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Case Conference

A Dementia Syndrome of Dependency?

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The diagnostic process in a case of dementia associated with physical and emotional dependence is described. The utility of sodium amylobarbitone abreaction is considered. The role of both psychodynamic and organic factors are emphasised in the aetiological work-up. The organic factors discussed include alcohol, sedative tranquillisers, solvents, and metals. The term 'pseudodementia' is not favoured, and the phrase 'dementia syndrome of dependency' is proposed.

Objectives

Dementia in 'functional' psychiatric illness can lead to diagnostic and conceptual problems for the clinician. This is well illustrated by dementia in depression, where the term 'pseudodementia' is widely used to indicate the non-organic element. Pseudodementia has acquired a number of meanings, and because of this it has been argued that the term is best avoided (Mahendra, 1983). Much less common than dementia in depression is dementia in those exhibiting 'dependency'. Such a case was presented to a neuropsychiatry case conference held at the Institute of Psychiatry, and the conference was asked to weigh up the aetiological factors – emotional, social, and organic – and to comment on the diagnosis.

Presentation of case

A 53-year-old woman was referred by Dr George Stein from Farnborough Hospital for further assessment of abnormal behaviour associated with apparent intellectual decline.

Family history

The patient's mother died in 1985, aged 70. She was described as a resentful, demanding person, who obstructed her children's activities and boxed her daughters' ears until late adolescence. She had had in-patient treatment with ECT for depressive illness on many occasions. Her father was a violent man who drank heavily. He died in an accident when the patient was 12. The patient is the eldest of four siblings; both she and her sister are deaf. Her brothers have no physical or psychiatric problems. There is no family history of neurological illness or dementia.

Personal history

The patient was born and raised in south London. Her birth history and subsequent development were unremarkable. She passed her 11-plus examination, but left school at 15 with no further qualifications.

The patient had worked on the shop floor of a chromium plating factory for two years, as a chemist's assistant for three years, and in a dry-cleaning firm for some years.

The patient first married at 18, after a three-year courtship. The couple lived with the husband's family. There was considerable disharmony, and the patient sought solace with her husband's nephew. A divorce ensued, with custody of the two children given to the husband. When aged 30, the patient married the nephew, and they now have a son aged 28, who described the marriage as tumultuous, observing that he was effectively brought up by his father. At present the couple live in their own home, and the husband works as a night security guard.

Medical and psychiatric history

The patient has good general health but for a hearing problem. Symptoms suggestive of Menière's disease were present at the age of 19, and when 29 she had a left labyrinthectomy.

Her psychiatric history began at 29 when she was admitted to the Maudsley Hospital, suffering from depression in the context of marital difficulties. She would not discuss the conflict, and there was no response to tricyclic antidepressants. It transpired that before this presentation she had been treated by her general practitioner with a variety of drugs, including dexamphetamine sulphate, chloral hydrate, and phenelzine. At the age of 40 she was referred by her ENT specialist to the Department of Psychological Medicine at King's College Hospital for apparent elaboration of her aural symptoms. She was seen regularly, and twice admitted after attempted self-poisoning. A diagnosis of personality disorder was made, and sedative tranquilliser abuse was documented.

Her abuse of drugs was substantial. Between the ages of 40 and 44 she was known to be abusing glutethimide. When 49 she was taking high doses of benzodiazepines (flurazepam, 90 mg, with diazepam, 35 mg, daily). At the age of 50 she was discharged from out-patient follow-up to the care of her general practitioner on trimipramine (150 mg), nitrazepam (50 mg), diazepam (20 mg), prochlorperazine (20 mg), and betahistine (32 mg). The patient has always smoked 20–30 cigarettes a day. Since her mid-40s abetted by her spouse, her alcohol intake has steadily increased, reaching as much as 20 units a day in the last two years.

The patient was shy, but liked to be the centre of attention in her own circle. From the outset she had a dependent relationship with her husband, and was distant from her children. When she was 49, the hospital social worker for the deaf had commented upon the high level of domestic stress and the husband's threats to leave. It was said by her relatives that she took refuge from her worries in her symptoms.

Present illness

Against the domestic background described, the family first became aware of more disturbing behavioural changes in February 1986, when the patient was 52. Her husband found her going over her three-times table aloud, which she struggled to get right. Thereafter, he became aware of a gradual decline in her faculties. She would make gross mistakes when laying the table, leave the gas cooker on unlit, and made cold coffee. She had difficulty dressing, and seemed to become forgetful. She began to repeat herself, asking the same question over and over. Her mood would change rapidly from inappropriate happiness to tearful irritability. The change progressed over nine months. There was a diminution in her range of interests, and neglect of personal hygiene. Her husband employed a night-sitter while he was at work.

In July 1986 she was admitted for investigation following a domiciliary visit by a consultant psychiatrist. All physical tests, including EEG and computerised tomography (CT), were normal. Psychometric testing showed generalised intellectual underfunctioning, but normal orientation.

A tentative diagnosis of Alzheimer's disease was nonetheless made. She attended an Alzheimer's disease day centre, where according to staff she behaved similarly to other patients. Problems arose when attempts were made to reduce her medication, unchanged since 1984 (see above). She became increasingly hostile, aggressive, and difficult to manage at home. Once readmitted to hospital, haloperidol (10 mg t.d.s.) settled her disturbed behaviour; repeat EEG and CT were normal.

Two months later, owing to difficulty swallowing and unsteady gait, she again required admission, and at this point she was referred to the Maudsley Hospital for assessment. On arrival she required total nursing care, although as her medication was withdrawn the motor signs resolved, and she was able to walk normally and, with encouragement, dress herself. She continually requested her medication be reinstated. While she was on the ward her husband was a constant visitor, and the nursing staff observed his indefatiguable attentiveness (for example, he would light and hold her cigarettes for her, although she was able to do this for herself).

Mental state examination

Her speech was dysarthric as a result of poorly fitting dentures. She gave monosyllabic responses, which were often inappropriate and of a clipped, repetitive quality. There was perseveration on verbal tasks, but no dysphasia. She was distractible and disinhibited. Her mood was often labile, and alternated between mild euphoria and tearfulness. There were no depressive or psychotic features.

Cognitive examination was impeded by her distractibility and poor motivation. However, she was fully orientated, and, despite poor attention and concentration, she was able to register and repeat a name and address at one attempt, with full recall at three minutes. She had difficulty in performing complex motor and sequencing tasks. Her general knowledge was commensurate with her educational background.

Physical examination

On physical examination she had a kyphosis, and prominent corneal arcuses. She was totally deaf in the left ear, and had impaired hearing in the right ear, which was corrected by the use of a hearing aid. There were no primitive reflexes. There were no physical abnormalities in any of the major systems.

Investigations

Informant histories were obtained from four relatives. The following investigations were normal: full blood count, erythrocyte sedimentation rate, liver function tests, calcium, glucose, lipid studies, B12, folate, thyroid function tests, angiotensin-converting enzyme, porphyrins, auto-antibodies, drug screen, poisons screen, copper, lead, syphilis serology, lysosomal enzymes, urine culture, ECG, EEG, chest and skull X-rays. A CT scan showed mild frontal atrophy.

Abnormal macrophage-like cells were seen in the cerebrospinal fluid (CSF) on the first but not subsequent specimens (very large rounded cells with rounded nuclei larger and less dark than in a plasma cell). Oligoclonal banding was absent, and all other studies of the CSF were normal.

Sodium amytal abreaction (amylobarbitone 0.4 mg over 30 minutes) gave a dramatic improvement in the patient's mental state. She seemed an entirely different person. She spoke in a fluent, friendly manner, and her repetitive demands ceased. She apologised for her behaviour, and after the interview she engaged in conversation with other patients for the first time. She was able to lay the tables normally, and washed up after the meal correctly. This state lasted four hours, wearing off slowly and leaving a slight residual improvement. The procedure was repeated five weeks later, but at this stage it made no difference; this may have been due to intervening changes in medication.

The patient was neuropsychologically assessed when she originally presented, and while her pre-morbid IQ was estimated at 109, all other tests indicated performance in the defective range. She was tested at the beginning and end of her stay at the Maudsley Hospital. Her orientation was normal throughout. Initially on the Kendrick battery (Kendrick *et al*, 1979), she scored well in the demented range, with virtually identical scores on two occasions days apart. When tested three months later, there was a 30% improvement, taking her into the non-demented range. Impairment in attention was present at both test periods, and she consistently adopted a poor strategy to copy a complex figure – findings compatible with organic damage in the frontal region.

Interview

The patient was interviewed by Professor Lishman, her consultant. She entered the conference room unperturbed by the audience, recognised one of her doctors, and kissed him on the forehead. Throughout the interview she paid little attention to the questions posed, but constantly asked for cigarettes and leave to go home. Her cognitive ability, as described above, was demonstrated to the audience.

