a mixed sample including acute and recent-onset cases. Carers of recent-onset sufferers are often caused considerable stress by the sufferer's positive psychotic symptoms. Carers of long-term sufferers seem also under stress, but more from the less obvious, but equally pernicious, problems presented by the unrewarding nature of their relative's behaviour.

The present sample of reliable attenders at an IMI clinic may be a selected subgroup, and present their families lesser problems than do clients who have a less stable contact with services. For example, they may have their medication more closely monitored, or have better social networks.

It seemed, from interviewing the carers, that families fell into two categories, those who were coping well with relatively few difficulties, and those who were struggling with a wide range of problems which were both related and unrelated to the patient. It proved difficult to confirm this from the data, but it may have been reflected in the GHQ scores, which seemed to be split in this way, with subjects scoring either below the threshold, or considerably above (17 subjects scored 3 or under on the screening score, the other seven all scored over 9).

One should not dismiss the importance of the finding that, although some carers are finding it difficult to cope, a substantial proportion manage to cope with the difficulties posed by their relative without suffering from psychological ill-effects. This is a more positive outcome than is sometimes implied in discussions about caring for sufferers of long-term mental illness.

Acknowledgements

The authors wish to acknowledge the considerable assistance received from Dr Carole Baughan, and from all the relatives who participated in the study. Comments from Dr I. Gwyndaf Pryce, Professor Ray Hodgson, and Miss Joy Abbati were also appreciated.

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Neurosyphilis and Schizophrenia

KANDIAH SIVAKUMAR and CHIKE I. OKOCHA

Neurosyphilis continues to present in atypical forms, leading to erroneous diagnoses by physicians and psychiatrists. This patient, with a previous history of psychosis, presented in a catatonic state with rhabdomyolysis and renal failure. A subsequent breakdown was thought to be schizophrenic until unusual features led to a reassessment and discovery of neurosyphilis which was treated with penicillin and resulted in a remarkable clinical recovery.

British Journal of Psychiatry (1992), **161**, 251–254

The diagnosis of neurosyphilis is often overlooked, not only because of its rarity but also because of its unusual and atypical patterns of presentation. In terms of disease manifestation, syphilis has always been a great masquerader. Lishman (1987) observes that "the psychiatrist must continue to bear it constantly in mind to check regularly with serological tests and look for cardinal signs in the pupillary reactions and tendon reflexes. The classical presentation of general paresis is nowadays rare and syphilis of the central nervous system can present with virtually any psychiatric complaint". We report a case diagnosed as schizophrenic until

https://doi.org/10.1192/bjp.161.2.251 Published online by Cambridge University Press

unusual features led to reassessment of the patient's physical status resulting in the discovery of unequal pupils, and subsequent serological testing confirmed a diagnosis of neurosyphilis.

We report this case because we feel that there are lessons of practical importance that can be learnt from it. Firstly, it emphasises the importance of a detailed appraisal of the physical status of every patient on admission, a matter that has caused some concern in recent years (Rigby & Oswald, 1987). Secondly, our patient presented with catatonic features and this helps to illustrate a further lesson which is that although the catatonic syndrome has a special relationship to schizophrenia, it can occur as a result of other causes and is often due to an organic state (Kellam, 1987). It is, therefore, imperative that whenever catatonic states are encountered, the search for an underlying organic cause should be thorough. Thirdly, the presence of a psychiatric history should in no way minimise the need for a comprehensive physical assessment on each medical admission.

Case report

A 46-year-old single homosexual man was referred to the psychiatric team in 1973 with vague disorganised thinking and first-rank Schneiderian symptoms. There was no record of his parents and he grew up in an orphanage. On leaving school, he joined the Army after a few years of unemployment and travelled to Hong Kong and Germany. He left after five years and got a job as a technician with a telephone company. He was made redundant after nine years and has since been unemployed. He was never married.

A diagnosis of schizophrenia was made, and oral and later depot antipsychotics started. He was lost to follow-up a couple of years later. In 1984, he came under our care referred with complaints of being difficult to cope with in the group home where he lived. He was leaving the gas cooker on and throwing burning cigarette ends, and was a fire risk. He had persecutory ideas of being chased by people he could not identify. His medication was reinstated and he was followed up in the out-patient clinic. In 1987, he was admitted twice, following attempts at reducing his medication.

In November of the same year, he was admitted to the District General Hospital in a collapsed state. It was discovered that he had stopped all his medication three weeks earlier. On admission, he was found to be unconscious but rousable, sweaty, hyperthermic and hypotensive. Carotid pulse was 140 beats per minute and other pulses were impalpable. He had circumscribed red patches on the dorsum of both calves and knees, hypertonia in all limbs and 'unequal' pupils. No mention was made of the responsiveness of the pupils to light or accommodation.

Blood investigations showed raised erythrocyte sedimentation rate, haemoglobin, haematocrit, total white cell count, urea and electrolytes.

