

A catatonic syndrome in a postpartum major depressive episode with psychotic features

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Summary

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It has been recognised that catatonic symptoms can be associated not only with schizophrenia but also with affective disorders. Catatonic syndromes during postpartum affective episodes have rarely been described. We report a female patient suffering from recurrent depression with a diagnosis of postpartum major depressive episode with psychotic features who developed a catatonic syndrome. Her family history is positive for recurrent depressive disorder. She reported that the severe psychomotor retardation and mutism had a strong negative impact on her ability to care for her child. The administration of intravenous lorazepam led to a rapid improvement in the catatonic syndrome. Our case highlights the need for clinicians to be on the watch for the emergence of a catatonic syndrome, not only in cases of psychosis but also in cases of affective disorders, especially during the postpartum period.

Keywords: catatonic syndrome; recurrent major depressive disorder; postpartum onset; lorazepam

Introduction

Since Kahlbaum's work [1] at the end of the nineteenth century it has been recognised that catatonic symptoms can be associated not only with schizophrenia but also with affective disorders. During the first half of the 20th century, probably

under the influence of Bleuler [2] and Schneider [3] who considered catatonia only as a subtype of schizophrenia, the nosological system excluded catatonia from affective disorders [4]. More recently, increased interest has been shown in catatonic symptoms appearing in affective disorders. Catatonic syndromes during postpartum affective episodes have rarely been described. To our knowledge, Hanson and Brown [5] reported one case and Lai and Huang [6] have very recently described three cases.

In this report we describe the case of a young woman with a recurrent depressive disorder who presented a severe major depressive episode with psychotic features and a catatonic syndrome during the postpartum period. The patient improved dramatically after lorazepam perfusions.

Case report

A 32-year-old woman with a DSM-IV diagnosis of recurrent major depressive disorder experienced her first depressive episode at the age of 18 in the context of an entrance examination to a business school. She remitted completely from this mild episode with a psychotherapy treatment and received no medication at that time. At the age of 31, she experienced a miscarriage that triggered a second mild to severe depressive episode. She remitted completely from this episode without any pharmacological or psychotherapeutic treatment. These data are reported by the patient and her siblings.

Her family history is positive for recurrent depressive disorder but is negative for other psychiatric diseases.

She is married, employed in an insurance company and comes from an upper-class background.

She gave birth five weeks prior to her admission to our clinic. The course of pregnancy and delivery was normal, and she did not present any psychiatric symptoms at that time. She began to feel sad and useless to her child about two weeks after giving birth. She experienced difficulties

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breast-feeding and developed intense feelings of guilt, believing herself to be a bad mother incapable of caring for her child or breast-feeding him. In this context she contacted a psychiatrist who admitted her with her baby to a psychiatric unit in the general hospital due to a diagnosis of postpartum depression with suicidal ideations.

At admission she was sad and anhedonic with intense feelings of guilt linked to her belief that she was a poor mother and suicidal ideations. At the same time, she presented intense negativism about herself, especially her role as a mother, and psychomotor retardation. Renal and hepatic functions were normal as ionogram and thyroid function. No EEG or MRI scans were performed.

She spent two months in an open unit. The depressive symptoms worsened with intensification of suicidal ideations despite two antidepressant trials (sertraline 50 mg/d for 2 weeks followed by venlafaxine 150 mg/d for 5 weeks). The low dosages employed in both trials were prescribed due to her difficulty in accepting the idea of an antidepressant treatment. In parallel with both antidepressant treatments an anxiolytic (alprazolam 0.5 mg/d) was prescribed. After three weeks of hospitalisation she began to have delusional thoughts leading her to believe that it would be better for her child to kill him and herself than to live with such a poor, deficient mother. A treatment of amisulpiride 200 mg/d was prescribed.

Concerning the catatonic syndrome, her motor retardation worsened, almost becoming a state of stupor. She spent the majority of her time lying in bed, unable to care for herself. She communicated with great difficulty, her negativism worsened, and mutism began to appear. It became physically difficult for her to hold her child and to play with him, especially, as she told us after recovery from the catatonic syndrome, because of the severe psychomotor retardation and the mutism. At this stage, the child psychiatrist observed signs of depression in the infant.

