scribed beforehand, but that magnesium was administered (up to a maximum of 20 g during the first hour) until an effect was achieved. This means that lower doses of magnesium had been ineffective. After administration of the higher Mg^{2+} doses, both mechanical ventilation and management of bronchoconstriction were successful. Since our high-dose $MgSO_4$ regimen was limited to very severe status asthmaticus, we definitely encourage further dose-finding studies in mechanically ventilated asthmatics, with even higher Mg^{2+} doses than for other medical indications.

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M. Sydow (✉) · T. A. Crozier
S. Zielmann · H. Burchardi
Zentrum für Anaesthesiologie,
Rettungs- und Intensivmedizin,
Georg-August-Universität,
Robert-Koch-Strasse 40,
D-37035 Göttingen, Germany

J. Radke
Klinik für Anaesthesiologie
und Operative Intensivmedizin,
Martin-Luther-Universität,
Magdeburger Strasse 16,
D-06112 Halle, Germany

P. Saura
Ll. Blanch
E. Capdevila
F. Baigorri
J. Martín
M. Corona
A. Artigas

Spontaneous rupture of the liver during pregnancy

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Sir: Liver hemorrhage and rupture is a rare complication of toxemia of pregnancy, with a high perinatal and maternal mortality rate. Although different treatments have been described, only a high degree of suspicion and prompt therapy can reduce mortality and morbidity [1, 2]. We report a case of liver rupture following toxemia and the HELLP syndrome, with successful term delivery in which ischemic lesions of the liver and their recovery were monitored by computed tomography (CT) scan and magnetic resonance imaging (MRI).

A 28-year-old woman, gravida II, was admitted to hospital in her 37th week of pregnancy due to epigastric pain radiating to the back and eclampsia. On arrival to the emergency room she became unconscious with generalized seizures. Blood pressure was 220/110 mmHg. Benzodiazepines and magnesium sulfate were administered. The patient underwent cesarean section, and a compromised baby was soon delivered. After surgery, the patient had hypertension that was difficult to control, a low platelet count of 40000/mm^3, and high levels of glutamic oxaloacetic transaminase (GOT) with 2600 U/l, serum glutamic pyruvic transaminase (GPT) with 1064 U/l, and lactic dehydrogenase (LDH) with 5500 U/l. Coagulation and hematocrit were normal. Twenty-four hours later the patient was admitted to the intensive care unit as her systolic blood pressure (SBP) had dropped below 100 mmHg and hematocrit to 12.3%. Laboratory analysis showed disseminated intravascular coagulation with a prothrombin time of 72.7 s (control 15 s); a partial thromboplastin time of 93.6 s (control 31.7 s); a platelet count of 18000/mm^3, and the presence of fibrin degradation products. There was no evidence of hemolysis, and bilirubin levels were normal at all times. Ultrasonography showed free intra-abdominal fluid. A hemoperitoneum of about 3 l and hepatic hematoma, together with rupture of Glisson's capsule were observed during an emergency operation. Surgical management consisted in intra-abdominal packing to control massive hemorrhage, and drainage. During the postoperative period, the patient developed renal failure and hemodialysis was carried out. Over the next few days, the patient's hematocrit stabilized and she recovered progressively, with normalization of renal function and coagulation. The abdominal packing was removed on the 5th day and the hepatic rupture was covered with epiploon. At that time, an enhanced abdominal CT scan showed a hypodense area involving the right lobe of the liver (Fig. 1a). A follow-up enhanced CT and MRI 8 days later showed hepatic regeneration, although some scattered areas of low density persisted (Fig. 1b). The patient was discharged from the intensive care unit 20 days after admission. After a follow-up period of 3 months, both the mother and baby were in perfect health and CT and MRI were normal.

A syndrome of hemolysis, abnormal liver function tests and low platelet count is recognized as a complication of pre-eclampsia-eclampsia. In 1982, the acronym HELLP was suggested for this collection of signs and symptoms as a differentiated entity in some preeclamptic patients [1, 2]. A significant percentage of reports have referred to patients with no evidence of hemolysis, and the term

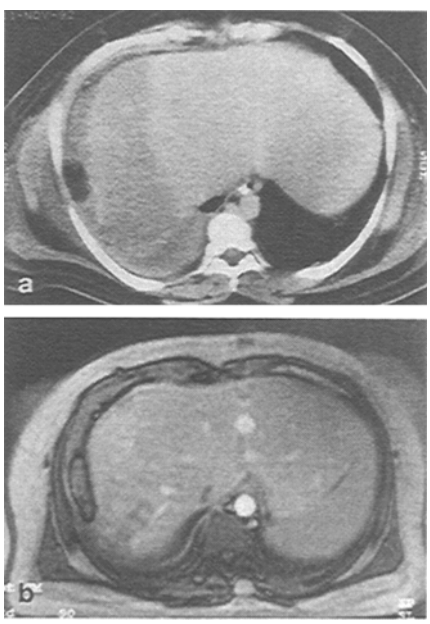

Fig. 1a Enhanced CT scan of the liver reveals a hypodense area involving the right lobe. Pleural effusion and ascites are present. Epiploon fat appears as low-attenuation juxtahepatic structure.
b Contrast-enhanced NMR shows a normal signal intensity area probably consistent with scar tissue

