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Kleptomania in a 13-Year-Old Boy

A Sequel of a 'Lethargic' Encephalitic/Depressive Process?

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A 13-year-old boy developed severe kleptomania after a depressive illness. This was a response to psychological trauma, and appeared in the context of a probable 'lethargic' encephalitic process.

Kleptomania is mentioned as a diagnosis in current classificatory systems in psychiatry, but little is known about it and the literature in the area is jejune. The person repeatedly fails to resist impulses to steal objects, which are not acquired for personal use or monetary gain. They may be discarded, given away or hoarded. The central psychopathological mechanism appears to be loss of impulse control but in the DSM–III differential diagnosis (American Psychiatric Association, 1980), pathological stealing should be distinguished from: recurrent stealing by healthy people (acts are planned and there is an obvious motive of personal gain); stealing which is part of a conduct disorder or antisocial personality disorder; organic mental disorder with memory impairment and intellectual deterioration; psychotic states. The condition is very rare in childhood.

Case report

John, aged 13 years, was transferred to our regional child psychiatric unit from a paediatric ward. Encephalopathy, presumed but not proven to be of viral aetiology, had been diagnosed, and he was profoundly depressed.

His symptoms had started eight months before admission (in December) with a flu-like illness followed by weight loss. We have few details of this illness. Symptoms included myalgia, lethargy, anorexia, mild disorder of the upper respiratory tract, and mild pyrexia. It appears that the majority of these symptoms resolved but were followed by marked weight loss. Five months afterwards his grandmother, who had been the primary carer for John since he was two years of age, attended the funeral of one of her daughters abroad. During this time John discovered the tragic details of the death of his mother, who had been brutally stabbed to death together with her four-year-old son when John was two years old, and he found out that, contrary to his belief until then, his grandmother was not his mother. When his grandmother returned she found John would cling to her and be overanxious of her well-being. Three months after her return John was admitted to a paediatric ward with 19 kg weight loss (three stone) in seven months, and two-week history of night sweats, listlessness, lethargy, withdrawal and anorexia.

On admission to the paediatric ward he had a low-grade pyrexia with no other signs. However, he became increasingly lethargic and miserable. He was noted at times to fall into a deep sleep during the day and he seemed very confused on arousal. He became more and more drowsy, sleeping for most of the day and at times he was barely rousable. His depression deepened and he would weep

uncontrollably. These catastrophic reactions were precipitated by talking about his mother's death.

Investigations included two electroencephalograms (EEGs) (performed within one week during the acute 'lethargic' phase of illness), which were grossly abnormal, with diffuse slow-wave activity, suggestive of diffuse encephalopathy and encephalitis. A computerised tomography (CT) scan and laboratory and clinical tests proved normal. EEGs performed two to three weeks later showed an improvement.

On admission to the psychiatric unit, physical and neurological examinations were normal. He wanted to spend his time sleeping in a foetal position, refused to do anything for himself, and shouted in a loud monotonous voice 'my legs', 'help me'. He complained of 'stabbing pains' in his legs which stopped him from standing unsupported although he was physically able to do so. He often described his behaviour as being that of a two-year-old and would only eat mashed, wet food. He looked profoundly depressed and would slap his face, shouting at nurses 'kill me', 'finish me off'. He talked frequently of frightening dreams and of not knowing the difference between dream and reality. He was reluctant to talk about his 'real Mum'.

John was the third youngest of four siblings. All children were in the care of the local authority and had been brought up by the maternal grandmother, the youngest two believing her to be their mother. The grandmother had cared for the children adequately, if anything tending to overindulge John. The eldest son had a history of minor behavioural problems. John's birth and early development were entirely normal. Before his illness he had had minor behavioural problems and was described as slightly below average ability in school. He was described as disruptive and immature at school, though likeable and sociable. There had been occasional truanting and one episode of shop-lifting with friends.

There was a strong family history of psychiatric illness on the paternal side of the family. Details are lacking since our only informant was John's grandmother and we were unable to obtain psychiatric records. John's natural father was admitted to psychiatric hospital following his wife's murder, for treatment of depression. Several of his siblings had a psychiatric history and the youngest sister, who was an accomplice to the murder of John's mother, was found to be suffering from morbid jealousy and was committed for treatment. A few months before John's admission to our unit, his grandmother's only remaining daughter was murdered abroad. We do not know who murdered either John's mother or aunt, but do not believe the two events to be connected.

Our formulation was that in the context of a probable viral encephalitis and in response to psychological trauma, John had developed a severe depressive illness. He was treated with much encouragement and intensive physiotherapy as a means of restoring function and activity and reducing withdrawal. He had individual therapy sessions and we conducted joint sessions with him and his grandmother for facilitating talk about his mother's death and grieving by both.