On catheterisation, urine was dark in colour. A provisional diagnosis of acute renal failure with rhabdomyolysis was made and treatment with antibiotics and intravenous fluids given. He improved in a few days and was referred to the psychiatrists. At that time an examination of his mental state showed no evidence of psychosis. It was thought that following discontinuation of his medication, he had relapsed into a catatonic state and that the rhabdomyolysis was a consequence of prolonged immobilisation. The possibility of neuroleptic malignant syndrome was considered and so he was discharged medication-free to be monitored as an out-patient by the psychiatrist.

Two months later he was admitted to the psychiatric hospital. He was unkempt and dishevelled and looked physically unwell. He was very vague and complained of being chased by people. He had been picked up by the police wandering the streets twice the previous week. Attention and concentration were poor and so a full cognitive assessment could not be done. He was restarted on chlorpromazine 100 mg three times a day and flupenthixol 40 mg every two weeks. His mental state improved and he was discharged four weeks later.

He was readmitted to the psychiatric hospital six months later with the similar features of wandering about town trying to escape imagined persecutors, causing a small fire at home and hearing voices telling him to get undressed. Within the first week of admission he was disorientated, jumped out of windows for no apparent reason and when he managed to get away from the ward was found in dangerous situations such as the middle of busy roads obstructing traffic. When brought back he had no recollection of these events. He continued on flupenthixol 40 mg every two weeks, but chlorpromazine was increased to 100 mg four times a day and p.r.n., up to 400 mg a day. There was no improvement and he became more confused. Worried about his lack of progress we reassessed him for an organic disorder and the discovery of unequal pupils, with the left reacting only to accommodation but not to light, whereas the right pupil reacted to both light and accommodation, aroused suspicion of neurosyphilis, and serology and cerebrospinal fluid (CSF) tests were carried out.

Serology showed venereal disease research laboratory test (VDRL) positive with titre 8 and treponema pallidum haemagglutination (TPHA) positive with titre 2560 and fluorescent treponemal antibody (FTA) positive. CSF also showed VDRL positive, FTA positive and TPHA titre of 1/5260. White blood count (WBC) was 22, proteins 0.6 g/l and culture showed no bacterial growth. He was treated with procaine penicillin 600 mg i.m. twice daily for two weeks. He made remarkable improvement and it was possible to decrease and then stop medication.

At 2½-year follow-up he was free of both symptoms and neuroleptic medication, and repeat CSF testing showed a clear, colourless specimen with CSF protein of less than 0.2 g/l, glucose of 3.6 mmol/l and WBC of less than 1/mm^3. CSF VDRL was positive, TPHA positive 1/160, FTA – IgG positive, IgM negative, and Captia Elisa IgM negative. Repeat serology at the same time showed VDRL positive ¼, TPHA positive 1/2560, FTA – IgG positive and IgM negative.

https://doi.org/10.1192/bjp.161.2.251 Published online by Cambridge University Press

Psychometric tests at six months and two years after treatment showed no significant differences and the results are as follows: with the revised Wechsler Adult Intelligence Scale (Wechsler, 1981), a verbal–performance discrepancy was obtained suggesting a decline in verbal abilities from premorbid levels. Full-scale and performance IQs were in the low–average range (full scale IQ = 78, performance IQ = 88). His verbal IQ was significantly lower and in the borderline intelligence range (verbal IQ = 70). Tests of frontal lobe functions (word fluency, cognitive estimation and modified Wisconsin Card Sorting Tests (Nelson, 1976)) all showed decline, with word fluency tests suggesting left frontal damage. The computerised tomography scan showed no significant abnormality.

Discussion

General paresis can manifest itself in many forms and these have been classified on the basis of the salient feature in the mental state, e.g. grandiose form, depressive form, etc. The major manifestations can show a close resemblance to functional illnesses, so much so that the underlying disorder fails to be discovered.

Although not as frequently as the affective psychosis, there have been reports of clinical presentations leading to a diagnosis of schizophrenia (Dewhurst, 1969; Sirota *et al*, 1989). It is said that the clinical presentation can be so convincing that the CSF findings in these cases may come as a surprise (Froshaug & Ytreus, 1956). Bruetsch (1975) refers to a case described by Bumke where the patient had shown negativism and had been diagnosed as suffering from catatonic schizophrenia for a year in the pre-Wasserman days until he suddenly died of paralytic convulsions.

As the syphilitic process advances, clearer evidence of organic pathology, both physical as in taboparesis, and in mental state such as gross changes in personality, impaired emotional control, and intellectual decline, appears. Serological tests would clinch the diagnosis and remarkable improvement in the psychosis can be achieved by treatment with penicillin (Brooke *et al*, 1987). In our patient it seems unlikely that the 15-year history of psychosis could be wholly attributed to neurosyphilis. What appears to be more likely is that the psychotic illness was made worse by neurosyphilis. The episodes became more frequent, the response to medication was poor, and clouding of consciousness supervened.

