Case Report

Repeated Cranial Computed Tomographic and Magnetic Resonance Imaging Scans in Two Cases of Eclampsia

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In two cases of eclampsia with consumptive thrombocytopenia, the maximum increase in blood pressure and the lowest platelet count coincided with the maximum degree of neurologic and neuroradiologic abnormality. Computed tomograms showed decreased attenuation, and T2-weighted magnetic resonance images showed increased signal intensity focally in the cerebral cortex and the deep gray and white matter. Blood pressure, platelet count, clinical status, and roentgenograms normalized completely in both cases. Severe arterial hypertension and disseminated transitory microvascular occlusions presumably caused multiple small foci of brain edema that resolved without remaining detectable ischemic brain damage. (Stroke 1989;20:547-553)

Eclampsia is the advanced stage of pregnancy-induced hypertension, resulting in convulsions; its etiology is unknown. There have been reports of cases of eclampsia investigated with cranial computed tomography (CT) scans. We report the course of arterial hypertension and consumptive thrombocytopenia related to clinical status and findings of repeated CT and magnetic resonance imaging (MRI) scans in two cases of eclampsia.

Case Reports

Case 1

A 20-year-old woman, gravida 1, para 0, with signs of preeclampsia in the 28th week of gestation presented with subcutaneous edema, severe proteinuria, a rapid increase in blood pressure to 180/100 mm Hg (Figure 1), and a weight gain of 22.3 kg during the preceding 14 weeks. She was admitted to a local hospital and was treated with 100 mg/day atenolol, 50 mg/day dihydralazine, and 40 mg/day furosemide. Her platelet count (117X10^9/l) (Figure 1) and serum albumin concentration (22 g/l) had decreased, and her serum urate concentration (463 /mol/l) had increased slightly. She had two generalized seizures without prodromal symptoms the next day. Her blood pressure decreased from 165/110 to 145/100 mm Hg after an intravenous infusion of dihydralazine-chlorpromazine-meperidine, and she was transferred to the referral hospital.

She was free of symptoms on arrival, but termination of pregnancy was indicated because of eclampsia. A 930-g male infant with Apgar scores of 4, 6, and 7 at 1, 5, and 10 minutes was delivered by cesarean section 10 hours after the seizures. Phenytoin (250 mg i.v.) was given repeatedly preoperatively and postoperatively followed by oral administration of 300 mg/day phenytoin. Her hemoglobin concentration (97 g/l) and platelet count (98 x 10^7/l) decreased postoperatively (Figure 1). Plasma fibrinogen level was normal, and plasma prothrombin time (Normotest, Nycomed Als, Oslo, Norway) was increased (150-180-130%), as were her serum concentrations of aspartate (0.8 /kat/l), alanine aminotransferase (0.7 /kat/l), urate (678 /mol/l), and lactate dehydrogenase (10.2 /kat/l).

Two days after the seizures her neurologic examination was normal except for slight bilateral horizontal nystagmus that disappeared within a few days. Cranial CT and T2-weighted (T2W) MRI scans showed a 1X1.5 cm area of decreased attenuation and increased signal intensity, respectively, in the left parieto-occipital region of the cerebral cortex (Figure 2, a and b). Her blood pressure gradually normalized, as did the laboratory values in blood and urine, except for a slight protracted anemia (Figure I). Extensive investigation of her coagulation and fibrinolytic system on the third and 11th days post-

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parturition revealed nothing remarkable apart from minor aberrations related to gestation and the puerperium. Repeated blood films showed anisocytosis and polychromasia, but no schistocytes or signs of hemolytic anemia were found. CT (including contrast enhancement) and MRI scans 1 month later showed complete normalization (Figure 2, a and b).

