Neurologic Complications in Pregnancy

Mauricio Ruiz Cuero, MD[a], Panayiotis N. Varelas, MD, PhD[b,*]

KEYWORDS

• Pregnancy • Stroke • Eclampsia • Status epilepticus • Brain death

KEY POINTS

- This article discusses the neurologic complications during pregnancy.
- This article analyzes and summarizes the most recent data about stroke, eclampsia, status epilepticus, neuromuscular disease, and brain death during pregnancy.
- This article emphasizes the physiopathology, epidemiology, and modality of treatments of the previously mentioned conditions.

INTRODUCTION

Obstetric practice occasionally mandates for admission of unstable patients to an intensive care unit. The reasons why such patients become critical can either be linked directly to the physiologic changes that occur during pregnancy and the puerperium or can be independent and coincidental. Because of its complexity and importance for the functional outcome of the mother, the nervous system, if affected, requires specialized management that in the past was offered by consulting services in neurology or neurosurgery. These days it may be better delivered by a team that includes neurointensivists and obstetricians in the neurosciences intensive care unit (NSU).

PREECLAMPSIA-ECLAMPSIA

Preeclampsia (PREC) is defined as the constellation of newly diagnosed hypertension (systolic/diastolic blood pressure [BP] $\geq$140/90 mm Hg on 2 occasions at least 4 hours apart after 20 weeks' gestation in a woman with previously normal BP; or BP $\geq$160/110 mm Hg, confirmed within a few minutes to facilitate timely antihypertensive

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[a] Neurocritical Care, Henry Ford Hospital, 2799 West Grand Boulevard, Detroit, MI 48202, USA;
[b] Neurosciences Critical Care Services, Neuro-Intensive Care Unit, Department of Neurology, Henry Ford Hospital, Wayne State University, K-11, 2799 West Grand Boulevard, Detroit, MI 48202, USA
* Corresponding author.
E-mail address: varelas@neuro.hfh.edu

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treatment) and proteinuria (>300 mg/24 h or ≥1+ in dipstick testing; because of variability of qualitative determinations, this method is discouraged for diagnostic use unless other approaches are not readily available[1]; or protein/creatinine ratio ≥0.3). Alternatively, in the absence of proteinuria, it is defined by the presence of new onset of hypertension, as described earlier, with new onset of any of the following: serum creatinine level greater than 1.1 mg/dL or a doubling of the serum creatinine level in the absence of other renal disease, new onset of cerebral or visual symptoms, right upper quadrant or epigastric pain, pulmonary edema, thrombocytopenia less than,100,000/μL, or increased aspartate aminotransferase (AST) or alanine aminotransferase (ALT) level (to twice the normal concentration).[1] These systemic organ dysfunctions together with BP greater than or equal to 160/110 mm Hg should be considered severe features of PREC. Eclampsia (EC) is defined as seizures occurring before, during, or after delivery, and in up to 38% of cases it can occur without symptoms or signs of PREC.[1,2]

Epidemiology and Pathophysiology

In the developed world the incidence for PREC is 6% to 8% of pregnancies and for EC 1 in 2000 deliveries, although in the developing countries the numbers are much higher. EC confers a 1.8% maternal mortality and a 35% complication rate and in the United States ranks second only to embolic events as cause of maternal mortality.[3] PREC and its sequelae account for 20% to 50% of obstetric admissions to the NSU.[4]

The cause of PREC-EC is unknown. Pathophysiologic changes in the placental circulation, such as alteration of the ratio of prostacyclin/thromboxane/Flt-1, platelet activation/aggregation, and endothelial damage with fibrin deposition, lead to placental ischemia, diffuse maternal vasospasm, and microangiopathy.[5] The cerebral features include disruption of the capillary tight junctions and extravasation of fluids into the perivascular spaces, white matter edema, and cortical microhemorrhages or macrohemorrhages. Areas of infarction or ischemia are common, and are usually seen in the parieto-occipital watershed zones.[6,7] Because EC may develop at BPs that are considerably lower than those reported with hypertensive encephalopathy, a shift of the cerebral autoregulatory curve to the left or a reduced shift to the right, or loss of autoregulation with development of hydrostatic-vasogenic cerebral edema as a result of brain hyperperfusion at less than the necessary cerebral perfusion pressures, has been postulated.[8]

Clinical Presentation

Seizures are the hallmark of EC. They usually occur before childbirth or during labor, but in some women they occur as late as 10 to 23 days postpartum.[3] After the first 48 hours postpartum, clinicians should look for another cause, because only 3% of women experience late seizures from EC.[9] Seizures are usually generalized tonic-clonic, but occasionally may have a focal onset.[10] Headache, visual hallucinations, photophobia, confusion, and coma are other symptoms associated with EC.

Radiographic Features

In most EC cases computed tomography (CT) of the head is normal,[11] but in some it reveals focal lesions, such as cerebral edema, subarachnoid hemorrhage (SAH) or intraparenchymal hemorrhage, or, in patients with cortical blindness, occipital symmetric hypodensities.[6,12] MRI may show reversible low-signal intensities in T1 and high-signal intensities in T2-weighted images (with high apparent diffusion coefficient; ie, without diffusion restriction).[13]