Conference discussion

DR MARIA RON *(consultant neuropsychiatrist)*: This case demonstrates the fact that patients become more complex the more complicated the investigations done. The most dramatic feature in her mental state at present is a facile disinhibition. It certainly seems that even at her best over the past months of observation, she has never regained her normal level of function. I believe chronic organic brain disease underlies the clinical picture, although there is no obvious reason why her abnormal personality traits and previous neurotic illness should not colour the presentation. It is also significant that she is deaf. Alzheimer's disease is a possibility, but unlikely. The two other relevant factors are firstly the history of alcohol abuse, which could explain the dementing illness or have at least exacerbated the underlying pathology, and secondly her drug usage, which is possibly relevant to her fluctuating behaviour and certainly the explanation of her earlier extrapyramidal signs. There are too many aetiologies here perhaps!

PROF. LEVY *(professor of old age psychiatry)*: This is definitely not the clinical picture of Alzheimer's disease; for instance she recognised and correctly named her doctor here today. It also seems that in fact her cognitive function has not altered substantially since her first admission in 1963. This woman has probably been ill since her late 20s. It may be a rare degenerative brain disorder, the exact nature of which will remain uncertain, at least in life. The functional disturbance grafted on reflects her pre-morbid personality.

PROF. LANTOS *(professor of neuropathology)*: There is a toxic flavour to the presentation that suggests that alcohol or drugs play a part in the aetiology. Her work in either the chromium plating factory or the dry cleaners may have brought her into contact with potential toxins. Dr Janota noticed the unusual cells in the CSF, which raises the remote possibility of a storage disease. Unfortunately, the repeat samples did not show similar abnormalities, but it would be worth pursuing further, maybe with a rectal biopsy in order to exclude this.

DR JACOBY *(consultant in old age psychiatry)*: I have little to add to what has been already said; only the post-mortem will tell. The case reminds me of the typical Russian addictive personality – they will consume anything they can lay their hands on, including aircraft engine fluid, and give rise to many obscure neurological syndromes by these practices. There may have been some self-administered unknown toxic factor which we will never identify.

DR SAHAKIAN *(lecturer in neuropsychology)*: Recently Dr David Freed [Freed & Kandel (1988)] reported that occupational exposure, particularly to metal vapours and hydrocarbon solvents, was nearly eight times more common in patients with Alzheimer's disease relative to control subjects. Dementia has been described occurring many years after exposure to toxic solvents, and may well be relevant here. It is certainly an area worthy of further study.

DR STEIN *(consultant psychiatrist)*: The management problems to address are presently those of counselling the husband to help him deal with her total dependence on him, and our views on the likely future course of his wife's condition. Also to be considered is the need for some form of pharmacological behavioural control.

The consensus on the diagnostic issue was that this is unlikely to be Alzheimer's disease. The differential diagnosis discussed lay between an early form of non-Alzheimer's frontal lobe dementia, an alcohol-induced dementia, and a dementia due to an environmental toxin. The colouring of the clinical presentation by personality factors and substance abuse was emphasised.

Decisions were made to perform further investigations, including a rectal biopsy to exclude any possibility of a lipid storage disease such as Kufs' disease, single-photon emission computerised tomography (SPECT), and visual-evoked responses.

Trials of a variety of medications were recommended, including chlormethiazole, carbamazepine, sulpiride, and amylobarbitone, with the aims of stabilising mood (her lability was felt to reflect frontal lobe pathology rather than a functional affective state), to control behaviour, and to avoid extrapyramidal side-effects.

Addendum

A SPECT scan showed no abnormality; in particular, normal anterior uptake was seen. A rectal biopsy gave no diagnostic results, and visual-evoked responses proved normal.

The only effective medication was amylobarbitone. She was discharged on 100 mg t.d.s., which produced a small improvement in mood stability and sleeping pattern, but little change in repetitious behaviour. Over the ensuing four months this was gradually reduced and stopped. The gross features of dementia lifted, leaving minor impairment only. The patient is able now to manage the cooking of simple meals, shopping with her husband, ironing, and writing to people. She remains demanding, however, and is difficult to distract when she has decided she wants something. She remains heavily dependent on her husband, although this is expressed in a more reasonable manner, and the couple's relationship has improved considerably.

The husband was counselled on what to expect of his wife, and given a frank explanation as to our views, that we could not exclude an organic process, but felt physical and emotional dependency had coloured the presentation. He was encouraged to increase her independence.

Discussion

The case illustrates the value of broad contextual inquiry; valuable insights were gained by exploring the psychosocial context alongside the organic. The

potential rewards of such an approach have been alluded to in a recent description of the scope of organic psychiatry, which emphasises that there are "psychodynamic, social and cultural aspects of neuropsychiatry" to be considered (Lishman, 1987).

