self-diagnosis, not remaining compliant to one regimen of treatment and no structural abnormality rooted in the Brain.

CASE 3: TORMENTED BY THE TEMPORAL LOBE

72 years old male, farmer, diagnosed as a case of Psychoses NOS (Not Otherwise Specified) was under treatment with olanzapine 25 mg, for 3 years. Although compliant to medication no improvement was appreciated in behavioral abnormality. Patient acknowledged and emphasized that he was mostly not aware of such changes in him and even when aware occasionally, could not exercise control and was not doing it "knowingly". Historical review disclosed episodic nature of such behavioral changes, lasting 20 minutes to 45 minutes, following which he would be in his premorbid state. There was no history of substance use. The episodes were characterized by acute onset of behavioral changes with verbal abusiveness without pretext, physical assault to family members when confronted, disruption, excessive use of foul words. Informant (Son) reported that whatever changes the patient exhibited during such spells, were not characteristic feature of his father. The patient did not have any depressive episodes in the past, nor were any schniederian first rank symptoms elicited. He apparently had a well-adjusted premorbid personality.

An MRI of head revealed right temporal lobe atrophy with gross atrophy of right hippocampus with prominence of right temporal Horn. Supported by this finding a diagnosis of Focal seizure with impaired awareness (Temporal lobe Semiology) predominant features being behavioral abnormality and aggression was made and treatment started with oxcarbazepine 150 mg BD in a gradually incremental dose up to 450mg BD, resulting in complete remission of symptoms within a month. The behavioral abnormalities turned out to be ictal phase of Temporal Lobe origin epilepsy and not a Psychoses as an independent entity. The patient is normal now.

Key features which lead us to reassess the diagnosis of Psychoses and consider epilepsy were episodic nature of behavioral abnormality that came about for a well- defined period of time, lack of awareness during the episodes, complete inter-episodic remission, stereotypical presentation, with symptoms not occurring on a pretext of known psychiatric illness.

DISCUSSION

All the cases presented in this case series had one common issue – Missed primary diagnosis! Evaluation of the cases as a team (Psychiatry and Neurology), with emphasis on the primary complaint, the onset, the duration, comorbid factors and attention to gradual unfolding of clinical symptoms over time helped arrive to a conclusion about the primary etiological domain of the illness. A treatment approach based on addressing the root cause of illness brought about near complete remission of symptoms among the patients.

Dizziness, unresponsive episodes, movement related disorders, headache are common presenting symptoms in both, neurology and psychiatry. Each domain can present with similar symptoms, sometimes one preceded by the other, sometimes coexisting, and at times mistakenly indicated in one domain, while the real problem lay in another domain. While it is understood that clearly demarcating what is functional and what is neurological in origin is difficult in many cases, it is worthwhile to approach a patient's symptoms methodologically with a thoughtful consideration and following the basic doctrine of history taking, physical examination. As it is evident with above mentioned cases,

clues to functional origin or neurological origin were derived from history and physical examination and no sophisticated investigative procedures were required to straighten out the underlying primary pathology.

Commonly it has been speculated that there are shortcomings of the current diagnostic system in psychiatry which are based on syndrome/symptoms, that appear to be an impediment to correctly diagnose whether a symptom or a set of symptoms is of mental illness(functional) or neurological illness.

The First case "Driver Driven Dizzy" represents a common error made during psychiatric assessment, overlooking certain symptoms which are assumed to be a part of anxiety disorder. Dizziness is one of the most frequent complaints encountered in the medical practice affecting 15-20 % of adults yearly, and it can be a challenging symptom to assess.[5] Most patients use dizziness as a nonspecific term, and thus suffer prejudice from the physician's end and can be disregarded frequently.[6]

The term "dizziness" may represent various feelings including vertigo, lightheadedness, pre-syncope, unsteadiness, and just not feeling well. The traditional approach to dizziness as in vertigo is vestibular, presyncope is cardiovascular, disequilibrium is neurologic, and nonspecific dizziness is psychiatric or metabolic may be misleading.[7] A recently proposed approach to dizziness/vertigo begins with classifying those as Acute prolonged spontaneous dizziness/vertigo, Recurrent spontaneous dizziness/vertigo, Recurrent positional vertigo, or Chronic persistent dizziness and imbalance. Approximately 40 percent of dizzy patients have peripheral vestibular dysfunction; 10 percent have a central brainstem vestibular lesion; 15 percent have a psychiatric disorder; and 25 percent have other problems, such as presyncope and disequilibrium. The diagnosis remains uncertain in approximately 10 percent. [8]

The patient mentioned in this case falls under the category of Acute Spontaneous Dizziness/Vertigo due to Vestibular Neuritis.

The Second case "Derealized Doctor" represents a case where a precipitating factor (RTA) is present, and may give rise to concussion or DAI, and henceforth explain derealization/depersonalization, however it is always not necessary that the cause-and-effect relationship exists. When no structural abnormalities are noted, and course of symptoms remain prolonged and fluctuating, psychiatric co morbidities should be considered and treated accordingly to relieve anxiety and misinterpretation of symptoms. Among medical professionals who present as a patient, symptom elaboration/explanation becomes more complex for physicians/psychiatrist to fathom.

