Preeclampsia masquerading as catatonic relapse in Schizophrenia, a case report

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Abstract

Preeclampsia and eclampsia can have neuropsychiatric manifestations due to acute brain injury which has been well recognized. There is developing evidence for association between preeclampsia / eclampsia and mental health problems. Psychiatric disorders and preeclampsia / eclampsia are commonly seen clinical problems during pregnancy, and untreated cases can have serious outcomes.

We present a case of a patient who had developed catatonic features for the first time in the late perinatal period. Her presentation was accompanied by new onset hypertension and proteinuria. This case highlights the importance of careful monitoring during the prenatal period of women with psychotic mental illness and the necessity and importance of communication between psychiatry and other medical specialties as well as the unique medical and psychiatric complications they face. Confounding factors such as mutism and a psychotic appearance may further complicate the clinical picture in such cases.

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Case Report

Mrs. X , a 22 -year- old primipara Omani lady originally from Tanzania, a diagnosed case of Schizophrenia according to DSM 5 and ICD 10 since 2 years. There was no medical history of the patient apart from sickle-cell trait, no surgical history, and she has good socioeconomic status. There is family history of mental illness in a brother diagnosed with Schizophrenia followed up at a tertiary mental health hospital, on active maintenance treatment and currently in remission.

She was off antipsychotic treatment since she became pregnant about 9 months back and remained stable from her first trimester of pregnancy until she was about to deliver her baby. Her glucose tolerance test was reported to be normal (4.2/3.8 mmol/L) in the second and third trimesters subsequently. Urine protein level was tested repeatedly and it was only trace (normal level in pregnancy <0.3g/L). Her blood pressure ranged from 111/57 mmHg earlier in the pregnancy, to a maximum of 146/98 mmHg towards the later stages of the pregnancy, when she had proteins in her urine consistently.

At 38 weeks gestation, her weight was 68 Kg, BMI 24.4 Kg/m2, and blood pressure 179/113 mmHg. Investigations identified normal serum B12 and folate levels, hemoglobin 12g/dl (normal 11.0- 14.5 g/dl). HIV and VDRL were negative. At 39 weeks of gestation Mrs X was seen at the obstetric outpatient clinic and identified as a high-risk pregnancy due to decreased fetal movement. She was planned for induction of labor but the patient refused and left against medical advice.

Two days later, she was brought by her mother and sister to the emergency department with 1 day history of change in behavior, refusing to eat, with poor sleep and poor hygiene. Further history taken from patient's mother revealed that the patient started to have disturbed behavior, irritable, not

cooperative, refusing food and drinks. She was not talking much and sometimes keeps mute. She expressed herself as fearful and terrified with no reasons. When she was seen in ED, she was avoiding eye contact, refused to talk, restrictive to being moved and showed marked rigidity. There was no history suggestive of hallucinatory behavior or suspiciousness.

Patient was admitted in psychiatry ward with provisional diagnosis of psychotic relapse of schizophrenia because of the catatonic symptoms. Mrs. X was kept under close monitoring and observation with no regular medications, only Haloperidol and Promethazine as needed. She was very difficult to move for CTG as a result of posturing and being mute.

At day two of admission, patient developed high blood pressure and vomited 3 times; around 1 liter of coffee ground vomitus mixed with fresh blood along with unresponsiveness due to reduced level of consciousness and posturing behaviors, soon after with dropping saturation. She was transferred to the emergency unit for immediate resuscitation as advised by obstetric team ,then was transferred to the labor room for further monitoring where she had 2 more episodes of vomiting before a delivery proceeded by emergency lower caesarian section with no complications. She delivered a healthy baby girl with APGAR scores of 9 and 10 consecutively. Later an obstetric diagnosis of preclampsia was confirmed with BP 179/112mmHg, oxygen saturation drop to 80%, urine Protein/creatinine of 58.12 mg/mmol (abnormal >45mg/mmol), urine protein 1.11 g/L (normal 0.00-0.15 g/L), LDH 276 U/L (normal 135-214U/L), and uric acid 0.45 mmol/L(normal 0.15-0.35 mmol/L).

