Catatonic syndrome related to acute disseminated encephalomyelitis (ADEM)

Dear Editors,

Although it is widely accepted that inflammatory CNS disease can cause schizophrenia-like symptoms (Davison, 1983), there is no evidence in the literature of co-occurrence of acute disseminated encephalitis (ADEM) and psychosis—as has been reported in multiple sclerosis (MS). We would like to draw attention to a case where both a catatonic syndrome and ADEM occurred. A 25-year-old female law student without family history of psychiatric disease was hospitalized for fever without bacteremia, motor aphasia, disorganized thinking, visual hallucinations and psychomotor agitation. Despite antipsychotic treatment, symptoms progressed to a catatonic state with mutism, stereotypy, rigidity, catatonic stupor with intermittent excitement and waxy flexibility. Extensive neurological examinations revealed traces of oligoclonal bands and a few small white matter defects on MRI only. These findings were considered to be unspecific, a diagnosis of catatonic schizophrenia was made, and olanzapine lead to remission. About 2 years later, after reduction of olanzapine to 5mg/day, the patient presented with sleep disturbance, restlessness and generalized anxiety. Despite dosage increase to olanzapine 20mg/day and additional administration of benperidole strong agitation and suicidal ideation arose. Eventually, a full-blown catatonic syndrome developed and a seizure stroke the left arm and the face. Cranial MRI showed white matter lesions of the right superior and medial temporal gyri, the right centrum semiovale and the left internal capsule; gadolinium enhancement was present in the temporal lesions. Oligoclonal bands were positive, CSF virologic examinations negative, and evoked potentials of the optic nerve were abnormal bilaterally. At this time, an ADEM was diagnosed and treated respectively with a 5-day course of methylprednisolone. Thereafter, clozapine and the antiepileptic carbamazepine were administered. Symptoms improved slowly but steadily; after remission of catatonia, acoustic hallucinations were still present for several weeks. The patient was discharged after 8 months in complete remission. Five years later, she agreed to a follow-up examination. She had been well throughout, seeing both a psychiatrist and a neurologist regularly. Antiepileptic medication had been stopped but antipsychotic medication continued (risperidone 3.5mg/day). She worked full-time as a lawyer and led a full life. Psychiatric examination was normal. However, neurological soft signs (Schröder et al., 1992) were in the pathological range, accounted for by poor performance of motor coordination tasks with increased difficulties on the left side. MRI findings revealed small spotted white matter glioses where the plaques had previously been described, consistent with the residual stage of a subsided inflammatory disease.

In general, organic diseases affecting the temporal lobe are known to cause psychotic symptoms (Flor-Henry, 1961). Although there is evidence in the literature for the co-occurrence of psychosis—including catatonia—and MS (Davison, 1983), an association of catatonia with ADEM has not been reported so far. The above demyelinating diseases can be differentiated only to a limited extent, some authors even argue that there is no useful diagnostic criterion for distinction (Schwarz et al., 2001). ADEM usually is a potentially fatal monophasic illness with a good prognosis if treated timely. It is typically preceded by an infection (as was present in this patient) or vaccination. It produces widespread CNS disturbance with drowsiness, coma, seizures and multifocal neurological signs. MS is by definition multiphasic, mostly presents as a monosymptomatic syndrome and frequently results in stepwise or steadily progressive neurologic deterioration. MRI demonstrates white matter pathology, a pattern of multifocal asymmetric lesions can occur in both MS and ADEM but serial MRI scans typically reveal new lesions in MS only (Kesselring et al., 1990; Lindsey and Wolinsky, 1999). The MRI criterion and the remitting clinical course best distinguish ADEM from MS. Although oligoclonal bands are a characteristic feature of MS, they may occur in ADEM. Optic neuritis can
also be found in both conditions, however, bilaterally in ADEM and unilaterally in MS.

Given the indicated features of MS and ADEM, we argue that our patient suffered from ADEM: although two catatonic episode occurred and oligoclonal bands were positive, she first presented with an infection, signs of bilateral optical neuritis were present, MRI lesions resolved to scar tissue and remained stable over the course of 5 years, and most importantly her clinical symptoms recovered except for minor neurological findings and she was fully functioning in all areas of life on follow-up. The interval after which the definitive diagnosis of ADEM can be made is uncertain, suggestions range from 1 month (Poser et al., 1983) to 2 years (Miller and Evans, 1953) without relapse—this time-criterion of ADEM was certainly fulfilled in the presented case. Concerning the biphasic course, it has been reported that episodes of CNS disturbance rarely recur after a typical attack of ADEM (Miller and Evans, 1953; Alcock and Hoffman, 1962). In this case, antipsychotics may have exerted an antiinflammatory effect as an antiinflammatory potential of antipsychotics has been shown in vitro (Jones-Brando et al., 2003).

We conclude that ADEM as well as MS may mimic schizophrenia and that atypical symptoms and courses in schizophrenia-like psychoses require intensive neurological work-up and specific treatment where necessary.

Acknowledgement

The authors acknowledge the advice of Klaus Kirchhoff, MD Department of Neuroradiology, INF 400, 69120 Heidelberg, Germany, and thank Aoife Hunt, MD for proofreading the manuscript.

References


Silke Bachmann
Department of Psychiatry and Psychotherapy,
University of Halle, Julius-Kühn-Str. 7,
06097 Halle, Germany
E-mail address: silke.bachmann@medizin.uni-halle.de.
Corresponding author. Tel.: +49 345 5573624;
fax: +49 345 5573500.

Johannes Schröder
Department of Psychiatry, University of Heidelberg,
Vossstr. 4, 69115 Heidelberg, Germany

16 March 2006