anomalies than expected in the lithium babies, and cautioned about the teratogenic effects of lithium. However, they mentioned that the abnormal babies were more assiduously reported than the normal babies.

By record linkage of a discharge diagnosis registry and a medical birth registry, Källén & Tandberg (1983) identified 350 women with manic–depressive disease who had borne a child. The total delivery outcome was poorer than expected, with a high perinatal death rate and a high malformation rate among infants born of women who had used drugs in early pregnancy, and this phenomenon was concentrated in women who had used lithium.

Schou & Weinstein (quoted by Linden & Rich, 1983) collected 217 cases in which the mother had taken lithium during at least the first trimester of pregnancy. Of these, 183 babies were apparently normal at birth, 7 were stillborn, 2 had trisomy 21 (both mothers were in their late 30s) and 25 (11.5%) were malformed. Of the 25 malformed babies, 18 had anomalies of the cardiovascular system. Unlike their earlier stand (Schou *et al*, 1973), the conclusion of the lithium register workers was that lithium is likely to be teratogenic with respect to the cardiovascular system, and they suggested that lithium should not be used during the first trimester of pregnancy unless considered clinically essential.

In our patient, who did not have any past or present medical or gynaecological disorder, the still birth appears to be directly related to lithium intake during pregnancy. It is estimated that 70% of affective disorder patients will relapse within a year of discontinuing lithium, compared with 20% of those who are maintained on lithium (Linden & Rich, 1983). Our patient remained well while on lithium and relapsed soon after lithium was stopped. Some female patients will require the benefit of lithium without a break. Patients must be informed of the likely teratogenic risks of lithium, and its use must be avoided if possible at least during the first trimester of pregnancy.

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Suspected SSPE – a Delayed Psychiatric Presentation

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A case of suspected subacute sclerosing panencephalitis is described, presenting to a psychiatric hospital 39 years after a natural measles infection.

Subacute sclerosing panencephalitis (SSPE) is a very rare disease affecting 0.2 per million of the UK population (Kipps *et al*, 1983). It is a form of measles encephalitis with an average incubation period of seven years (ranging nine months to 18 years) from the measles infection (Bellman & Dick, 1980). The disorder largely affects children and adolescents, and is characterised by an insidious onset of mental deterioration, and later motor dysfunction, proceeding to coma and death, usually within a couple of years.

A greatly elevated titre of measles antibody may be discovered in the serum and cerebrospinal fluid, and characteristic electroencephalographic abnormalities consist of periodic complexes of high voltage occurring very regularly in each individual tracing (Wulff, 1982). Histological lesions may be widespread and variable, including perivascular small vessel cuffing by lymphocytes and plasma cells, proliferation of microglial cells and astrocytes, and intranuclear and intracytoplasmic inclusion bodies. In this report a middle-aged woman presents with a suspected functional psychiatric disorder.

Case report

Mrs E. G., a 44-year-old English woman, was admitted via a casualty department to St Mary's Hospital, Stennington, in an agitated, unkempt state (smeared with faeces), having collapsed in a shopping centre. She was feeling miserable, had not eaten for several days, and was incontinent of urine. She was fully conscious, orientated in person only, with very poor recent memory and a rather flat affect. Her behaviour was disinhibited and her emotions labile and often inappropriate. She had always been very dependent upon her husband (whom she claimed was still alive), and since his death seven years earlier (aged 63) she had neglected herself, her home and her family – four of her five children subsequently being taken into care. Psychiatric opinion (1982) was of an inadequate personality or schizophrenic defect state, while a psychologist reported her to be of normal intelligence and ability.

Her previous personal history was unremarkable, and her medical history limited to measles when five years old, and a tubal tie. She had no previous psychiatric history, although her mother had required hospital admission for a manic illness some years earlier. She did not drink alcohol and took no medication. She was pale and thin, with finger sclerodactyly and erythema ab igne. There were occasional semi-purposive movements of her upper limbs, and bizarre speech, which while normal in articulation, was monosyllabic and repetitious. She had a broad-based unsteady gait (tending to rock backwards) with Rombergism. There was increased tone in all limbs with full power, and pathologically brisk reflexes throughout, with bilateral extensor plantar responses and sustained bilateral ankle clonus. Routine blood tests and radiology were normal and she was transferred to the Royal Victoria Infirmary, Newcastle upon Tyne, for further investigation. Electroencephalographic recording showed a normal record of low potential, and a week later "moderate slowing of background rhythm together with some intermittent delta activity most evident in the right superior frontal region", suggesting a rather diffuse disturbance of cortical function. Autoantibodies and serum electrophoresis were normal, and CT scan showed cerebral atrophy only. Cerebrospinal fluid findings were 1 WBC/ml, total protein 0.46 g/l (with normal gammaglobulin subfraction), glucose 0.43 mmol/l, VDRL negative; viral screening revealed a 1/8 measles titre with a parallel blood titre of 1/320. Her agitation increased and incontinence persisted and she was transferred back to St Mary's heavily sedated and catheterised – an *E. coli* urinary tract infection persisted owing to multiple antibiotic allergies. Her condition gradually deteriorated, with frequent falls and subsequent confinement to bed. She was able to say her name and obey simple first order commands only, and she slept for much of the time. Physical examination remained unchanged, but she suffered a gastrointestinal bleed, manifest by a 4 g/dl drop in her haemoglobin, and positive occult blood stool tests. This proved to be self-limiting and was treated conservatively. One month later, nine weeks after her admission, she died.

Discussion

During the course of chronic encephalitidies, behavioural symptoms suggestive of functional psychiatric disorders are often observed. This lady was felt to be depressed when seen in casualty; the differential diagnoses widened on admission to include schizophrenic defect state, Korsakov's psychosis, and later Huntington's chorea and normal pressure hydrocephalus. Himmelhoch *et al* (1970) report eight adults with SSPE, all of whom displayed symptoms characteristic of functional psychiatric disorder, and seven of whom were originally diagnosed as suffering from depression, schizophrenia or hysteria. When the data were published (14 years after the first case presented), three patients had died, but five were still living, and three had enjoyed long periods of return to normal intellectual functioning. Could this lady have suffered a seven-year encephalitic illness after a 32-year latent period?

The diagnosis of SSPE is based on the clinical history, raised measles antibodies in serum and cerebrospinal fluid, electroencephalographic findings and brain histology. All the new cases reported to the UK register between 1977 and 1979 satisfied the first two criteria, "upon which a confident diagnosis can be made" (Bellman & Dick, 1980). Among the 47 cases, 39 showed at least one characteristic electroencephalogram: this lady did not, but she suffered a chronic urinary tract infection, and the disappearance of complexes might be explained by a rise in body temperature due to a febrile illness (Wulff, 1982). Brain histology was carried out in none of the above cases, while in this case it revealed discrete granular plaque lesions in the periventricular white matter of the occipital and parietal lobes. These showed myelin breakdown, with foamy macrophages and astrocytic gliosis. There was mild focal perivascular cuffing with mononuclear cells (predominantly plasma cells). No inclusion bodies were seen, and while the above appearances are consistent with SSPE, many of the features would also be consistent with multiple sclerosis during active

demyelination. In view of the atypical nature of this case, acute multiple sclerosis must therefore be considered as a differential diagnosis.

Addendum

Following the examination of additional material, it was noted that the cerebral cortex was not involved. The lesions in the periventricular white matter showed a predominance of focal demyelination with gliosis and inflammatory cell infiltration. The vessels within the lesions showed concentric fibrosis. Virus inclusion bodies were not identified and measles virus antigen was not demonstrated.

These findings are typical of acute demyelination in multiple sclerosis. In the absence of evidence of panencephalitis and with a predominance of demyelination over both neuronal degeneration and gliosis, this diagnosis must therefore be favoured.

This report typifies the dilemma that can arise with a psychiatric presentation of an atypical neurological illness, and illustrates the need (for statistical if not therapeutic reasons) to follow all available diagnostic pathways.

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