After delivery, Mrs X became comfortable, conscious, oriented, communicating well, with good eye contact and no psychomotor agitation or retardation. Her mood was euthymic and her affect reactive. She revealed that she is happy to have the baby and that she breastfed her with warm feeling. She denied hearing voices or seeing abnormal things and she denied any abnormal thoughts or suicidal and homicidal thoughts. She couldn't remember what happened before delivery. She added that since 4 months she had been having occasional disturbance in sleep but was emotionally stable with no disturbed behavior or thoughts. A full neurological examination revealed no focal or generalized abnormalities.

During her hospital stay, Mrs X was monitored for changes in psychopathology but remained asymptomatic thereafter delivery and cared for her infant adequately, discharged after four days and advised to follow with parent hospital and to seek immediate medical or psychiatric attention if any signs or symptoms appear.

Discussion

This is, to our knowledge, the first case in our institution, reported of preeclampsia manifested with catatonic symptoms masquerading as a psychotic relapse in a patient with Schizophrenia, and posed an interesting diagnostic challenge. The patient clearly had a short period of catatonic experiences and behaviors with disturbed sleep and activities of daily living. There were accompanying physical symptoms like high blood pressure, proteinuria and vomiting as well as changes in consciousness, indicating that the catatonic syndrome was of organic cause rather than of functional etiology. The provisional diagnosis of the psychiatric team was relapse of schizophrenia but as symptoms completely resolved after delivery, indicating the possibility of pre-eclampsia.

Emergence of symptoms during later stages of pregnancy of hypertension, proteinuria, and most importantly, rapid resolution of symptoms after delivery, indicates that the most likely explanation of her symptoms was preeclampsia. The clinical picture did not fit well with typical functional psychiatric illness presentation as the consciousness was impaired in this patient.

In a case of neuropsychiatric effects of preeclampsia reported by Deepak Garg et al in 2015, a primiparous female developed auditory and visual hallucinations at 33 weeks gestation which resolved following delivery and remained asymptomatic thereafter.

Preeclampsia is mainly responsible for the world's large maternal mortality rates; between 2006 and 2008, the rate was 0.83 per 100,000 maternities in the UK, accounting for 18% of direct maternal deaths mostly due to acute cerebral complications. From clinical observation, eclampsia may occur despite a mild clinical picture and before the development of hypertension or proteinuria. Furthermore, failure of cerebrovascular auto-regulatory mechanisms in response to either an acute and/or relatively large blood pressure increase may be more important than the absolute blood pressure value. It may be the acuity of the blood pressure rise in the setting of endothelial dysfunction that interrupts the delicate balance between capillary and cellular perfusion pressures that leads to the neurological complications of preeclampsia.

Neurological symptoms in hypertensive encephalopathy have been attributed to marked vasospasm or forced vasodilatation that result in a major proportion of this morbidity, including blindness, persistent neurological deficits secondary to stroke, and later cognitive impairment. A study done by Zunker P found that neurological symptoms in preeclamptic patients have been attributed to marked vasospasm or forced vasodilatation. Intracranial arterial blood flow velocities were elevated in ten out of 12 patients. The preliminary findings suggest that a forced vasodilatation, probably due to passive over-distension of cerebral arterioles and vasogenic edema rather than vasospasm, is responsible for the observed high cerebral flow velocities and developing symptoms.

Psychiatric perspectives:
Women with psychotic disorders are at an increased risk of obstetric and psychiatric complications (Howard 2005). Psychotic relapse during pregnancy is rare but women with a history of affective psychosis are at a high risk of postpartum relapse. There is high risk of obstetric complications, mixed evidence of stillbirths and neonatal deaths, and there is some weaker evidence of an association with sudden infant death syndrome (Howard 2005). In a study investigating the psychosocial outcomes of pregnancies in women with a history of psychotic disorder, 27% of women had a psychotic episode and 38% had a non-psychotic depression in the first year after birth (Howard et al 2004). Women with non-affective psychosis were at a significantly higher risk of postnatal depression compared with controls (Howard et al 2004). The author concluded that women with history of psychotic disorder are at higher risk of psychiatric illness postpartum, particularly a two-fold risk of postnatal depression (Howard et al 2004).

