Atypical Psychiatric Symptoms Associated with Left Temporal Lesion: Two Cases

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ÖZET:
Sol temporal bölge lezyonu ile ilişkili atipik psikiyatrik semptomlara sahip iki olgu sunumu


Anahtar sözcükler: Duygudurum, beyin lezyonu, temporal lob, hasarlanma, antiepileptik

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ABSTRACT:
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Brain lesion is a major risk factor for the development of psychiatric symptoms. There is an association between lesion type, location, and psychiatric symptoms. Common psychiatric comorbidities of brain lesions include affective, cognitive, and behavioral disorders; therefore, organic causes should be carefully investigated by clinicians and treatment regimens planned by considering organic causes. In this report, two patients with left temporal lesions, who developed atypical psychiatric symptoms, are described and the distinctive features of their diagnoses are summarized.

Key words: Mood, brain lesion, temporal lobe, injury, antiepileptic

She had mood-incongruent delusions of a persecutory type with euphoria. Her immediate and recent memory were impaired. Neurological examination showed no abnormal findings such as nystagmus, speech disturbance, ataxia, motor and sensory disturbances, or involuntary movements. The rest of her general physical examination was within normal limits. In her past medical history, she had sustained an unclear head injury which had been accompanied by loss of consciousness two years ago. Before the trauma, she had no significant medical or psychiatric problems. When the patient was evaluated by an electroencephalography, focal wave complexes which were localized in the left temporal area and slight neuronal hyperexcitability were noted. The brain MRI demonstrated an area of malacia (3X2.5 cm) in the anterior part of the left temporal lobe (Figure 1).

Clinical Progression: During admission to the psychiatry clinic, the patient was disoriented, with inappropriate affect and psychomotor agitation. Rapid and abrupt changes in emotional tone were observed. A mixed and atypical symptomatology was noted. All psychiatric symptoms and findings continued, although her disorientation improved within a day. It was thought that this clinical presentation was not due to delirium. It was proposed that these symptoms were likely related to an underlying neuropathological condition after consideration of the EEG and MRI findings. Carbamazepine 800 mg. and clonazepam 1.5 mg. per day were prescribed for the patient. Over the next 15 days psychomotor agitation and mood lability gradually decreased.

**CASE 2**

The patient is a 32-year old right handed woman; she is married and has 4 children. She had complaints of markedly diminished pleasure, loss of energy, feelings of worthlessness, and thoughts of death. The complaints had gradually increased and she had attempted suicide by jumping. On psychiatric examination, she was pessimistic during the psychiatric interview. She expressed suicidal ideation. Her mood was irritable and depressed. She had auditory hallucinations and nihilistic delusions. The neurological examination showed no abnormal findings; however, her electroencephalogram showed focal spike-wave discharges which localized in the left temporal area. Her general physical examination was within normal limits, but the brain MRI demonstrated an area of malacia (5X2 cm size) which was localized in the left temporal lobe (Figure 2). She had a history of head trauma. She had fainted and lost consciousness one year ago. She had been treated for cerebral hemorrhage. Post-traumatic mania had developed after the trauma and this episode ended four years ago.
months later without treatment. Before the trauma, she had no significant medical or psychiatric problems. There was no family history of neurological and psychological abnormalities.

**Clinical Progression:** Electro-convulsive therapy (ECT) was given for her suicidal thoughts and depressed mood. Agitation occurred after each ECT application and her agitation did not respond to haloperidol injections. Although her depressed mood and suicidal thoughts improved, her agitation increased and emotional lability began. Both depressed and manic reactions were observed during the day after ECT applications.

Carbamazepine 600 mg., amitriptyline 50 mg., and clonazepam 1.5 mg. per day were started because of continued symptoms and the ECT-induced manic reaction. Mood lability, anxiety, and agitation improved with this medical regimen.

**DISCUSSION**

Our results suggest that brain lesions can cause vulnerability to psychiatric disorders without neurological symptoms. Malacia is a common sequela of parenchymal injury. Lesions occur in brain injuries, both after an immediate insult and at a later time, due to the biomolecular and physiological changes (5,6). Psychiatric disorders are a major cause of disability after traumatic brain injury (7). The rate of axis I disorders in patients with traumatic brain injury ranges 14-77% for major depression and 2-17% for bipolar disorder (8-10). Koponen and colleagues (2002) have also showed rates as high as 48.3% for any psychiatric disorder starting after the brain injury, with major depression being the most common diagnosis (26.7%) (11). Neither mania nor depression are rare complications of brain lesions, but case reports of depressive and manic episodes related to brain injury are rare. In case 2; the patient had a manic episode after brain trauma and post-traumatic mania had been diagnosed. After 1 year from the first episode, the depressive episode occurred with psychotic features. The patient’s clinical condition did not improve with ECT and antipsychotic treatment, but symptoms gradually decreased with antiepileptic treatment. Major depression is a frequent complication of brain injury and may present with many different features. Executive dysfunction, negative affect, and prominent anxiety symptoms are common (12). A manic episode caused by a brain lesion is characterized by irritable mood rather than euphoria and combativeness and the symptoms generally differ from the symptoms which occur in the absence of a brain lesion (13).

It is important to recognize the association between lesion location and psychopathology. Left and right brain hemisphere lesions may cause different psychiatric symptoms. Studies have shown that patients with left hemisphere lesions have higher depression rates than those with right hemisphere lesions (14, 15). A study has indicated an important role of left temporal lobe pathologies in mediating or inducing a complex association.
of mood and cognition disorders. Robinson has described this relationship as “depression following left hemispheric brain injury may not be a nonspecific neurological or psychological response, but rather may be a symptom of injury to specific pathways, such as the catecholamine-containing ones, as they pass through the frontal cortex” (16). Right hemisphere lesions may produce different neurochemical and metabolic brain changes that may underlie the production of either a bipolar disorder or a unipolar mania (17). Right hemisphere lesions seem to be associated with secondary mania, while left hemisphere lesions are usually associated with depression; however, it should not be forgotten that psychiatric symptoms caused by brain lesions can be complex and atypical (18-20).

After traumatic brain injury, prominent impulsivity, affective instability, and disinhibition are seen frequently (21). Neurobehavioral consequences of a brain injury, such as mood swings, apathy, irritability, aggression, poor concentration and memory, and difficulty in planning have been reported extensively in the past, but the type of psychiatric syndromes have rarely been studied. Brain lesions resulting from brain injuries sometimes do not allow the diagnosis of a specific psychiatric syndrome, such as in case 1. Additionally, it is difficult for researchers to assess the pathophysiological aspects of these conditions, which in turn may limit the development of diagnostic criteria; however, the DSM-IV-TR recommendations (for example close temporal relationship, atypical symptomatology, absence of additional explanations) seem to be useful (22). With respect to treatment, Warden et al. noted that “there is insufficient evidence to support any standards or guidelines for the treatment of affective disorders, mania, or psychosis in the brain injury population” (23). Antiepileptic treatment can be prescribed for these symptoms and patients may benefit from this treatment regimen rather than antipsychotic or antidepressant treatments.

References:


