Obsessive-compulsive disorder secondary to a right parietal lobe haemorrhage: a case report

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Abstract

This case report presents a 39-year-old previously healthy man, who presented with typical obsessive-compulsive disorder and neurological symptoms and was found to have a right parietal lobe haemorrhage. His psychiatric and neurological recovery was associated with a complete radiological resolution. This report is consistent with recent imaging studies implicating the parietal lobe in the pathogenesis of obsessive-compulsive disorder and adds haemorrhage to the list of organic cerebral lesions causally associated with this disorder.

Key words: Obsessive-compulsive disorder; intracerebral haemorrhage; cerebral lesions; right parietal lesions.

Introduction

Obsessive-compulsive disorder (OCD) is a common psychiatric condition with prevalence in the general population estimated at 2% (1). It is the fourth most common psychiatric condition, twice as prevalent as panic disorder and schizophrenia (1). Obsessive-compulsive disorder has been causally associated with a number of cerebral lesions including traumatic brain injury, tumours and infarcts (2, 3). Report on vascular tumours or intra-cerebral bleeding causing OCD are rare in literature. In fact, we have found only one recent report of a 24-year-old man, who developed OCD symptoms after a unilateral caudate nucleus haemorrhage due to a cavernoma (4). The majority of these lesions have been localised to the frontal cortex and the basal ganglia (5), which has led to the development of the hypothesis implicating dysregulation of the frontal-striatal-thalamic circuits in the pathogenesis of OCD (5, 6).

Here we report a case of a 39-year-old male whose onset and remission of OCD symptoms corresponded to the appearance and subsequent resolution of intra-cerebral bleeding in the right parietal lobe.

Case report

A 39-year-old previously healthy male underwent psychiatric assessment after developing distressing and intrusive thoughts regarding his own safety, well-being and death as well as an irresistible urge to check the stove, windows and doors in the preceding three months. His obsessive-compulsive symptoms met the DSM criteria. The patient experienced a feeling of internal compulsion. He experienced undesirable thoughts coming to his mind in the form of inflow of thoughts. He regarded those thoughts as inappropriate and nonsensical. The compulsory activities significantly reduced his anxiety. The patient was feeling that his obsessive thoughts and activities were entirely alien to his personality, however they were coming from his interior and not from the outside. All attempts at rejection of the undesirable thoughts or activities made by the patient strongly intensified his fear. No depressive or psychotic symptoms were elicited. His paternal grandfather had schizophrenia. The patient had never sustained a head trauma or loss of consciousness. He did not abuse alcohol or illicit drugs. His pre-morbid character was described as easy-going and socially amiable. He had high-school education and job as a mechanic. He was married and had one child and a stable and content family life.

A review of systems revealed that for the last two months the patient was having frequent headaches associated with nausea and vomiting. His mental state and physical exam were normal; he did not have any meningeal or focal neurological signs.

Brain MRI imaging showed an area of haemorrhage in the right parietal lobe with surrounding oedema. Cerebral angiography was normal.

The patient was diagnosed with OCD according to DSM-IV-R criteria, as well as the Yale-Brown Obsessive Scale, NIMH Obsessive-Compulsive and Maudsley Obsessional Compulsive Inventory. He was treated with sertraline titrated to 200 mg daily as well as with individual psychotherapy. As
the patient refused an invasive neurosurgical intervention, he was treated conservatively with sertraline and psychotherapy.

He responded to the treatment and achieved a complete remission of his OCD symptoms after three months of treatment. Concomitantly, he reported an improvement and finally cessation of headaches, nausea, and vomiting. Subsequent brain imaging showed a resolution of intracerebral blood and oedema. After a three-month treatment, sertraline was tapered down and eventually discontinued. His symptoms have not recurred during an ongoing five-year follow-up.

Discussion

In this case report we describe a patient, who developed OCD symptoms as well as frequent headaches and nausea due to bleeding in the right parietal lobe. Furthermore, resolution of his OCD symptoms corresponded to a remission of the vascular lesion. This report adds to a body of evidence suggesting organic brain lesions including intracerebral haemorrhage can induce obsessive-compulsive symptomatology.

The relatively prompt remission of OCD symptoms in the patient presented in this case is more likely due to an improvement in the cerebral oedema and bleeding rather than serotonergic medication, which usually has a longer onset of action. On the other hand, permanent lesions of neuroanatomy may partially explain the resistance to treatment with psychotropic medication and psychotherapy in about 30% of patients with OCD (5). As shown in the present case, searching for a structural correlate in OCD patients may be reasonable if the disorder is associated with neurological symptoms, occurs in patients with risk factors for intracerebral bleeding or in the context of head trauma.

The location of the intracerebral lesion in this patient was atypical since recent neurobiological models have postulated that dysfunction in the orbito-frontal cortex and the thalamic nuclei underlie the etiology of OCD (6). Lateral parietal regions are involved in attention and visual-spatial processing, which are frequently impaired in OCD patients (9). Thus, dysfunction in the parietal lobe could affect the functions of the other regions which have been implicated in OCD. This report is consistent with recent findings showing a reduction in grey matter in the right parietal associative cortex in 19 OCD patients using Voxel-based morphometry (10). As such, this report supports the hypothesis that the parietal lobe may be involved in the pathophysiology of OCD.

REFERENCES


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