References

CAROTHERS, J. C. (1945) Miraa as a cause of Insanity. *The East African Medical Journal*, Jan. 4–6.

EDDY, N. B., HALBACH, H., ISBELL, H. & SEEVERS, M. H. (1965) In "Drug Dependence: its significance and characteristics" *Bulletin of the World Health Organisation*, 32, 721–733.

GIANNINI, A. & CASTELLANI, S. (1982) A manic-like psychosis due to Khat *Journal of Toxicology — Clinical Toxicology*, 19(5), 455–459.

HALBACH, H. (1972) Medical aspects of the chewing of Khat leaves. *Bulletin of the World Health Organisation*, 47, 21–29.

KALIX, P. (1984) The Pharmacology of Khat *General Pharmacology* 15 (no 3), 179–187.

LAURENT, J. M. (1962) Toxique et Tixocomonie peu connus, "Le Cath". *Annales Medico-Psychologiques* 120, 649–657.

UNITED NATIONS DOCUMENT (1975) Études sur la composition chimique du Khat; recherches sur la fraction phenylalkylamine. *MNAR*, No 11.

*Stephen Critchlow, MB, DCH, *Registrar in Psychiatry*; Ruth Seifert, MRCPsych, *Consultant Psychiatrist, Hackney Hospital*

*Correspondence: *Hackney Hospital, Homerton High Street, London E9 6BE*

(Accepted 4 June 1986)

British Journal of Psychiatry (1987), **150**, 249–250

Down's Syndrome with Mania

Sovner *et al* (1985) proposed that the presence of Down's Syndrome (DS) precludes the possibility of concomitant mania, and this is not a new claim, since Earl (1961) had stated that neither cyclothymia nor manic-depressive psychosis occurred in the mentally retarded. While Sovner *et al* conceded that cases of mania in individuals with DS may simply have gone unrecognised or unpublished, they felt this to be unlikely. However, lest this suggestion enter the canon, we present a previously unreported case of a young adult with both DS and recurrent mania.

Case report

A moderately mentally retarded, 23-year-old man was brought to a university psychiatric clinic by his mother because she was concerned about his increasing activity, increasingly grandiose ideas and decreased sleep. Down's Syndrome had been previously diagnosed on the basis of physical stigmata at examination, as well as trisomy 21 on chromosomal analysis. There was no family history of psychiatric illness. The patient had had a similar episode 2 years previously, characterised by increased hyperactivity, increased rate and volume of speech, and decreased appetite and sleep. Thioridazine treatment was instituted at that time, following which the patient became somewhat sedated; the symptoms had remitted within a month.

Two months before the most recent referral, the patient had once again shown increasing levels of activity and impulsivity and his mother had been forced to restrict him to the home because he had wandered off and almost got lost. His deteriorating behaviour was accompanied by a preoccupation with grandiose delusions, including that of his becoming a famous musician. He also became increasingly irritable, particularly when his grandiose ideas were challenged. Physiological changes included decreased appetite, a weight loss of 9 kg (20 lb), and decreased sleep. On each of the two nights immediately before being seen, he slept at most 2–3 hours, and was reportedly dancing and singing when awake, although it was in the middle of the night.

When seen, the patient was dressed like a certain pop singer, and demanded to be called by that singer's name, stating that the examiner was a television producer interviewing him for a job. The patient's affect was characterised by rapid swings from elation to irritation, when his mother did not respond to his grandiose statements, but when not challenged, he was most engaging. The examination revealed no evidence of hallucinations or mood-incongruent delusions. The physical, neurological, and laboratory examinations were all within normal limits, while a cardiology consultation revealed no change in the patient's asymptomatic ventricular septal defect with intraventricular and intra-atrial conduction defects.

The diagnosis was made of bipolar affective disorder, manic with mood-congruent psychotic features (DSM-III). This diagnosis was later confirmed independently by two attending psychiatrists.

https://doi.org/10.1192/bjp.150.2.249 Published online by Cambridge University Press

Discussion

Mania and DS can and do occur concomitantly, as this case illustrates; there is a strong suggestion that ours is not the first report of this coincidence. Although not designated as cases of 'mania' (perhaps because of the then-prevailing diagnostic criteria), Rollin (1946) described a behaviour change, frequent among 'mongols', in which patients underwent

> a period of excitement amounting in some cases to an almost manic phase. They became wild, noisy and restless, talking incessantly, emitting weird animal noises or inarticulate gibberish. This phase passes after a variable interval, only to recur without any obvious cause.