The point was made in the conference that a final diagnosis would probably emerge only at post-mortem. Brain biopsy has represented the only feasible way of obtaining a neuropathological diagnosis ante-mortem (Sim *et al*, 1966), but with new imaging techniques, brain biopsy has become relatively uncommon.

The clinical utility of sodium amylobarbitone abreaction was instructive, and a case might well be made for its resurrection in selected cases. Amylobarbitone abreaction is reported to improve non-organic psychiatric illness in the context of dementia (Ward *et al*, 1978 (case 1)), and exacerbate certain chronic organic brain syndromes (Weinstein *et al*, 1953).

The patient's pre-morbid personality and marital situation are of considerable interest as factors modifying the form of any putative underlying organic disorder. The patient's emotional problems, including her dependence on her husband, might be reasonably hypothesised to be the cause of her later dependency on drugs and alcohol. In turn, the latter certainly coloured the dementia, but may even have caused it. Some of the potential ways in which psychodynamic and organic factors may interact are thus illustrated; investigations should not be only organic if an entire understanding of the patient is to be achieved.

Should further aetiological understanding be sought? The conference participants made helpful comments on the possible organic pathologies. Candidate processes included alcohol-induced dementia, a dementia produced by exposure to a neurotoxic substance, and a primary dementing process, the presentations being modified by personality, alcohol, and abuse of sedative tranquillisers.

The patient's level of consumption and clinical presentation are consistent with alcohol-induced dementia, in which characteristically only moderate disturbance of brain function occurs, with the potential for reversal in some degree (Lishman, 1981). The CT appearances of more general cortical shrinkage and ventricular dilatation usually, although not invariably, seen in this form of dementia were absent in this patient. The improvement after the withdrawal of the drugs and alcohol is as would be expected; these substances may also be expected to exacerbate an underlying unrelated organic process.

While sedative tranquillisers such as glutethimide and the benzodiazepines are able to cause a degree of cognitive impairment, with long-term use it is less certain that they can cause dementia (Golombok *et al*, 1988). It would be tempting to speculate that the amylobarbitone-induced reversal came about through the temporary reversal of a chronic change in receptor sensitivity, which had been originally induced by drug abuse. Although plausible, such an idea must at present be regarded as speculative.

What of the possibility that this woman may have an underlying organic dementia induced by solvent or metal vapour? Authoritative reviews of the Danish literature on the association between occupational inhalation of organic solvents and pre-senile dementia conclude that the case is unproven (Errebo-Knudsen & Olsen, 1986; O'Flynn, 1988). It should be recalled that carbon disulphide, one of the best-known occupational neurotoxins, is a solvent, and can cause chronic cognitive deterioration even with low-level exposure (Hanninen, 1971). This patient would have been exposed to carbon tetrachloride, which used to be commonly employed in the dry-cleaning industry, but unlike carbon disulphide there are no studies which show chronic cognitive deterioration (Hartman, 1988).

The patient was exposed to the metal chromium while working in a plating works in her youth. A number of metals are associated with the development of dementia. These include iron, lead, manganese, aluminium, copper, and mercury. Chromium is not as yet included, although it has been reported to cause an encephalopathy (Hartman, 1988).

There is a possibility that the patient may be suffering from an unrecognised and insidiously progressive brain disease, such as a storage disease. The first CSF specimen contained abnormal cells, but this was not confirmed on the second tap or by the rectal biopsy.

The case has raised a number of valuable diagnostic and conceptual issues, and demonstrates the complexity of investigating those cases between 'functional' and 'organic' categories of disease. Our conclusions on reviewing the evidence was that this was a woman with an abnormal pre-morbid personality, who developed emotional dependence on her husband, and later dependence on sedative tranquillisers and alcohol. In time, this state coloured a dementing process of uncertain origin. Whether this might have been related to alcohol or any of the other causes of dementia discussed remains an open question. The recognition of the alcohol and sedative tranquilliser abuse and its gradual removal has brought about a partial reversal of the deficit.

An issue remains – might this woman be said to be suffering from pseudodementia? Rather than using the term 'pseudodementia' to describe the association of substance dependency and/or emotional dependency with dementia, we would like to follow the precedent of Folstein & McHugh (1978), and propose the use of the phrase 'dementia syndrome of dependency'. The use of the phrase has the advantage of recognising the association of the two conditions, leaving the clinician to make a full aetiological inquiry, both psychosocial and organic.

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