A prospective population-based study found that 623 consecutive nulliparous women with singleton pregnancies were studied, of them, 28 (4.5%) women developed preeclampsia. Depression was observed in 185 (30%), women and anxiety was observed in 99 (16%) in early pregnancy. Depression was associated with increased risk (odds ratio [OR] 2.5; 95% confidence interval [CI] 1.1, 5.4) for preeclampsia, as was anxiety (OR 3.2; 95% CI 1.4, 7.4). Either depression or anxiety, or both, were associated with increased risk (OR 3.1; 95% CI 1.4, 6.9) for preeclampsia.

Several studies found that preeclampsia during pregnancy, as well as during the time around delivery, significantly increased the risk of schizophrenia. But risk of developing preeclampsia in a patient diagnosed with schizophrenia is not well studied.

Little is known about the psychiatric manifestations of preeclampsia. De Araujo and Salgado in 2016 for the first time described the case of a patient admitted with preeclampsia and psychiatric symptoms of psychosis and catatonia with the details of diagnosis and management. Their case report highlighted the role of the clinician in considering the possibility of neuropsychiatric manifestations secondary to pathological conditions.

As Gelenberg (1976) said, catatonia is not a rare phenomenon, does not automatically imply a “functional” disorder, and certainly does not always indicate Schizophrenia.

Catatonic disorder due to medical condition must be first considered in every patient with catatonic signs, particularly in a patient with new onset catatonia. In addition to a careful psychiatric and medical history including history of drug use, and a detailed neurological, medical and psychiatric examination, diagnostic procedures like CT, MRI, EEG should be used, if indicated by the patient’s history. Like hypertension or Parkinsonism, catatonia discovered on physical examination should spur a medical investigation to uncover the probable cause (Gelenberg, 1976). It is good to be aware of firstly, the presence of catatonic signs in a variety of disorders (Carroll et al, 1994) and secondly, the potential serious organic illnesses which may underlie the catatonic syndrome(Talbot-Stern et al,2000).

Nina Gonzales et al (2014) reported a case of perinatal catatonia and a review of literature of published cases of catatonia. They were able to identify only 3 case series of perinatal catatonia in the medical literature: Sobel described 33 pregnant patients with “psychotic agitation or catatonia” treated with ECT and a small 2004 study in Taiwan of 15 postpartum mentally-ill patients with a 26.6% prevalence of catatonic features in 4 patients. Finally, a 2013 Indian case series described 13 patients who received ECT for catatonic agitation in a cohort of 78 patients psychiatrically hospitalized for postpartum psychosis, yielding a prevalence of 16.7% in that population. An additional 16 cases of perinatal and postpartum catatonia in the literature was found when they searched the English literature using Pubmed. The authors summarized the conditions associated with the catatonic syndrome in these cases which included neuroleptic malignant syndrome, paraneoplastic limbic encephalitis, atypical posterior reversible encephalopathy, eclampsia, and cerebrovascular disease. The unexpectedly high rate of organic etiologies in the individual cases may indicate a need for heightened concern for underlying medical and neurologic conditions when clinicians are faced with a perinatal patient with catatonia. Eclampsia, with its associated autonomic instability and neurotoxic complications, should be specifically ruled out. Both seizures and posterior reversible encephalopathy are known sequelae from eclampsia and maybe associated with, and even causative of, catatonia. Additionally, the treatment of eclampsia is emergent delivery of the fetus. NMDA receptor encephalitis was specifically diagnosed in 3 pregnant patients with catatonic symptoms; serological studies, magnetic resonance imaging of the brain, electroencephalography, and cerebrospinal fluid analysis should be obtained in similar cases.

Relatively few studies have evaluated the extent to which maternal psychiatric diagnoses are risk factors for preeclampsia, and results from available studies particularly related to maternal mood and anxiety disorders are associated with increased preeclampsia risk however there is still scarcity of data related to risks from psychotic disorder particularly in schizophrenia.

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