Although altered consciousness and unequal pupils were noted at the time of admission in a collapsed state, the long psychiatric history, the discontinuation of medication and muscular rigidity suggestive of catatonia had clouded issues and led to the diagnosis of a relapse of schizophrenia. This resulted in the possibility of an organic condition such as neurosyphilis being overlooked.

It is possible that the rhabdomyolysis may have been due to a neuroleptic malignant syndrome and not catatonic schizophrenia. The findings of hyperthermia, tachycardia, hypotension, hypertonia and altered consciousness all point to this possibility. Depot injections with long half-lives may cause neuroleptic malignant syndrome weeks after discontinuation of medication.

The role of partial treatment of neurosyphilis with antibiotics, prescribed for other purposes by general practitioners and other specialists, in making presentations atypical must also be borne in mind. The usefulness of conducting serological tests on all patients admitted to psychiatric hospitals should be considered but in this day and age of financial stringency, carrying out tests on all patients may be called into question (Sharrar & Goldberg, 1988).

Selective screening may be more appropriate. O'Neil & McCaffrey (1989) recommend screening in five groups: those with a clinical picture of dementia; those with signs of an organic component to their illness at mental state examination; high-risk groups – prostitutes, those with a history of sexually transmitted diseases or promiscuity, and homosexuals; those with abnormal neurological signs; and those with atypical illnesses not responding to treatment.

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Risk for Definite Neuroleptic Malignant Syndrome

A Prospective Study in 223 Consecutive In-patients

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The occurrence of neuroleptic malignant syndrome (NMS) was studied prospectively in two series of consecutive psychiatric in-patients ($n = 223$). The first group ($n = 120$) suffered from schizophrenia and was treated only with haloperidol. The second group ($n = 103$) was treated with diverse neuroleptics. All patients were on a single antipsychotic agent with no anticholinergic drug as prophylaxis. The incidence of full NMS per admission and first neuroleptic exposure was 5/223 (2.2%). Patients with bipolar affective disorder and those treated with injections were significantly over-represented in the NMS group.

British Journal of Psychiatry (1992), **161**, 254–257

Neuroleptic malignant syndrome (NMS) has recently been attracting increasing attention, as shown by the rising number of publications annually on this issue (Kellam, 1987; Shalev *et al*, 1989). This interest is due to the alarming mortality rate which, in the past, was higher than 20% of NMS episodes (Shalev *et al*, 1989; Lazarus *et al*, 1989), and the awareness that this risky side-effect is not as rare as had been previously thought (Addonizio *et al*, 1986; Pope *et al*, 1986). However, even basic data about NMS are still scarce, including prevalence and incidence as well as figures concerning mortality rate and the risk per exposure to a neuroleptic. According to some studies, NMS is a very rare adverse side-effect occurring in 0.02–0.07% of neuroleptic-treated patients (e.g. Spiess-Kiefer *et al*, 1988). Based on his two-year survey of all cases of stupor in central South Wales, Kellam (1987) estimated "the annual incidence of NMS as one case per 1–5 million population, an incidence that would give an average psychiatrist about an even chance of seeing one case during his working lifetime". Most others reported an incidence of from 0.12% (e.g. Deng *et al*, 1990) to 0.4%. However, some studies (Addonizio *et al*, 1986; Pope *et al*, 1986; Keck *et al*, 1987) found an incidence closer to 1–2%, as did Delay *et al* (1960) in their early descriptions of NMS.

In order to estimate the incidence of NMS we conducted a prospective study, using standardised diagnostic criteria of NMS in newly admitted patients undergoing their first neuroleptic treatment.

Method

Criteria for full-blown NMS were those suggested by Levenson (1985), Lazarus *et al* (1989) and Pope *et al* (1986).

(a) Hyperthermia (axilla > 37.8 °C, per oral > 37.9 °C, per rectum > 38 °C). It is considered by all reviews and epidemiological studies as a critical sign. In the absence of elevated temperature the diagnosis of NMS would be inconclusive and would not be included in this study.

(b) Additional autonomic disturbances. At least two of the following: increased diastolic blood pressure above baseline of more than 20 mmHg, tachycardia of more than 30 beats/min above baseline pulse, prominent diaphoresis, incontinence, and tachypnoea.

(c) At least two of the following marked motor and extrapyramidal signs: hypertonus of either cogwheel or lead-pipe type, pronounced tremor, choreiform and dyskinetic movements, festinating gait and flexor–extensor posturing, prolonged retrocollis, opisthotonus, trismus, and considerable sialorrhoea.

(d) Clouded mentation (i.e. marked sedation, delirium, stupor, or coma).

https://doi.org/10.1192/bjp.161.2.251 Published online by Cambridge University Press