Journal of the Neurological Sciences 271 (2008) 158–167

Journal of the Neurological Sciences

www.elsevier.com/locate/jns

Eclampsia: A neurological perspective

A.K. Shah [a,*], K. Rajamani [a], J.E. Whitty [b]

[a] Department of Neurology, Wayne State University/Detroit Medical Center, Detroit, MI 48201, United States
[b] Department of Obstetrics & Gynecology, Meharry Medical College, Nashville, TN 37208, United States

Received 19 July 2007; received in revised form 28 March 2008; accepted 8 April 2008
Available online 20 May 2008

Abstract

Eclampsia is a poorly understood disorder characterized by seizures or unexplained coma in setting of gestational hypertension. Its neurological manifestations are varied and are an important cause of the morbidity and mortality associated. We present a comprehensive prospective study of forty women recruited over four years describing neurological symptoms and signs, neuroimaging and laboratory studies as well as prognosis including 3–6 months follow-up. The seizures occurred in the postpartum period in majority of women (55%), while 45% had seizures before labor, and the rest (5%) during labor. Interestingly, one third of the women suffered their first seizures more than 48 h postpartum (late postpartum eclampsia). A sizable minority suffered more than one seizure and some had documented partial seizures. Headache preceded seizures by more than a day and was described as throbbing or pounding pain by most. The visual symptoms in decreasing frequency were blurring, blindness, scotoma and visual processing deficits. The most common finding during the neurological exam was memory deficits, followed by increased deep tendon reflexes (asymmetric in some), visual perception deficits, visual information processing deficits, altered mental status and cranial nerve deficits. Intracranial or intraspinal pressure when examined was elevated. Among neuroimaging studies, MRI was more sensitive compared to CT scan. The MRI abnormalities included both white as well as gray matter and the most common location of abnormalities was high frontal/parietal lobe. The laboratory studies revealed proteinuria in majority, but not in all. The liver function tests were abnormal in many, while few patients had HELLP syndrome. The neurological deficits resolved by the time of discharge in all. At follow-up, some patients developed new neurological problems such as recurrent headaches or seizures.

Keywords: Eclampsia; Hypertensive encephalopathy; Seizures; Headache; Visual disturbance; Pregnancy; Toxemia of pregnancy; Neurological; MRI; CT scan; Neuroimaging

1. Introduction

Preeclampsia–eclampsia is a complex hypertensive disorder of pregnancy affecting multiple systems. The nervous system is commonly affected and is a cause of significant morbidity and mortality in these women. Fortunately, the neurological deficits are often reversible and in most cases do not leave them with permanent disability. Systematic and detailed studies of the neurological aspects of this condition are done in the past but are of limited scope in recent literature because these patients are most often cared for by obstetricians.

Preeclampsia and eclampsia are not distinct disorders from each other. The differentiation is artificial as they are part of a continuum starting with gestational hypertension on one end. A more severe form is called preeclampsia and when convulsions or unexplained coma occur in the setting of gestational hypertension, the condition is referred to as eclampsia. The nervous system can be affected both in gestational hypertension and preeclampsia. Current criteria to define preeclampsia include 1) hypertension and proteinuria detected for the first time after 20 weeks' gestation; 2) hypertension defined as systolic blood pressure $\geq$140 mm Hg or diastolic blood pressure $\geq$90 mm Hg, 3) proteinuria defined as $\geq$300 mg/24 h or $\geq$30 mg/mmol in a single specimen or $\geq$1+ on dipstick [1]. In most instances, seizures are prerequisite for diagnosis of eclampsia. None the less, some have included patients who

* Corresponding author. 8D, UHC, 4201, St. Antoine, Detroit, MI 48201, United States. Tel.: +1 313 745 8808; fax: +1 313 745 4216.
E-mail address: ashah@med.wayne.edu (A.K. Shah).

0022-510X/$ - see front matter © 2008 Elsevier B.V. All rights reserved.
doi:10.1016/j.jns.2008.04.010

lapse into coma without a witnessed seizure in studies of eclampsia [2,3].

