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Klüver–Bucy Syndrome and Psychiatric Illness

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A 52-year-old woman, whose initial psychiatric presentation at the age of 15 was with a disorder resembling schizophrenia, developed symptoms of Klüver–Bucy syndrome, and is now thought to suffer from an organic psychotic disorder. Klüver–Bucy syndrome must be distinguished from symptoms of schizophrenia or affective disorder; its presence suggests an organic process.

Klüver & Bucy (1939) described a syndrome which followed bilateral removal of the temporal lobes of rhesus monkeys, and which consisted of 'psychic blindness' (inability to recognise familiar objects, even after repeated exposure), 'hypermetamorphosis' (a strong tendency to attend and react to every visual stimulus), oral exploration of available objects, emotional unresponsiveness, and an increase in sexual activity. A similar syndrome was reported in man by Terzian & Dalle Ore (1955), who described a 19-year-old man with epilepsy who underwent bilateral anterior temporal lobectomy with removal of most of the uncus and hippocampus. This patient was unable to recognise close relatives. He displayed an increase in sexual activity, emotional unresponsiveness, increased appetite, and a severe memory disturbance, but did not show oral exploratory behaviour. Further patients with elements of the Klüver–Bucy syndrome have been described in association with Alzheimer's disease, Pick's disease and encephalitis (Lilly *et al*, 1983), adrenoleukodystrophy (Powers *et al*, 1980), trauma (Lilly *et al*, 1983; Stewart, 1985), toxoplasmosis and hypoglycaemia (Poeck, 1969), acute intermittent porphyria (Guidotti *et al*, 1979), Huntington's disease (Janati, 1985), and vascular lesions (Poeck, 1969; Shraberg & Weisberg, 1978).

When Klüver–Bucy syndrome occurs in man it may be associated with a mixed anterograde–retrograde amnesia; with verbal, rather than physical, hypersexuality; with manual, rather than oral, exploratory behaviour; and with rage reactions (Shraberg & Weisberg, 1978; Stewart, 1985). Rage reactions and some other features of Klüver–Bucy syndrome in man may respond to treatment with carbamazepine (Stewart, 1985).

Psychiatrists unfamiliar with Klüver–Bucy syndrome may fail to distinguish the features from symptoms of schizophrenia or affective disorder. The importance of the syndrome lies in its association with organic disorders.

Case report

M was born in 1936 after an uneventful pregnancy and delivery. Her mother was a 26-year-old housewife, her father a 26-year-old factory worker. She attended school from the age of five and was described in contemporary reports as an average scholar. She left school at the age of 15 to start manual work with a large industrial company, but gave the job up after a few months because she had difficulty concentrating and felt tired. She complained of a peculiar sensation, which she described as a cloud settling on her, and told her parents that she could hear voices in her head.

In 1951 she was admitted to a psychiatric hospital and was noted to be pyrexic. She was treated with electroconvulsive therapy (ECT), improved, and was discharged after three weeks. At home, however, she rapidly became withdrawn and silent, and would sit for hours hardly moving and not replying to questions. At other times she had difficulty keeping still and would bite her nails continually. She giggled to herself, refused to wash, and insisted on sleeping in her clothes.

In 1952 (aged 16) she was admitted to a different psychiatric hospital, and at this time was incontinent of urine and faeces. A diagnosis of hebephrenic schizophrenia was made, and insulin treatment started. She had a total of 30 comas lasting 5–20 min, induced by doses of insulin of up to 260 units daily. She had tonic–clonic seizures on 18 occasions. There was no improvement. By March 1953 she was extremely restless, was emotionally incongruous, and talked to herself as if hallucinating. Over the following months she became mute and then developed elements of the Klüver–Bucy syndrome. She continually explored objects with her hands and mouth, ripped fabric from furnishings and ate it, ate her faeces, removed her clothes, and was often physically aggressive for no apparent reason. She also giggled, clucked and grimaced continually, and although she did not speak, could sing simple songs if prompted. M was nursed in a high-dependency unit. Treatment with reserpine, pyridoxine, nicotinic acid, ECT, perphenazine, thioridazine, and trifluoperazine produced no improvement. Between 1969 (aged 33) and 1971 (aged 35) she had four spontaneous tonic–clonic seizures.

Her distress and her behavioural abnormalities were so great that after discussion with, and consent from, her father, and with the agreement of an independent psychiatrist, she was referred for psychosurgery.

Pre-operatively electroencephalography (EEG) showed high-voltage sharp and slow waves appearing synchronously between the frontal bases and the parietal convexities on both sides.

A posterior cingulectomy and cortical biopsy was performed in 1974, when M was 38. The biopsy contained normal brain tissue. A paramedian frontal lobotomy was performed after an interval of three months. M improved after this operation, becoming less agitated and aggressive, although with a tendency to become more disturbed premenstrually. In 1985, at the age of 49, M developed an intestinal obstruction. A colonic mass was removed and a colectomy and hysterectomy were performed. A specimen of the mass sent for histological examination showed lymphocytic lymphoma, with involvement of lymph nodes but with cut ends free of tumour. M was referred to a haematologist, who felt no further action was necessary. Since the operation she has had occasional episodes of constipation, but no weight loss or other signs of malignancy.