ELLP syndrome is used in these [1]. The patient described above developed a HELLP syndrome without hemolysis, complicated by hepatic rupture. Liver capsule rupture, following subcapsular hematoma is a rare complication of pregnancy with related maternal (0–24%) and fetal-neonatal mortality (7.7–60%). Microscopic study of these cases showed areas of acute necrosis throughout the hepatic parenchyma and the presence of fibrin in the vessels of the liver, thus supporting the possible pathogenetic significance of disseminated intravascular coagulation leading to thrombohemorrhagic complications [4].

In the present case report, dynamic enhanced CT scan and MRI imaging techniques were both useful in detecting hypoperfused areas of the liver and in the follow-up of the patient until total recovery. Chiang et al. [5] suggested that the management of such patients could be modified if liver hematomas were diagnosed early, before rupture. The same authors described a patient with toxemia of pregnancy and the HELLP syndrome, in whom a follow-up abdominal CT scan 14 days after liver hemorrhage revealed a reduction in the areas of hepatic necrosis. Unfortunately, the patient died on day 16 and the time course of the hepatic lesions could not be evaluated. In our case report, an abdominal CT scan showed marked hepatic regeneration, and MRI images were consistent with scar formation. It is of interest to note that, 3 months later, both CT scan and MRI evaluation revealed a seemingly normal liver.

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P. Saura (✉) · Ll. Blanch · E. Capdevila
F. Baigorri · J. Martín · H. Corona
A. Artigas
Intensive Care Department,
Hospital de Sabadell,
Parc Taulí s/n, E-08208 Sabadell, Spain

K. Terasako
N. Seo
T. Murayama
T. Kai
S. Hirata
T. Fujiwara

Are autoimmune mechanisms involved in critical illness polyneuropathy?

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Sirs: We recently encountered a condition that was diagnosed as critical illness polyneuropathy (CIP) on the basis of the clinical features [1, 2]. In this case, we detected a high protein content with pleocytes in the CSF and high titers of IgG antibody against GM_1 ganglioside in the patient's sera.

An 81-year-old man developed multiple organ failure following multiple hepatic abscesses caused by *Klebsiella pneumoniae* infection. The patient had complained of muscle tenderness and presented a considerably elevated serum CPK level of 12310 IU/l (CPK-MB 113 IU/l). After drainage of the abscesses, the patient's condition gradually improved: muscle tenderness subsided and serum CPK levels were normal (50 IU/l) on day 17. However, the patient still remained on a ventilator and neck stiffness was observed. Lumbar puncture revealed an increased protein content (467 mg/ml) with pleocytes (14 cells/3 fields, monocyte 10, segmented cell 4) in the CSF. A week later, he developed flaccid, areflexic tetraparesis with mild peripheral sensory disturbances. The cranial nerves were intact, and CT head and cervical scan, normal. Electromyographic studies were almost normal, and nerve conduction studies of the median nerve revealed normal motor and sensory conduction velocity and M-wave amplitude, but prolonged F-wave latency. Muscle and nerve biopsy in the sural nerve region demonstrated no specific changes. There was no serologic evidence of *Campylobacter jejuni* infection. Steroid pulse therapy followed by six plasmapheresis treatments did not alleviate the symptoms. About 3 months after admission, thin-layer chromatography with immunostaining revealed that serum IgG from the patient had reacted with GM_1. Enzyme-linked immunoadsorbent assay showed that anti-GM_1 antibodies (IgG) were obviously elevated in the patient's serum (absorbance at 492 nm was 1.11), compared with healthy volunteers (mean ± standard deviation of absorbance = 0.036 ± 0.009). Electromyographic studies revealed long-duration and high-voltage waves during voluntary contraction of the biceps. The patient was not completely weaned off the ventilator until about 4 months later.

CIP is similar to the Guillain-Barré syndrome (GBS), but appeared to be related to severe infection and nutritional disorders [1–3]. The condition in our patient was diagnosed as CIP on the basis of its clinical features.

It has recently been suggested that the pathogenic mechanisms of GBS are related to cell-mediated immunity, antibody-targeted macrophage-mediated demyelination or complement-dependent demyelination [4]. However, attribution of the autoimmune response remains to be defined in CIP.

The high protein content with pleocytes in the CSF was not characteristic of CIP and high serum concentrations of anti-GM, antibodies have never been reported in CIP. The pathophysiology of these findings was unclear, but might correspond to symptoms of acute axonal polyneuropathy in a subgroup of GBS [5]. This case suggests that autoimmune mechanisms may also be involved in the pathogenesis of CIP.

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