CASE REPORTS

Failure to thrive and anorexia nervosa

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Summary

A case study is reported of an anorexia nervosa mother who battered her elder male child ('battered baby') and in collusion with her psychopathic husband starved to death her 10-week-old daughter. The association of anorexia nervosa in the mother with death by starvation has not previously been reported in the literature. The possible underlying psychopathology is briefly discussed.

THIS paper describes the case of a woman with a history of anorexia nervosa, who battered her first child and starved her second child to death. The matter came to attention during a major research study into the psychiatric aspects of parents of battered babies. The association of anorexia nervosa in a mother with failure to thrive and death of her child does not appear to have been previously reported.

Case report

Mrs P when first seen was aged 22 years. In August 1967 she was involved in an automobile accident. Her injuries were not severe consisting of minor bruising, abrasions and lacerations over her body but not her head. Following this she developed loss of confidence and a phobia of driving or being driven. In June 1968 she was referred to a consultant neurologist, complaining of frequent 'blackouts', general nervousness and loss of weight from 69.3 kg to 48.0 kg and marked loss of appetite. Although she did not actually suffer from amenorrhoea her periods were very scanty, consisting of a relatively slight loss lasting a day or two and occurring every 19 days. Physical examination revealed a very thin woman but with no other abnormal physical signs. Haematological and biochemical investigations were normal. Her EEG showed only a slight non-specific abnormality. A diagnosis of anorexia nervosa was made and the patient was referred to a consultant psychiatrist who concurred with this.

Her early development was normal apart from slight feeding difficulty in infancy. She reached a

weight of 69.3 kg and 69 cm height during adolescence. Her school years were uneventful apart from occasional 'giddy turns'. On leaving she became a shop assistant.

In January 1968 she married. Her parents strongly objected to her choice of husband. He was 32 years of age, suffered from a squint and a stammer and had often been ridiculed at school. He later had several unskilled jobs, and was dismissed or made redundant from most of them. When seen he had not worked for 18 months, in order, he said, to look after his wife. He showed some features of an aggressive but inadequate psychopathic personality. There was no history of any psychiatric disorder in either the patient's or her husband's family.

In September 1969 Mrs P gave birth to a normal male infant weighing 3.1 kg. Labour and delivery were normal. Two months later it was noticed that the baby was undernourished with very little subcutaneous fat. Head bruising was noted several times by the health visitor and each time was inadequately explained. Both parents denied the baby was difficult to feed and denied inflicting trauma. However, they were extremely reluctant to allow the general practitioner and health visitor to examine the child. The child is still rather thin and appears retarded in development and some slight facial bruising is still seen on occasions.

In December 1970 Mrs P gave birth to her second child, a female weighing 3.2 kg. Labour and delivery were normal and the baby fed well in hospital and shortly after discharge. Ten weeks later, however, the baby was admitted as an emergency following a visit by the health visitor who noticed she was extremely cold and underweight. Examination revealed an extremely emaciated, dehydrated and corpse-like child. Temperature was 25° C, weight 2.7 kg. She was anaemic and there was no pulse. She had an ulcerated nappy rash. A diagnosis of hypothermia and marasmus was made. Despite intensive efforts at resuscitation she died the following day. Necropsy revealed no evidence of bruising, fractures or internal injury. The failure to thrive was attributed to poor

feeding. Neither parent could account for the rapid weight loss. They stated the baby was feeding well and denied starving or neglecting her in any way. A verdict of 'lack of care' was recorded.

Since the death of the baby Mrs P has lost another stone in weight (present weight 42.7 kg). Mr P is still not working and the family is living on social security benefits.

Intelligence, personality and attitude tests were given to Mrs P 3 months after the death of her child. These showed an IQ of 84 (WAIS) and on the Eysenck Personality Inventory (EPI) an extremely high N score and an E score well above average. Her lie (L) score was very low.

Fould's test (Caine, Foulds & Hope, 1967) showed her to be extremely hostile, chiefly towards other people but with considerable self-criticism and guilt. As she was still denying responsibility for the death of her child and failing to express grief she was given the TAT (Murray, 1943). Her responses were brief and could generally be taken to indicate lack of involvement. However, the chief emotions expressed in her stories were anxiety, guilt and fear sometimes linked with physical aggression and in the final story, personal dejection and pre-occupation with an undefined lost object.

An inquiry into difficulties around the time when her baby was alive showed that she had found the child extremely demanding. Scores on a semantic differential scale (Osgood, Suci and Tannenbaum, 1957) suggested moderate reactions to the child's attention-seeking, but very disturbed reactions, especially on the Sensitivity and Depression scales to problems with neighbours, which appear to have been present before the death of the child.

Her husband contradicted himself on questions relating to difficulties with the baby, and denied that they were anything to do with his wife or outsiders. His responses to the semantic differential test were evasive. This is consonant with his high score on the L scale of the EPI where he gave a picture of an extremely stable, moderately extraverted person. On the Hostility Questionnaire he expressed an average amount of hostility, most of which was projected; there were no guilt feelings and practically no self-criticism. His IQ was 82.

