To the Editor

Levetiracetam Induced Acute Pancreatitis Case in Pregnancy

It is reported that at least 25% of people with epilepsy are women of child-bearing age. Levetiracetam, a second generation antiepileptic drug, has been preferred in pregnancy and lactation due to promising safety in pregnancy[1,2]. Herein, we report a pregnant case of levetiracetam induced pancreatitis. Twenty-five year old pregnant woman at 27 weeks gestation was consulted to gastroenterology department due to abdominal pain, nausea and hyperamilasemia of 396 U/L (normal range in our laboratory: 28-128). Her lipase level was also high 264 U/L (N: <60 U/L). Ultrasound showed enlarged, edematous pancreas but no gallstones. Serological analysis was normal. Blood count disclosed anemia (Hb: 9.1 g/dl). Serum calcium and lipid levels were within normal limits. Her past medical history was unremarkable except levetiracetam monotherapy start due to epileptic seizure at 12 weeks gestation. Levetiracetam was discontinued. Supportive measures such as fluid-electrolyte therapy was given. Her amylase level declined to normal level in a week. Abdominal pain was over. Antiepileptic drug was not recommended by the neurology department until the delivery.

Pancreatitis in pregnancy had been associated in the past with a high maternal and fetal morbidity rate or preterm labor[3]. Earlier diagnosis and greater treatment options improve the prognosis of pancreatitis in pregnancy. Pancreatitis in pregnancy is mostly related with gallstones. Although levetiracetam has been known to be a safe drug in pregnancy, pancreatitis should be considered in patients with epilepsy on levetiracetam.

Murat Sarikaya[1], Nesibe Taser[1], Zeynal Dogan[1], Bilal Ergul[1], F. Irsel Tezer[2], Levent Filik[1]
[1]Ankara Research Hospital
[2]Hacettepe University, Ankara, Turkey
Email: drfilik@gmail.com

References

1. Mawhinney E, Craig J, Morrow J, et al. Levetiracetam in pregnancy: results from the UK and Ireland epilepsy and pregnancy registers. Neurology. 2013;80(4):400-5.
2. Reimers A, Brodtkorb E. Second-generation antiepileptic drugs and pregnancy: a guide for clinicians. Expert Rev Neurother. 2012; 12(6):707-17.
3. Diegelmann L. Nonobstetric abdominal pain and surgical emergencies in pregnancy. Emerg Med Clin North Am. 2012;30 (4):885-901.

To the Editor

Pulmonary Edema and Simultaneous Cardiac Dysfunction After Epileptic Seizures

Severe pulmonary and/or cardiovascular dysfunctions may be the result of acute insults of the central nervous system (CNS). Acute pulmonary edema occurring shortly after a significant CNS injury was termed neurogenic pulmonary edema (NPE). It was described mainly as a rapid consequence of subarachnoid bleeding or intracranial hemorrhage, but many injurious CNS events can be associated with this pathology[1]. The pathogenesis of NPE is not entirely clear yet, but the pathophysiological mechanism is thought to be an increase of intracranial pressure with an increased central sympathetic nerve activity transmitted via peripheral α- or β- adrenergic receptors. Clinically, these patients may demonstrate a broad spectrum of presentations from an asymptomatic clinical picture to acute and severe pulmonary edema[1,2].

On the other hand, clinical studies have shown that acute cerebral lesions (in particular stroke, haemorrhage and seizures) may induce changes in cardiovascular functions including hypertension, arrhythmias and myocardial necrosis. Moreover, it is demonstrated that damage to the insular cortex, the amygdala, lateral hypothalamus and brain stem is likely to cause disturbances in either sympathetic or parasympathetic autonomic system with subsequent cardiac dysfunction[1-4].

It is well known that commonly during seizures both transient changes in the respiratory function (e.g. dyspnea, apnea, cyanosis) and cardiac symptoms (e.g. tachycardia, bradycardia) can occur[1,3].

We report an interesting case of acute bilateral pulmonary edema and concomitant left ventricular dysfunction occurring after epileptic seizures.

Case Report

A 61-year-old woman, with previous acute myocardial infarction in chronic therapy with acetyl salicylic acid and in current good cardiovascular condition, was admitted to the Emergency Department due to two generalized tonic-clonic seizures within short distance of each other. As reported by witnesses, the seizures were associated with prolonged apnoea and cyanosis. During the first medical observation, she was not responsive and showed respiratory failure with evidence of large amounts of blood-stained secretions. There was no evidence of gastric acid aspiration or acute lung infection. Metabolic blood tests were not altered.

Her electrocardiogram (ECG) showed tachysystole, heart rate was 110 beats/minute (min) and cardiac enzyme levels were no significantly above the normal range. The chest auscultation revealed widespread fine crackles, arterial blood gas tensions suggested alveolar hypoventilation and respiratory acidosis (pH: 6.7; pCO_2: 84 mmHg; pO_2 52 mmHg). A chest X-ray showed widespread bilateral pulmonary edema (Figure). Moreover, echocardiography demonstrated depression of left ventricular function with a shortening fraction of 30%. The EEG revealed diffuse slowing as post-critical state. Brain computed tomography (CT) scan and magnetic resonance imaging (MRI) were both normal. The patient was sedated, intubated and ventilated. She was treated with antiepileptic therapy (levetiracetam 2000 mg/day).

https://doi.org/10.1017/S0317167100022629 Published online by Cambridge University Press