valuable baseline. Combinations of different classes of antipsychotic drugs are very rarely, if ever, justified (Davis, 1985). Combinations cause difficulty in determining exactly which drug provoked the reaction. All drugs given at the time of the reaction tend to be avoided in future, possibly unnecessarily. In the two cases reported, agranulocytosis developed after a butyrophenone was added to treatment with phenothiazines. It remains to be seen whether this association will prove to be spurious or causal.

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Cholinergic Syndrome following Anticholinergic Withdrawal in a Schizophrenic Patient Abusing Marijuana

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A 27-year-old neuroleptic-stabilised schizophrenic patient presented with a three-day history of psychomotor retardation, disturbed sleep, and social and emotional withdrawal following reduction of his anticholinergic dosage; his symptoms had intensified after an increase in neuroleptic dosage, based on a diagnosis of psychotic decompensation. Recognition of a cholinergic syndrome and institution of appropriate anticholinergic treatment resulted in rapid improvement. The clinical distinction between a cholinergic overdrive state and schizophrenic exacerbation, while sometimes difficult, can be critical in selecting appropriate management.

The recognition of withdrawal reactions following discontinuation of psychotropic medications with anticholinergic properties is important in the treatment of various psychiatric disorders. Rapid withdrawal especially may result in a clinical syndrome characterised by bradykinesia, motor retardation, slowed thinking and speech production, lassitude, sleep disruptions, nightmares, emotional withdrawal, irritability, dysphoria, nausea, vomiting and diarrhoea (Dilsaver *et al*, 1984*a*). The mechanism is believed to be central and peripheral, cholinergic hyperfunction secondary to regulatory changes in muscarinic receptors occurring as a result of sustained anticholinergic blockade (Dilsaver *et al*, 1984*a*; Gardos *et al*, 1978). Patients who use marijuana may be at a higher risk for development of this syndrome (El-Yousef *et al*, 1973).

The anticholinergic withdrawal syndrome resembles the negative schizophrenic syndrome in many important respects (Crow, 1980*a*,*b*; Andreasen, 1982; Greden *et al*, 1987). Its occurrence in a schizophrenic patient may be mistaken for psychotic decompensation and result in inappropriate treatment. We report the syndrome in a marijuana-abusing schizophrenic patient.

Case report

Mr A, a 27-year-old male janitor with a two-year history of paranoid schizophrenia and a several-year history of cannabis abuse, was brought to the University of Michigan Medical Center psychiatric emergency room by his wife, because of a three-day history of increasing confusion, sluggishness, inability to work, feeling "frozen", irritability, disturbed sleep with nightmares, social and emotional withdrawal, and inability to perform the activities of daily living without assistance. There was no current evidence of delusions or hallucinations, suspiciousness, or ideas of reference. The patient had been in stable remission, with good occupational and fair social functioning on treatment with thiothixene (30 mg per day), imipramine (150 mg per day), and benztropine (4 mg per day). Two days before the appearance of the above symptoms, however, his out-patient psychiatrist had reduced the dose of benztropine to 1 mg per day because of concerns about overuse. The emergency-room psychiatrist diagnosed a psychotic exacerbation, abruptly discontinued the imipramine, and replaced the thiothixene with 30 mg of haloperidol per day, thereby doubling the effective neuroleptic dosage. The patient's symptoms progressively worsened, and he was admitted to the University of Michigan Schizophrenia Program two days later. The patient had last smoked marijuana one week before admission.

On examination, he was reasonably well groomed, sat slumped in his chair and displayed severe retardation of bodily movement and speech, gave mere yes or no answers to all questions after a prolonged latency, and reported a marked slowing of thought processes. His face was immobile and his affect was flat; despite his psychomotor deceleration, he was irritable and jittery. He had no involuntary movements, restlessness or rigidity, and he denied experiencing delusions, hallucinations, ideas of reference, thought control or other Schneiderian first-rank symptoms. He also reported the absence of depression or suicidal ideation. Formal testing of his cognitive functions was difficult, but he appeared orientated with respect to time and place. His vital functions were stable and no physical abnormalities were detected.

