ORGANIC PSEUDO-PSEUDODEMENTIA—A CASE REPORT

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SUMMARY

A case of right anterior thalamic infarction which presented with a picture of pseudo-pseudodementia of the type described by Lishman is presented here and discussed with relevance to diagnosis and management of such unusual clinical problems.

Dementia is an organic brain syndrome which is characterised by loss of intellectual abilities of sufficient severity to interfere with social or occupational functioning (APA, 1980). Sometime such an impairment is seen in patients with a primary psychiatric disorder and which remits with recovery from the psychiatric illness. This clinical mimicking of dementia by other disorders is called 'pseudodementia' (Madden et al., 1952). Sometimes a localised cerebral lesion may apparently provide the trigger or focus for pseudodementia (Carney, 1983). Lishman (1978) noted that in the early stages of organic brain disease a patient may occasionally react in such a manner that his dementia is suspect of being more apparent than real. He termed this pseudodementia-like picture in a true dement as 'pseudo-pseudodementia'. Carney (1983) cites reports of cases of cerebral tumour, normal pressure hydrocephalus and rarer conditions like subacute sclerosing panencephalitis presenting with a picture of pseudodementia. In such cases the pseudodementia may be diagnosed and the patient treated for a functional condition, the local organic lesion remaining unsuspected. A case of anterior thalamic infarction who presented with an initial picture of pseudodementia (pseudopseudodementia) is reported here and discussed with relevance to diagnosis and management of such unusual clinical problems.

CASE REPORT

A 59 year old man came to the psychiatry clinic with the complaints of forgetfulness, sadness and disinterest of acute onset since one week, which started the day after a disturbing family quarrel between his daughter and the son-in-law which threatened her marriage. The patient reported having similar complaints 6 months ago following a similar family crisis but which remitted spontaneously within a week along with the calming down of the family atmosphere. On present examination the patient looked very depressed and agitated with all the symptoms of a depressive syndrome. He was repeatedly complaining of inability to remember even day to day events. Clinical test of cognitive function showed inconsistent responses by the patient who often pleaded inability to do even simple tests despite prompting. No cognitive deficit could be made out on the mental status examination which could explain the degree of subjective handicap reported. A complete physical and neurological examination revealed no abnormality. A clinical diagnosis of depression with pseudodementia was made and the patient was advised to undergo a psychometric evaluation and a CAT scan of the brain to evaluate for any organic causes, which were suspect because of the age of the patient. As the patient could not afford the scan because of financial restraints it was postponed. The psy-

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chometric evaluation using the Bender Gestalt Test, Wechsler Memory Scale and the Back's Depression Inventory showed evidence for organicity and defective orientation to time and space, poor verbal recall and paired associate learning with an intact attention, concentration and remote memory. There was a mild degree of depression on the BDI. In the following 3 weeks of follow-up the patient improved in his state of depression with symptomatic treatment, but the defect in recent memory and topographical disorientation came to the surface prominently. At this juncture the CAT scan could be done, which revealed an infarction of the anterior thalamic region on the right side with no other cerebral lesion. The patient was referred to the neurologist for evaluation and management of the cerebrovascular problem.

DISCUSSION

The initial presenting clinical picture of the patient was highly suggestive of a depressive pseudodementia. The history and mental status examination revealed many of the differentiating features of a pseudodementia (Wells, 1979). These are: past history of depression, current psychosocial stress factor, the presence of depressive syndrome currently, symptoms of short duration before medical help is sought, both the patient and the family are aware of the dysfunction and its severity, the patient emphasising disability and subjective distress, pleading inability to do even simple clinical tests of cognition, and absence of clear cut deficits in cognition. The suggestion of organic nature of the problem came to surface in the formal psychometric examination and clinically as the depressive symptoms subsided. The CAT scan confirmed the diagnosis of an organic brain syndrome of dementia in its early stages.

It is of interest to note that the patient had a self-limiting attack of memory disturbance and depression previously in temporal relation to a similar psychosocial crisis. Retrospectively it might be speculated that this episode could have been due to an ischaemic disturbance of the thalamic region which became infarcted with the second insult. Evaluating the case from the psychosomatic point of view of disease causation, both the episodes of illness are related to psychological and not a direct somatic stress. It is widely accepted that stress, emotional reactions and social circumstances can affect both the onset and course of medical illnesses (Mayou, 1984), which is applicable to this case. It is also possible that the thalamic lesion is the basis for both the cognitive as well as the emotional disturbances observed in the case. Besides its role in the control of recent memory, the thalamus, especially its anterior nucleus, has an important role to play in the regulation of emotions through its connection with the limbic system (Noback and Demarest, 1981).

The case highlights that in cases presenting with a picture of pseudodementia, especially in the elderly, caution should be practised in diagnosing the problem. A careful follow-up and relevant investigations are necessary before reaching the conclusion that there is no organic lesion to explain the pseudodementia. This is important because discovery of a neurological lesion changes the management as well as the prognosis of the case.

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