M continues to be nursed on a high-dependency unit. Her activity on the ward is minimal and largely purposeless. She is occasionally physically aggressive, usually in response to provocation by other disturbed residents. Her appetite is reasonable and she is able to feed herself. She dresses herself with assistance. Her speech consists of a few words, which are poorly enunciated and sometimes used inappropriately. She appears to be disorientated in time and place, and needs to be escorted when walking within the hospital. M is visited regularly by her father, but she shows no anticipation of the visits or any recognition of her father or other relatives. On one occasion she needed regular intravenous injections, and when she saw the doctor approaching became agitated and shouted 'needle, needle'. She is incontinent of urine and faeces, and has an orofacial dyskinesia but no other abnormal movements. She has no gross motor or sensory deficits, and although her tendon reflexes are bilaterally brisk, they are not pathologically so. EEG performed in 1988 only showed abnormalities consistent with psychosurgery, and a computerised tomography scan of the head showed a minor degree of cortical atrophy in the parietal lobes, and frontal-lobe atrophy presumed to be a result of the leucotomy. No temporal-lobe abnormalities were seen.

In view of the Klüver–Bucy symptoms, seizures, incontinence, disorientation, amnesia, and aphasia, the presumptive diagnosis is now of an organic psychotic disorder of unknown aetiology.

Discussion

M's initial presentation was with symptoms suggestive of schizophrenia. The notes document disordered speech, thought blocking, and catatonic symptoms. The underlying basis for the psychotic symptoms may have been an encephalitis. Psychiatric symptoms were preceded by tiredness and lethargy, and she was pyrexic on first admission. Mahendra (1981) noted a decline in catatonic symptoms since the 1930s, and postulated the decline of an infectious illness as a possible cause. The normal brain biopsy, however, argues against an encephalitic aetiology. An alternative explanation is that treatment of an initial schizophrenic illness with deep insulin therapy produced a chronic organic psychotic syndrome (dementia) as a result of prolonged hypoglycaemia. Dementia was a recognised complication of insulin treatment (Spencer, 1948). However, the notes do not document a prolonged coma or a sudden deterioration in M's condition at any point during treatment with insulin.

Her present clinical state strongly suggests that the end result of whatever morbid process occurred has been an organic psychotic disorder (dementia) of unknown origin. The diagnosis could have been revised earlier if the Klüver–Bucy symptoms had been recognised, and a more accurate prognosis provided for the patient's relatives.

If several of the features of Klüver–Bucy syndrome occur in a patient with an apparently uncomplicated affective or schizophrenic illness, the psychiatrist should be alerted to the probability of pathology of temporal-lobe limbic structures. Symptoms identical to those in affective and schizophrenic psychoses may be produced by pathology in this area (Malamud, 1967). Psychiatric illness with low mood, delusions and hallucinations may precede the Klüver–Bucy syndrome when it occurs in association with Alzheimer's or Pick's disease (Pilleri, 1966). The presence of Klüver–Bucy symptoms should therefore suggest that any accompanying psychotic symptoms have an underlying organic cause.

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Mood Disorder, 'Pre-ictal' Psychosis and Temporal Lobe Damage

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A patient with an abnormality in the right temporal lobe presented with episodes of mania many years before the clinical manifestation of both a simple partial seizure and complex partial seizures.

An association between epilepsy and psychotic illness is well recognised. Falret (1854) was one of the first to suggest such an association which was further supported by Slater & Beard (1963) in their description of psychosis developing on average 14.1 years following the diagnosis of epilepsy. Flor-Henry (1969) linked schizophreniform and manic-depressive psychosis to abnormalities of the dominant and non-dominant temporal lobes respectively. Therefore, the development of psychosis following epilepsy has been associated with the duration of epilepsy, abnormality of the temporal lobe itself, and with brain damage. We report a novel case of abnormality of the right temporal lobe, with episodes of mania many years before the clinical manifestation of both a simple partial seizure and complex partial seizures.

Case report

The patient was a 19-year-old man brought to the UK from Africa at the age of one. There was no family history of mental illness or epilepsy, and his early developmental milestones and obstetric history were within normal limits. However, he was later ascertained as being in need of special education and subsequently attended a residential school for children with moderate learning difficulties. His pre-morbid personality was described as shy and friendly.

At the age of 15, following a sexual assault, he became overactive, distractible and destructive, and was admitted in quick succession to two different psychiatric hospitals. No specific diagnosis was made on either occasion. His condition settled rapidly and he was placed in a local children's home. The following year he again suffered a relapse in his mental condition and was admitted to yet another psychiatric hospital, where a diagnosis of hypomania was made. His mood settled rapidly and he was discharged to a local hostel on no medication.

At the age of 18, he suddenly was unable to sleep for more than three hours at night and again became overactive, distractible, irritable, and impulsive. There were no reports suggestive of delusions or hallucinations. After three years in this state he ran into the street carrying a billiard cue and attacked a car and its driver. He was arrested and placed in a local remand centre. There, on the basis of a full mental state examination (including cognitive assessment), a diagnosis of hypomania was made, and he was admitted to a secure unit. The patient settled after a few days on regular sodium amytal medication, and lithium carbonate