Information in Rollin's three case vignettes is not sufficient to diagnose mania retrospectively, but it is of interest that one patient was noted to have become irritable and 'talked incessantly all day'.

Although unipolar mania and bipolar affective disorder may be less prevalent than unipolar depression in individuals with DS, this case shows that the two disorders are not mutually exclusive. However, much remains unknown about the incidence and prevalence rates of psychiatric disorders in DS, as well as in other types of mental retardation. If the prevalence rate of bipolar illness in DS is indeed less than in control populations, interesting possibilities are posed for further investigation of genetic predisposition to and protection from psychiatric illness.

Sovner *et al* (1985) have suggested an explanatory hypothesis for mania being incompatible with DS—that alterations in monoamine activity inhibit the development of mania—but present understanding of the pathophysiology of both conditions is too limited to support the contention of mutual exclusiveness.

In fact a case can even be made for a heightened association of DS and bipolar affective illness. Prange *et al* (1974) hypothesised that low indoleamine activity 'permits' development of mania, when it is present with elevated catecholamine activity. In subjects with DS, blood 5-hydroxyindole levels (Rosner *et al*, 1965; Tu & Zellwegger, 1965) and serotonin uptake into platelets (Boullin & O'Brien, 1971) have been reported to be decreased, compared with controls. According to Prange's hypothesis, this should lead to an *increase* in the incidence of mania in individuals with DS, but as yet, too little is known of the biology of both DS and mania: there are still contradictory data. For example, CSF 5-hydroxyindoleacetic acid levels in patients with DS have been found *not* to differ from those of neurological controls (Lott *et al*, 1972, Tu & Partington, 1972).

Epidemiological studies of psychiatric illness among the mentally retarded have been only limited; as a whole, they suggest that such illness is at least as prevalent as in the general population (Parsons *et al*, 1984). Psychiatric diagnosis in the mentally retarded illustrates the importance of diagnosing each case on the clinical data, rather than on theories of pathogenesis (Sovner & Hurley, 1983).

References

Boullin, D. J. & O'Brien, R. A. (1971) Abnormalities of 5-hydroxytrytamine uptake and binding by blood platelets from children. *Journal of Physiology*, **212**, 287–297.

Earl, C. J. C. (1961) *Subnormal Personalities*. Baltimore: Williams and Wilkins.

Lott, I. T., Murphy, D. L. & Chase, T. N. (1972) Down's syndrome—central monoamine turnover in patients with diminished serotonin. *Neurology*, **22**, 967–972.

Parsons, J. A., May, J. G. & Menolascino, F. J. (1984) The nature and incidence of mental illness in mentally retarded individuals. In *Handbook of Mental Illness in the Mentally Retarded* (eds F. J. Menolascino & J. A. Stark), London: Plenum.

Prange, A. J., Wilson, I. C., Lynn, C. W. *et al* (1974) L-Tryptophan in mania. *Archives of General Psychiatry*, **30**, 56–62.

Rollin, H. R. (1946) Personality in mongolism with special reference to the incidence of catatonic psychosis. *American Journal of Mental Deficiency*, **51**, 219–237.

Rosner, F., Ong, B. H., Paine, R. S. & Mahanand, D. (1965) Blood-serotonin activity in trisomic and translocation Down's syndrome. *Lancet*, **i**, 1191–1193.

Sovner, R. & Hurley, A. D. (1983) Do the mentally retarded suffer from affective illness? *Archives of General Psychiatry*, **40**, 61–67.

——, —— & Labrie, R. (1985) Is mania incompatible with Down's syndrome? *British Journal of Psychiatry*, **146**, 319–320.

Tu, J. & Partington, M. W. (1972) 5-Hydroxyindole levels in the blood and CSF in Down's syndrome, phenylketonuria and severe mental retardation. *Developmental Medicine and Child Neurology*, **14**, 457–466.

—— & Zellweger, H. (1965) Blood-serotonin deficiency in Down's syndrome. *Lancet*, **ii**, 715–717.

*Edwin H. Cook, Jr., MD. *Chief Fellow, Child Psychiatry*; Bennett L. Leventhal, MD, *Associate Professor of Psychiatry & Pediatrics, Director, Child Psychiatry, Associate Chairman, Department of Psychiatry, University of Chicago*

*Correspondence: *University of Chicago, 5841 S. Maryland (Box 411) Chicago, Illinois 60637, USA*

(*Accepted 12 November 1985*)

https://doi.org/10.1192/bjp.150.2.249 Published online by Cambridge University Press