Neurological manifestations and symptoms of eclampsia are varied. Convulsions are the most frequent complication, related to the arbitrary definition of eclampsia. Besides seizures, other common symptoms include, headache, cortical blindness, blurring of vision, aphasia, psychosis, facial nerve palsy, cerebral hemorrhage, retinal blindness, retinal edema, cerebral edema, hemiparesis and other focal weakness [1–4]. However, these symptoms can also be present in women with severe preeclampsia. The spectrum of neurological manifestations is reported in the past, but large series focusing on neurological manifestations are few. In the present study, we have systematically collected information about neurological manifestations in women with eclampsia.

2. Methods

Patients were included from an inner city university hospital for this study. All women who suffered one or more seizures during the peripartum period (2 days pre-delivery to 1 month postpartum) were evaluated for possible inclusion. A patient was selected for the study if she suffered eclampsia (seizures/coma in setting of gestational hypertension). If the patient had history of recurrent unprovoked seizures in the past, she was excluded from the study. In addition, other clinical information and neuroimaging findings were reviewed to exclude other possible causes of seizures, such as history of hypoxia, central nervous system infections, intracerebral hemorrhage, brain tumors, metabolic derangements, and recent use of medications that can lower seizure threshold. All the patients were selected prospectively over a four year period. A neurologist (AS) interviewed all patients in a structured manner and performed a complete neurological exam within a day of the seizure. Relevant data was collected from charts. No specific investigations were required or suggested for participating women. A follow-up either by phone or clinic visits was conducted 3 months and/or 6 months after the event when possible. An informed consent was obtained in writing for participation in the study. The study is approved by the local IRB.

The following information was obtained; general demographic information including race, and age; handedness, gestational age at admission and at delivery. Past medical history, family history, personal history, and current as well as recent medication including over the counter medication history and past obstetrical history were also noted. History of present illness obtained in detail, including information about headache and seizure. Information was obtained from the patient as well as from witnesses of the seizure activity whenever available. Complete prodrome and seizure description was obtained from patient as well as the witness, usually a family member or hospital staff if the seizure(s) occurred in the hospital.

A detailed information regarding headaches was systematically collected via structured interviews. Following information was gathered; past history of headaches, onset time, duration, character, location, associated features; such as, photophobia, sonophobia, nausea, vomiting. Details of visual changes including blurring or loss of vision, diplopia, etc. were also noted. If there was any other neurological symptom, the details were recorded.

General physical exam including blood pressure, abdominal exam, and evaluation for peripheral edema was performed. A complete neurological exam was performed by the same neurologist (AS) in all women. The seizures were classified according to the classification published by International League Against Epilepsy (ILAE) [5]. Response to treatment was noted.

2.1. Laboratory studies

Following laboratory study findings were recorded when available; CBC with platelets, serum electrolytes, BUN, creatinine, calcium, magnesium, AST, ALT, Alkaline phosphatase, urinanalysis, urine drug screen, and cerebrospinal fluid analysis.

2.2. Neuroimaging

Neuroimaging was performed in most and reviewed by the same neurologist (AS) and reports were obtained. A clinical CT scan without contrast or the MRI scan was obtained on as needed basis and during the study period the MRI techniques changed, and therefore they were not performed uniformly. However, all MR imaging protocol included at least one T1 weighted images sequence and one T2 weighted images sequences. When clinically necessary, MR angiogram (MRA) and/or 2D flight of time MR venogram (MRV) were also obtained. Once diffusion weighted imaging (DWI) was available for clinical use, it was also included in some patients, and however, ADC maps were not available in any patient.

The CT scan and MRI results were analyzed separately. The MR imaging abnormalities on T1 weighted images with or without contrast; on long T2 sequences (T2 weighted or FLAIR) images as well as diffusion weighted images (DWI) and MR angiogram (MRA) and MR venogram (MRV) were described separately. In addition, the location of each hyperintense lesion identified on long T2 sequences was carefully scrutinized and was placed in one of the following 16 regions: anterior frontal (AF), high posterior frontal (PF), high parietal (HP), inferior parietal (IP), lateral temporal (LT), medial temporal (MT), lateral occipital (LO), medial occipital (MO), occipital pole (OP), basal ganglia (BG), internal capsule (IC), external capsule (EC), corona radiata (CR), thalamus (T), brain stem (BS), cerebellum (C).

Follow-up information was obtained by a telephone interview or clinic visit when possible at 3 to 6 month intervals. During follow-up, information about ongoing seizures or headache and their character was specifically asked. Information about any other health problem was also obtained.