During a weekend release from the hospital she tried to commit suicide at home by hanging herself.

As a result, she was transferred to a closed unit. At the time of entry she presented a severe depressive episode with psychotic features and a catatonic syndrome marked by mutism. It had become very difficult for her to form a complete sentence, and she presented stupor, severe psychomotor retardation and negativism.

We augmented the venlafaxine treatment to 300 mg/d for the depressive episode and switched the neuroleptic from amisulpiride 200 mg/d to quetiapine 600 mg/d due to the side effects she experienced (mammary tension).

Four days after admission, we proposed treating her catatonic syndrome with lorazepam perfusions. She received three 20-minute perfusions of lorazepam 4 mg in 250 cc of a saline solution over a period of four days. Immediately after the first perfusion about 50% of the catatonic syndrome disappeared. The next two perfusions were followed by the complete disappearance of all catatonic symptoms.

Her ability to care for her child improved and she recovered her maternal feelings.

The depressive symptoms progressively diminished during the next three weeks and she was discharged from the hospital to an outpatient clinic.

Discussion

The patient suffered, according to the DSM IV [7], from a major depressive episode with psychotic features with a postpartum onset appearing rapidly after childbirth (within two weeks). Although she had a personal and family history of affective disorder, the possibility that rapidly changing hormonal levels after delivery may have acted as a trigger for the development of a major depressive episode cannot be excluded in this context, especially with a postpartum onset [8]. The important role played by psychosocial variables (i.e. adjustment to child-care tasks) during this period in the development of a major depressive episode is also widely recognised [9].

For our patient a catatonic syndrome clearly developed insidiously during this severe depressive episode with psychotic features. Catatonia is defined as a syndrome in which at least two of its signs are present for a day or longer [10–12]. The principal signs of catatonia are mutism, stupor, negativism, posturing, waxy flexibility, stereotypy, automatic obedience, ambitendency, echophenomena and mannerisms. About 6 to 9% of patients admitted to academic psychiatric hospital units meet these criteria. For some patients these symptoms are transient, but they persist for others (like our patient) and can even worsen, leading to malignant catatonia, a potentially lethal condition [13]. The syndrome often has an acute onset. For our patient the syndrome seems to have developed gradually over the first two months of hospitalisation. Due to the potentially lethal evolution as well as the availability of effective treatment, a rapid diagnosis seems very important.

Benzodiazepine, and in particular intravenous lorazepam, alleviates the syndrome in more than 80% of the patients treated [14]. When this treatment has failed, convulsive therapy can be

considered. For our patient the introduction of intravenous lorazepam quickly alleviated her symptoms. To our knowledge, however, the scientific literature is unclear about the length and dosage of lorazepam administration.

The aetiology of catatonia remains controversial in the literature [15]. Catatonia and neuroleptic malignant syndrome are clinically divergent entities, but they are probably best viewed as diverse manifestations of a single syndrome. The neurobiological hypothesis suggests an imbalance in different neurotransmitter interactions: GABA hypoactivity at GABA-A receptors, dopamine hypoactivity at D₂ receptors, serotonin hyperactivity at 5-HT_{1A} receptors, serotonin hypoactivity at 5-HT_{2A} receptors and glutamate hypoactivity at NMDA receptors.

To our knowledge, no hypotheses have been made linking the neurobiology of affective disorders and that of catatonic syndromes. Although no specific literature exists on this topic, the patient reported that her catatonic syndrome negatively affected the development of the mother–child relationship. She reported that the severe psychomotor retardation and mutism, which dramatically responded to the lorazepam medication, had a strong negative impact on her ability to care for her child. On the other hand, the impact of depression on the development of the mother–child relationship has been extensively studied [16].

Our case, as well as the four others treated with lorazepam reported in the literature [5, 6], highlights the need for clinicians to be on the watch for the emergence of a catatonic syndrome, not only in cases of psychosis but also in cases of affective disorders, especially during the postpartum period.

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