Also it is an important reminder for us that as clinicians we need to be more cautious in selecting the words which we choose for explaining disease phenomenon to patients. What we speak has got a profound and long-lasting impact on a client's psyche. This patient was told that he "may" have developed concussion and diffuse axonal injury. In the pretext of his anxiety, this statement had such a long-lasting impact that he remained obsessed with organic origin of his derealization and in order to prove his point he unwaveringly kept persuading all the future clinician's he came across that he had derealization. Those clinicians who are not well versed with the real meaning of derealization appeared hesitant to deal with his symptoms and made referrals, which further made the patient feel that he actually was suffering from a complex organic phenomenon, thus reinforcing his own belief.

What he was actually experiencing was not a detachment from environment (During current experiences of derealization) but a sense of discomfort, worries and symptoms of autonomic hyperactivity in his body which he labeled as derealization without actually understanding the meaning and context of usage of the word.

Derealization refers to feelings of unreality or of being detached from one's environment. The variety of conditions associated with depersonalization complicates the differential diagnosis of derealization disorder. Depersonalization/Derealization can result from a medical condition or neurologic condition, intoxication or withdrawal from illicit drugs, as a side effect of medications, or can be associated with panic attacks, phobias, PTSD, or acute stress disorder, major depression, schizophrenia, illness anxiety disorder, or another dissociative disorder[9].

A thorough medical and neurologic evaluation is essential, including standard laboratory studies, an EEG, and any indicated drug screens. A range of neurologic conditions, including seizure disorders, brain tumors, metabolic abnormalities, migraine, vertigo, and Ménière disease, have been reported as causes[9]. Although the patient may have suffered concussion injury in the past (2 years prior to current assessment), current presentation of symptoms did not correlate with organic changes in the brain and is in

favor of psychogenic experience.This case represents an outline where we come across patients from medical background who engage in relating certain symptoms in great details and self-diagnosis to an extent that the treating clinician tend to get biased and end up making error on more ominous side of pathophysiology.

The Third case "Tormented by the Temporal Lobe" is a good reflection of how inadequate history taking and symptomatic treatment approach of cases presenting with behavioral changes can be falsely diagnosed as a psychiatric illness and managed erroneously.

Approximately 60 % of all forms of epilepsy are focal in origin, with majority originating in the temporal lobe[10]. Mesial temporal lobe epilepsy is the most common form of epilepsy and is most commonly due to a neurodegenerative process known as hippocampal sclerosis found in the majority of patients diagnosed with this condition, upon histological examination[11]. The seizures can either be focal aware seizures, focal seizure with impaired awareness, and there can also be seizure activity which originates in the temporal lobe but extends to involve both cerebral hemispheres commonly manifesting as focal to bilateral tonic-clonic seizures[12]. Aggressive and violent behaviors have also been associated with epilepsy, especially temporal or frontal lobe seizures[13]. The diagnosis of seizures should always be considered in cases of episodic stereotyped behavior[14]. In this patient we concluded that the behavioral changes comprised of both ictal and post ictal phases owing to a prolonged duration of symptoms. Aggression and violent behavior with disruptiveness in an episodic and stereotyped way was the manifestation of temporal lobe epilepsy (Probably with frontal lobe propagation?). Ictal and post ictal aggression are often associated with confusion and psychosis[15] . The real prevalence of aggressive behavior in epilepsy remains controversial[16].

CONCLUSIONS

With advances in brain research and availability of advanced investigations, the understanding about biological basis of various mental illnesses and behavioral abnormalities has improved. The etiopathological factors of psychiatric illnesses are no more limited to the conventional characterization of being "functional".

Although in resource poor countries like ours, the luxury of brain research and access to high end equipment is far from real, we are not exempt from providing a justified and evidence-based approach based on what is available.

The most important aspect of patient approach is history taking and clinical examination. A systematic approach, focus on onset of symptoms, their evolution in time, information from collateral sources, the psychodynamic formulation, drug history, family history, personality assessment etc. are the cornerstone of psychiatric evaluation. Psychiatrists are required to be well informed with various symptom profile of physical illness presenting with psychiatric manifestations.

Interdisciplinary consultations are cardinal, especially in Neurology and Psychiatry. Needless to emphasize, if we have doubts, we should make it a point to consult professionals in different specialties. It is not humanly possible to have knowledge about everything. Neurologists and psychiatrists working together on cases provide fruitful results. Both psychiatrists and neurologists gain much by sharing and exchanging their complementary skills[17].

It is necessary to stress that adequate time be spent in case assessment and conceptualization, the basics of approach to a patient not be overlooked; When no improvement is witnessed the diagnosis be reviewed instead of add on treatments; Due considerations be made for possibilities of mixed states (co-existence of both Psychiatric and Neurological illness).

Presence of such factors complicate the clinical presentation among many neurological and psychiatric illnesses further blurring the boundaries of clinical assessment. This ultimately leads to needless suffering among patients and their attending family members, forming a complicated loop, where both clinicians and clients get entangled. These occurrences are common in our precis and result in economic and psychological burden among service seekers which adversely affects the outcome of a disease process.

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