Case 2

A 32-year-old woman, gravida 5, para 4, with a twin pregnancy, developed signs of preeclampsia with a rapid increase in blood pressure, proteinuria, and subcutaneous edema from the 29th week of gestation (Figure 3). She was treated with a diuretic and restricted fluid and salt intake but was admitted to a local hospital 3 weeks later with a weight gain of 28.8 kg during pregnancy. Her blood pressure was 190/100 mm Hg, and she complained of headache. She was treated with 6.25 mg i.m. dihydralazine and 20 mg i.v. furosemide and was transferred to the referral hospital. Hemoglobin concentration (97 g/l), platelet count (53 × 10^9/l), and serum albumin concentration (19 g/l) were decreased. Plasma fibrinogen level was normal; prothrombin time was slightly increased (160%), as were the serum concentrations of alkaline phosphatase (5.4 μkat/l), aspartate (1.0 μkat/l), alanine aminotransferase (1.0 μkat/l), and urate (530 μmol/l). Urine specimens showed both albuminuria and hematuria.

She was treated orally with 50–100 mg/day atenolol and 15 mg/day prednisolone for 3 days and 5 mg/day prednisolone for 4 days and with 20 mg i.v. furosemide intermittently. Her blood pressure dropped to 150/90 mm Hg, and her body weight decreased promptly by 2–3 kg. Her platelet count increased to >100 × 10^9/l (Figure 3). However, her preeclampsia was difficult to control, and termination of the pregnancy was indicated 5 days after admission. Labor was induced with amniotomy and intravenous oxytocin infusion, and the patient delivered two healthy boys with full Apgar scores and birth weights of 2,080 and 2,240 g. The combined treatment with atenolol and prednisolone was terminated.

Her blood pressure fluctuated between 150/100 and 200/120 mm Hg the first days postpartum. She was agitated and complained of neck pain. A recrudescence of her decreased platelet count occurred (Figure 3). She had three generalized seizures 31 hours after delivery and was treated with repeated intravenous injections of 5 mg diazepam and 250 mg phenytoin. She did not regain full consciousness and was transferred to the intensive care unit, intubated, and artificially hyperventilated. An acute CT scan showed several ill-defined areas of decreased attenuation bilaterally within the mesencephalon, thalamus, and basal ganglia, larger on the left than on the right side (Figure 4, a and b), which were initially interpreted as fresh infarcts. She received 500 ml/day dextran and betamethasone (16 mg the first day, thereafter in tapered dosage) intravenously.

She was still unconscious the next morning, 5–6 hours after her seizures. Her left pupil was dilated and failed to react to light. Oculocephalic responses were absent. The corneal reflex was absent on the left side and sluggish on the right. She responded to noxious stimuli mainly with minimal movements of her right arm and leg. The deep tendon reflexes were equally hyperactive bilaterally throughout her arms and legs, with bilateral extensor plantar responses. Repeated CT and MRI scans showed changes that extended to the white matter lateral to the left ventricle. An electroencephalogram (EEG) showed generalized slow activity (7–8 Hz alpha rhythm) and a paradoxical arousal effect.

She responded monosyllabically and recognized her relatives the next day. She was treated with 1.5 mg/day betamethasone and 300 mg/day phenytoin orally for 2 weeks. She was awake and alert but still had a slight right-sided hemiparesis the fifth day...
FIGURE 2. Area of hypodensity on computed tomogram (a) and increased signal on T2-weighted magnetic resonance image (b) in left parieto-occipital region of cerebral cortex (arrows) of 20-year-old patient (Case 1) 2 days after eclampsia. Complete normalization after 4 weeks (c, d).

postpartum. A T2W MRI scan showed increased signal bilaterally in the mesencephalon, thalamus, and basal ganglia (Figure 4, c and d), but the changes appeared less confluent and less extensive than those 2 days earlier. In addition, multiple minute spots were observed bilaterally in the cerebral cortex, the parieto-occipital region, and the central white matter.
FIGURE 3. Time course of blood pressure increase, thrombocytopenia, and albuminuria in 32-year-old patient with eclampsia (Case 2) who reported headache at A, showed agitation at B, had seizures, paresis, and coma at C, and then improved successively. Cranial computed tomograms were made at C (see Figure 4, a and b), D, and F; magnetic resonance images were made at D, E (see Figure 4, c and d), and F (see Figure 5, e and f).

One week later she complained only of slight dizziness and difficulties in concentration. Repeat CT and MRI scans at that time showed almost complete normalization of the alterations previously noted (Figure 5). The CT scan was unaltered by contrast enhancement. An EEG showed normal alpha rhythm frequency (10–11 Hz). Blood pressure, blood and urine test results (Figure 3), and neurologic examination were normal on her discharge from the hospital. A repeat MRI scan 6 months later showed complete normalization.