After reviewing all information, we formulated a tentative differential diagnosis of cholinergic syndrome versus akinesia. We discontinued haloperidol and restarted treatment with thiothixene at the previous dosage of 30 mg per day. The dosage of benztropine was increased to 2 mg p.o. t.d.s. and 2 mg orally as needed, not to exceed 10 mg per day. His symptoms remitted rapidly. Within the next few days, the patient became relaxed and verbal, his affect brightened and he became more expressive, irritability decreased and his speech and thought returned to normal. One week after admission, he had returned to baseline according to him and his wife. Despite the fairly high dosage of benztropine, he remained afebrile, his pupils were equal and reactive, and his pulse did not increase by more than 10 beats per min and was always regular. At no time did he report any psychotic or depressive symptoms. He was observed for another week and discharged on treatment with thiothixene 30 mg per day, and benztropine 2 mg p.o. t.d.s. He has returned to work, is asymptomatic and is functioning normally on this regime one month later.

Discussion

Several factors may have contributed to a state of cholinergic overdrive in this patient. The dosage of benztropine, an anticholinergic agent, was abruptly decreased in a patient who may have already been susceptible to the development of a cholinergic syndrome because of chronic marijuana abuse (Dilsaver *et al*, 1984*b*). We believe that symptoms of a cholinergic syndrome resulted and these intensified even more following abrupt discontinuation of imipramine (with moderate anticholinergic activity), and substitution of haloperidol for thiothixene. The symptoms resolved rapidly with anticholinergic 'replacement' treatment. While some authors may suggest that a diagnosis of akinesia may have been in order (Rifkin *et al*, 1975), akinesia is an extrapyramidal syndrome that can result from dopaminergic underactivity or cholinergic overactivity. Akinesia is a behavioural diagnosis and cholinergic overdrive the probable underlying pathogenetic mechanism in this case, and the two terms may thus reflect different aspects of the same phenomenon (Tandon & Greden, 1987; Tandon *et al*, 1988).

The case highlights the importance of careful differentiation between a cholinergic overdrive state and exacerbation of the schizophrenic illness. This differentiation is especially difficult when patients present with predominantly negative symptoms, as was observed in this case. Distinguishing between a cholinergic syndrome produced by drug withdrawal and psychotic decompensation is sometimes difficult, but can be critical in selecting appropriate management. A careful history, specifically noting recent medication changes, can often point to the correct diagnosis. Clinicians should be sensitive to the possibility of development of cholinergic overdrive in a schizophrenic patient due to a variety of mechanisms. Although it is sometimes difficult to distinguish from schizophrenic exacerbation, the cholinergic syndrome responds rapidly to aggressive anticholinergic treatment when accurately diagnosed. The case also points out one of the risks of cannabis consumption in schizophrenic patients (Treffert, 1978).

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De Clérambault's Syndrome (Erotomania) in Organic Delusional Syndrome

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A patient with de Clérambault's syndrome (erotomania) following brain damage and epilepsy is described. The delusion appeared after brain surgery for subarachnoid haemorrhage, and remained chronic. Erotomania in this patient may be judged to be aetiologically related to organic brain damage.

De Clérambault, in the 1920s, described the most well-known of the 'erotic delusions' as "pure erotomania". This syndrome is characterised by the 'fundamental postulate' of a woman having the conviction of being in amorous communication with a person of higher rank, whom she claims was the first to fall in love (Enoch & Trethowan, 1979). The delusion becomes chronic, and secondary or mixed forms can occur.

Erotomania has so far defied precise psychiatric classification. Although de Clérambault delineated a pure form of the syndrome, most modern writers tend to consider it as related to one of the major psychoses, i.e. schizophrenia or the affective disorders. The nosological controversy may in part be due to the poor description of pre-morbid history, personality and phenomenology. The revised DSM-III (DSM-III-R; American Psychiatric Association, 1988) classifies it as a specific type of delusional disorder.

Until recently, few cases of erotomania have been reported where there was an association with organic pathology. I present a case of erotomania in a patient with organic delusional syndrome.

Case report

Miss K., a 29-year-old right-handed single female, was found on referral to have erotic and paranoid delusions. She believed that she was deeply in love with a man who worked as an estate agent, from whom she rented a flat. She also believed that he was deeply in love with her and wanted to marry her, but was prevented from doing so because of pressure from "the DHSS and others". She pestered him with letters and telephone calls to such an extent that he had to move to another city, but she managed to find him. The man strongly denied any involvement with her, apart from the time he rented her the flat. She also believed that people were spying on her and trying to kill her.