He was also started on imipramine, increasing to 100 mg daily. After two weeks he became less sleepy, more alert and mobile, and he no longer complained of pains in his legs. He still, however, expressed a wish to be dead. Concurrent with his leg pains resolving, he began stealing (money, purses, pens and jewellery were disappearing from offices, drawers and lockers). He was devious. When discovered he became distressed, hit himself, and would shout "kill me, I wish I were dead". He promised faithfully not to steal again but within minutes he would be looking for opportunities. He did not spend the money or make use of any of the items, but simply hid them in his bed. Behavioural therapy failed. When the stealing started he said that he did it because when he was found out we would kill him. As his mood improved he ceased to use this explanation but could not give any other.

At this time we were able to complete psychometric testing. His full-scale IQ was 68 (77 on the verbal scale, 63 on the performance scale), and his reading age was 9 years 11 months. These results were likely to be an underestimate, as he was lethargic and drowsy during testing. When his reading age was checked three months later (on a different test), it was 11 years 1 month, with a comprehension of 11.8 years.

Repeat EEGs (six weeks and four months after the initial abnormal recordings) showed a low-voltage desynchronised record, poorly formed for the patient's age but otherwise normal. A repeat CT scan showed the ventricular system to be a little full but not grossly abnormal. The cortical sulci were also large, but no focal lesion was seen. There were no areas of altered attenuation or pathological enhancement.

Within two months of starting his antidepressant medication he was considered to have recovered from his depression. However, as he emerged from this he showed many features of a personality deficit: disinhibition, impulsiveness emotional lability, and lack of affective depth. Emotional lability was observed particularly with respect to his compulsive stealing: when confronted with it he would become distressed, shouting that he wanted to be dead and weeping, but then within seconds he would be laughing and joking again. When confronted at later stages in his condition he would become aggressive and threatening. He was talkative but repetitious in his topic of conversation. He showed little interest in ward and school activities and he remained prone to sleep during daily activities.

His stealing became increasingly uncontrollable and extended well beyond the confines of hospital and home. He was picked up and detained by the police on numerous occasions and had several court appearances, but his stealing continued unabated. Containment and placement became a major problem, with neither his family nor psychiatric or social services being able to provide an adequate setting for control and treatment. Ten months after he was first admitted to the psychiatric unit he was eventually placed in a secure local-authority unit for delinquent boys. The monitoring of his psychiatric state was taken up by the forensic adolescent psychiatric services. He remained at the secure unit for nearly one year with little change in his condition and no recurrence of a depressive state. He was then transferred to the behavioural management unit for psychiatrically disturbed adolescents of a private psychiatric hospital.

Discussion

This boy's stealing was characteristic of kleptomania as described in DSM-III. The thefts were recurrent, impulsive and apparently senseless, and John was observed to become tense before the act and to derive pleasure from it. The stealing started during his recovery from a severe depressive state and in the initial stages it was linked to his morbid thought content. Its persistence, however, after the affective state had resolved can be explained in terms of the remaining personality changes which accord best with DSM-III's organic personality disorder, characterised by emotional lability, impairment in impulse control, apathy, and indifference to most activities. In view of the previous profound depression, we considered the possibility of mania, but there was no clear evidence of an elevated, expansive or consistently irritable mood or of excitement, flight of ideas, or convincing grandiose ideas, and the stealing continued after his antidepressants were stopped.

Excessive sleepiness and lethargy were features of the condition. Although these symptoms can occur in depressive states, the associated encephalopathic signs and the personality sequelae raise the issue of whether this was an abortive form of encephalitis lethargica. Many authorities consider that encephalitis lethargica disappeared completely before the onset of World War II. However, possible cases have been reported since (Leigh, 1946; Espir & Spalding, 1956; Rail & Scholz Swash, 1981). Moreover, polymorphism was a striking feature of the disease (epidemics differed both in acute phases and in incidence of sequelae) and the possibility has been raised of present-day mild or attenuated clinical pictures – particularly in children and young adolescents – increasingly dominated by psychiatric manifestations and resulting in considerable subsequent psychiatric disability (Lishman, 1987). Our patient showed several features described by von Economo in his classic account in 1929: peak winter incidence, a pre-clinical phase resembling influenza, hypersomnolence persisting for several weeks, rapid debility, weight loss, and low-grade pyrexia. Investigations performed during the acute phase failed to isolate a causative agent. Neurological signs were lacking in this case; however, these, in particular disturbance of ocular movements, may be fleeting and it is possible that they were missed.

The account by Lishman (1987) of a typical post-encephalitic personality could certainly be directly applied to this patient. Fairweather (1947) gives a vivid illustration of severe sequelae in post-encephalitic patients without Parkinsonism or gross physical defect admitted to Rampton State Institution for violent and dangerous patients. These included damage to property and theft, as well as damage to others, sex offences and self-injury.

Our case highlights the lack of adequate services for adolescents with problems such as this boy's and raises the issue of what type of facilities should be available for the treatment/containment of adolescent recidivists with probable post-encephalitic personality disorders.

Severe psychopathology such as this boy's is rare in childhood but it can lead to considerable psychiatric disability. This case suggests that severe or particularly strong psychological stresses as well as biological factors may be required for its emergence.

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