Discussion

Our two patients fulfill the criteria of eclampsia, that is, rapid and severe increases in blood pressure and body weight, subcutaneous edema, proteinuria, and seizures with or without focal neurologic symptoms and signs developing after the 24th week of pregnancy. The combination of symptoms also suggests hypertensive encephalopathy.4,5 Alterations during the third trimester of normal pregnancy result in an increased capacity for intravascular coagulation and fibrin deposition.6–10 Minor activation of the hemostatic mechanisms may result in pathologic clotting in certain complications of pregnancy, for example, eclampsia.7,11–13 Multiple organs (e.g., brain, kidney, lung, liver) show fibrin-rich vascular deposition,14 edema, infarcts, and hemorrhages in severe and fatal eclampsia.2,15–18 Because the concentrations of many clotting factors increase successively throughout pregnancy anyway, their levels often appear to be within the normal range in consumptive coagulopathy associated with eclampsia.6 The only laboratory evidence of disseminated intravascular coagulation (DIC) may be thrombocytopenia and an increase in fibrin degradation products.6,17 In addition, intravascular hemolysis with fragmented erythrocytes (schistocytes) sometimes accompanies eclampsia.19–21 When thrombocytopenia and such microangiopathic hemolytic anemia but only clinically insignificant intravascular coagulation21–22 are found, eclampsia may be difficult to distinguish from thrombotic thrombocytopenic purpura.23 Thrombocytopenia due to platelet consumption is the most frequent and sometimes the sole hemostatic abnormality found in preeclampsia and eclampsia.22–24–28 It has been suggested that the rapid increase in blood pressure damages the arteriolar endothelium to which platelets adhere,22,27,28 but primary immunologic triggering mechanisms have also been proposed.27,29,30

In our two cases of eclampsia, neurologic symptoms and signs were preceded by a rapid increase in blood pressure and a decrease in platelet count. The time course and degree of arterial hypertension and thrombocytopenia correlated closely with the degree of involvement of the brain, liver, and kidneys. Normalization and "supernormalization" (Case 1) of the platelet count within days after delivery, in parallel with a decrease in blood pressure, indicated
rapid termination of consumptive thrombocytopenia. No detailed analysis of the disturbance of hemostasis was possible due to the lack of extensive laboratory investigations during the acute phase. However, increased plasma prothrombin times and normal plasma fibrinogen levels suggest that, although a low-grade and compensated DIC might have occurred, there was no massive intravascular
clotting. We found no major signs of coagulative-fibrinolytic abnormalities in the immediate post-eclamptic period (Case 1), indicating rapid normalization of the hemostatic alterations of pregnancy.6,10

In previously reported cases of eclampsia, cranial CT scans have shown hypodense areas (and sometimes hemorrhages) in the cerebral cortex and subcortical white matter, basal ganglia, subependymal region, internal and external capsules, and cerebellum. The hypodense areas, which frequently disappear within days or weeks, have been regarded as focal brain edema by most authors. Only occasionally is infarction with detectable brain tissue loss seen. In some cases a more generalized brain edema, located mainly in the subcortical white matter, has been the sole finding.2,18 We also found focal areas of low density in the cerebral cortex and the central gray and white matter. The MRI technique we used shows that the hypodense areas correspond to multiple minute spots of increased signal on T2W images, most clearly distinguishable in the resolution phase (see Case 2). A preferential distribution close to arterial end zones and border zones is seen, in both the present and previously published CT scans, even if not explicitly mentioned in previous accounts.21,33,39 In our opinion, the abnormal findings of the CT and MRI scans were caused by multifocal brain edema secondary to severe arterial hypertension and transitory platelet-rich microvascular occlusions. The rapid and complete normalization of the neurologic and neuroradiologic abnormalities suggests that no major ischemic necrosis occurred. As alternative diagnoses, venous thrombosis or thrombosis of the sinuses must be considered. However, the localization of the changes in our two patients was not compatible with these diagnoses, nor were there signs of venous occlusion.

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