Discussion

One view of the cause of anorexia nervosa is that it represents a rejection by the patient of her mother (Hobhouse, 1938; Ross, 1938). Bruch (1965) drew attention to these patients' 'pursuit of thinness' while Crisp (1967) claimed that primary anorexia nervosa is a disorder which has as its basis a phobia of normal adolescent weight. Crisp has also described how certain of his anorexia nervosa patients who have recovered and gone on to have children have

mentioned inability to feel affection for the child (Crisp, 1971).

Steel & Pollock (1968) have commented in their psychiatric study of parents of battered children that such parents have expectations of the child far beyond its chronological age. If no gratification of these expectations is received the parent responds with frustration and anger. In addition, the mother who does not receive adequate emotional support from her husband or parents and who has a difficult time with her pregnancy may feel the demands of child-rearing as overwhelming. If the child comes to represent someone to whom she herself expects to turn, she may look to the child for much of the understanding or support that is lacking in her present situation.

This case exemplifies the pattern. The patient was dependent on her husband who was unable to support her emotional needs, while her own parents disapproved of her marriage. It is possible that starving the younger girl may have been a substitute for battering her son, the passive behaviour in the first instance being a substitute for the more active in the past.

In any event the literature contains several references correlating failure to thrive and battering (Koel, 1969; Bullard *et al.*, 1967; Barbero & Shaheen, 1967). These studies have hypothesized that failure to thrive and battering of children are on a continuum. This case also illustrates this hypothesis.

Acknowledgments

We would like to thank Professor W. H. Trethowan for his helpful advice and criticism in preparing this paper.

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Postgraduate Medical Journal (June 1972) 48, 384–385.

Toxoplasma encephalitis in a raw steak eater

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Summary

A case of adult acquired toxoplasma encephalitis is described. The patient was in the habit of eating raw or very lightly cooked steak, and could have obtained her infection from this source.

Introduction

The vast majority of acquired infections with the protozoan parasite, *Toxoplasma gondii*, are symptomless and are recognized by finding serum antibodies (Beverley, 1969). The commonest clinical manifestation of acquired toxoplasmosis is a glandular-fever-like syndrome with a negative Paul Bunnell reaction (Siim, 1956). Other modes of presentation described include uveitis (Duke-Elder, Ashton & Brihaye-van Geertruyden, 1953), atypical pneumonia (Ludlam & Beattie, 1963), myositis (Chandar, Mair & Mair, 1968), myocarditis (Mullan, Henry & Beverley, 1968), and hepatitis (Vischer, Bernheim & Engelbrecht, 1967). Meningo-encephalitis, the most important feature of congenital toxoplasmosis (Nutt & Beverley, 1963) also occurs with the acquired disease; Sabin (1941), Sexton, Eyles & Dillman (1953) and Kayhoe *et al.* (1957) have reported severe cases which reached necropsy. However, since Sabin's original paper 30 years ago there have been few cases of toxoplasma encephalitis diagnosed during life (Beverley, 1969; Fleck, 1971). The following case is of particular interest in that the patient was fond of eating practically raw steak. This fact may be relevant to the as yet uncertain mode of transmission of the organism in man.

Case report

A 23-year-old married theatre sister was admitted to hospital in October 1970 as an emergency having become confused on her way to work. She had been

well until 8 days previously when she developed diarrhoea and vomiting which lasted 4 days. Forty-eight hours prior to admission she became drowsy, developed headache, photophobia and pain about the eyes and vomited. A history was obtained later that she ate extremely undercooked and, on occasions, even raw steak. She kept a budgerigar at home. There was no other history of contact with animals.

On examination: Temperature 99° F. Drowsy, irritable with photophobia and mild neck stiffness. Fundi normal. No neurological signs, lymphadenopathy or splenomegaly. General examination normal.

Investigations: Hb, 16.2 g/100 ml; WCC, 7700/mm³; lymphocytes, 52%; ESR 6 mm/hr. Cultures and microscopy of blood, urine, stool—normal. No viruses isolated from stool. CSF: normal pressure. WCC, 1/mm³; protein, 38 mg/100 ml; sugar, 50 mg/100 ml; WR and Lange, negative. No viruses grown. Glandular fever screening tests, negative. Extending Widal compatible with previous TAB—no rise in titre. Immunoglobulin levels—normal. Mantoux 1 in 10,000—positive. Serum glutamic oxaloacetic transaminase 76 Karmen units/min/ml. ECG—widespread T wave flattening. Serum bilirubin, alkaline phosphatase, chest X-ray, Reiter CFT and Kahn—all normal. Toxoplasma dye test titre—zero.

Progress. The patient's symptoms and low-grade irregular fever subsided spontaneously over 3 weeks and she was discharged to convalesce at home. Ten days later she was re-admitted with further headache, photophobia and fever, and a complaint of dropping things from her right hand. The physical signs were unchanged as were her blood count and CSF. The ECG and SGOT were now normal. An EEG was also normal. The toxoplasma dye test titre had now become positive at 1 in 2000. No toxoplasma were isolated from stool or CSF. A second serum showed no rise in titre or evidence of co-incidental infection with mumps, herpes simplex, lymphocytic choriomeningitis viruses, or leptospirosis.

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