PANIC DISORDER WITH AGORAPHOBIA FOLLOWING TUBERCULOUS MENINGITIS

S P Chand, P Chand

ABSTRACT

Brain damage has been reported to be associated with the onset of panic disorder. We report a case of panic disorder with agoraphobia (PDA) after recovery from tuberculous meningitis (TBM). The CT brain showed cerebral oedema and basal meningeal enhancement. On recovery from the TBM, the patient developed PDA as well as features of depression. She was treated with alprazolam, amitryptline and underwent behaviour therapy over a period of 18 months. At the end of this period, the patient showed improvement but had not reached her pre-morbid level of functioning. The possibilities of brain damage as aetiological factors for PDA are discussed.

Keywords: agoraphobia, panic disorder, meningitis, tuberculosis

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INTRODUCTION

The essential features of panic disorder with agoraphobia (PDA) are recurrent panic attacks associated with the fear of being in places or situations from which escape might be difficult or in which help might not be available in the event of a panic attack[1]. The agoraphobic element is considered to be secondary to the development of panic attacks[2,3]. An association has been reported between brain damage and the onset of panic attacks[4-8]. We report a case of panic disorder with agoraphobia which developed after recovery from tuberculous meningitis (TBM).

CASE REPORT

KA, a 35-year-old woman, presented with a 3-week history of headaches, vomiting and obtundation for 2 days. Examination revealed a febrile, obtunded patient with signs of meningeal irritation. The chest X-ray showed bilateral apical infiltrates; the CT brain showed cerebral oedema and contrast enhancement of the basal meninges. The CSF showed lymphocytic pleocytosis, elevated protein, reduced glucose and acid fast bacilli confirming the diagnosis of TBM. She was treated with rifampicin, INH, ethambutol and pyraznamide. She made a gradual complete recovery and had no residual neurological deficits.

As KA recovered from the illness, she began to experience a generalized feeling of anxiety. She worked as an officer in a bank and resumed work 3 months after recovery. Her job required quick and alert cognitive functioning and interaction with clients. She however found herself experiencing deficits in her memory function. She began to have frequent panic attacks. During the panic attacks, she experienced shortness of breath, feeling of unsteadiness, accelerated heart rate, trembling, a feeling of intense fear as well as a fear of doing something uncontrolled, like screaming. These attacks were triggered sometimes by stressful situations but more often, they were unexpected. After the onset of the panic attacks, the patient developed a persistent fear of having these attacks. She became fearful of going outdoors and avoided going outdoors altogether. KA had to be sent to work and picked up from the door step of her office building.

She had associated features of depression as well, namely feelings of sadness, worthlessness, hopelessness and early morning wakening. She was put on alprazolam 0.25 mg BD initially and later on amitryptline 50 mg at bedtime. She had some relief of insomnia but no improvement in panic attacks. She was further treated utilizing behaviour therapy techniques of Jacobson's Relaxation Procedure (JRP), followed by imaginal systematic desensitization (ISD). The JRP produces an overall reduction of tension and arousal. The technique of ISD involves pairing relaxation with imagined scenes depicting situations that cause anxiety to the patient and are presented in a hierarchy, starting with the least anxiety provoking scene and proceeding finally to the most anxiety provoking scene. In this manner the client gradually learns to experience relaxation rather than anxiety in real-life situations as well.

At a later stage, in-vivo desensitization (IVD) was utilized, where the patient actually confronted the anxiety provoking situations in a hierarchical manner similar to the ISD. By the end of 18 months, she had improved in that the panic attacks had ceased and she had begun to go outdoors on her own. She however continued to experience some feelings of anxiety. Her memory functions had improved as well. She had however, not reached her pre-morbid level of functioning.

DISCUSSION

The present case had a confirmed diagnosis of TBM and developed features of PDA during recovery from it: alprazolam and amitryptline have been reported to be of considerable success in the pharmacotherapy of PDA[9]. However in our patient, they were not of much help in reducing the panic attacks. Behavioural therapy techniques like JRP, ISD and IVD have also been reported to be useful in the treatment of PDA[9]. In the present case, these methods implemented for over 18 months did result in cessation of the panic attacks.

Evidence from several areas of research points to a biological substrate underlying panic disorder[4]. Reiman using positron emission tomography (PET) found evidence of right para hippocampal abnormalities in oxygen and glucose utilisation and regional cerebral blood flow in patients with panic disorder[6]. There have also been reports of patients who developed panic disorder in association with cerebral lesions[5,7,8] involving the right parahippocampal region. A number of residual neurological deficits are known to follow TBM and include cognitive defects, cranial nerve palsies, pyramidal, extrapyramidal signs and spinal

Department of Behavioural Sciences
College of Medicine
Sultan Qaboos University
P O Box 35, Postal Code 123
Al-Khod, Muscat, Sultanate of Oman

S P Chand, M Phil, C Psychol
Senior Clinical Psychologist

Department of Medicine
College of Medicine
Sultan Qaboos University

P Chand, FRCP

Correspondence to: Dr S P Chand

cord lesions[10]. Causal mechanisms include hydrocephalus, arteritis, cranial and spinal arachnoiditis. In the present case, residual cerebral structural lesions were not evident on CT scan. However, it is possible that the PDA in our case could be attributed to functional cerebral defects following TBM. Investigations of cerebral functions such as PET (not available at our hospital) could add more information in this direction.

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