Hepatic rupture complicating eclampsia in pregnancy

S. KARADIA, C. WALFORD, M. MCSWINEY AND M. S. NIELSEN

Summary
We describe hepatic rupture in a 37-yr-old woman admitted to the intensive care unit after an eclamptic convulsion. The intensive care and surgical management are discussed. (Br. J. Anaesth. 1996; 77: 792–794)

Key words

Hepatic rupture is a rare complication of pre-eclampsia or eclampsia. It may present with lower chest or upper abdominal pain. Hepatic rupture may be associated with disseminated intravascular coagulation (DIC) and subsequent haemorrhage. In unremitting hepatic failure, transplantation may have to be considered if other treatments fail to control haemorrhage.

Case report
A healthy 37-yr-old primigravida was admitted to hospital 12 days post-term for induction of labour after a previously uncomplicated pregnancy. Arterial pressure was 120/72 mm Hg, haemoglobin concentration 126 g litre\(^{-1}\) and platelet count 236 \times 10^9 \text{ litre}\(^{-1}\). Multistix urinalysis was negative for protein. Labour was induced using dinoprostone pessaries and proceeded uneventfully for 4 h during which time the patient satisfactorily self-administered Entonox and used TENS for analgesia. No further requests for additional analgesia were made. Two hours before delivery she complained of severe lower chest pain. The patient was not cyanosed and arterial pressure and heart rate were normal. Clinical examination and electrocardiography were unremarkable. The pain stabilized after administration of pethidine 75 mg i.m. One hour later the patient suffered a grand mal convulsion. The intensive care and electrocardiography were started to maintain uterine contraction. After a loading dose of 1 000 000 u. of blood and 6 u. of platelets. A dopamine infusion of 3 \mu g \text{ kg}\(^{-1}\) \text{ min}\(^{-1}\) was commenced and urine output remained at 1 ml kg\(^{-1}\) h\(^{-1}\).

The patient was transferred, with her trachea still intubated and her lungs ventilated, to the ICU for further monitoring of haematological and vital signs. She was sedated with an infusion of midazolam 50 \mu g \text{ kg}\(^{-1}\) \text{ h}\(^{-1}\) and morphine 30 \mu g \text{ kg}\(^{-1}\) \text{ h}\(^{-1}\). No further convulsions occurred.

Repeated infusion of FFP, platelets, cryoprecipitate and colloid were administered to maintain normovolaemia and coagulation function. An infusion of aprotinin 200 000 u. h\(^{-1}\) was commenced after a loading dose of 1 000 000 u. Six hours after admission to the ICU the patient became hypotensive and developed a distended abdomen. An ultrasound scan showed intraperitoneal fluid and mixed reflectivity in the region of the liver capsule, suggesting haemorrhage. After further prompt fluid resuscitation, laparotomy was performed. At operation a sheared rupture of the right lobe of the liver was seen. 2+ of protein on Multistix urinalysis (equivalent to 1 g litre\(^{-1}\)).

Eclampsia was diagnosed and i.v. magnesium sulphate was commenced in a dose of 5 g followed by an infusion of 1 g h\(^{-1}\). Haemoglobin concentration was 135 g litre\(^{-1}\), white cell count 22.5 g litre\(^{-1}\) and platelet count 101 \times 10^9 \text{ litre}\(^{-1}\). Invasive monitoring of arterial and central venous pressures was commenced in addition to pulse oximetry and urine output. Before transfer to the intensive care unit (ICU) the patient suffered a post-partum haemorrhage of 1500 ml requiring an emergency examination under anaesthesia. After anaesthesia, gynaecological examination revealed generalized oozing of blood from the external genitalia with an atomic uterus filled with blood clot. The blood clot was removed and bimanual pressure exerted to achieve uterine contraction. A vaginal pack was inserted to help tamponade the haemorrhage. Blood loss with this procedure was approximately 1000 ml. A continuous infusion of Synotocin at a rate of 10 u. h\(^{-1}\) was commenced to maintain uterine contraction. A full blood count at this stage revealed a haemoglobin concentration of 96 g litre\(^{-1}\) and a platelet count of 62 \times 10^9 \text{ litre}\(^{-1}\) with an international normalized ratio (INR) of 4. The patient received 6 u. of blood and 6 u. of platelets. A dopamine infusion of 3 \mu g \text{ kg}\(^{-1}\) \text{ min}\(^{-1}\) was commenced and urine output remained at 1 ml kg\(^{-1}\) h\(^{-1}\).

S. KARADIA, FRCA, C. WALFORD, DA, M. MCSWINEY*, FRCA, M. S. NIELSEN, FRCA, Shackleton Department of Anaesthesia, Southampton General Hospital, Tremona Road, Southampton SO16 6YD. Accepted for publication: July 29, 1996.

*Present address: Cheltenham General Hospital, Cheltenham.
found. The liver substance showed generalized fatty infiltration and petechiae. Only limited haemostasis was achieved and the peritoneal cavity was packed and the abdomen closed. After operation she was transferred back to the ICU where she continued to show cardiovascular and haematological signs of haemorrhage. Systolic arterial pressure was now persistently low at 90 mm Hg with a tachycardia of 110 beat min\(^{-1}\) and a decreasing urine output. Haemoglobin concentration was 96 g dl\(^{-1}\) despite 22 u. of blood, platelet count 46 × 10\(^9\) litre\(^{-1}\) despite 20 u. of platelets and INR was 2.4. A further laparotomy revealed a right subcapsular haematoma of the liver. A right hemihepatectomy was performed as the only means of achieving haemostasis; subsequent histology showed diffuse necrosis of the architecture of the liver.

After operation a pulmonary artery catheter was inserted. Cardiac index was 2.1 litre min\(^{-1}\) m\(^2\) with a pulmonary artery wedge pressure of 11 mm Hg. Dobutamine was commenced at a rate of 5 μg kg\(^{-1}\) min\(^{-1}\) which produced a cardiac index of 4.5 litre min\(^{-1}\) m\(^2\). Further volume replacement was necessary to maintain cardiac filling pressure.

Despite an eventual blood transfusion of more than 120 u., an infusion of 20 u. of cryoprecipitate, 42 u. of platelets and 48 u. of FFP, haemostasis was not achieved and the INR remained greater than 1.5. Two further laparotomies were performed to evacuate the intraperitoneal blood clot, replace intra-abdominal packs and attempt haemostasis. A subcapsular haematoma of the remaining left lobe of the liver had developed which was treated conservatively.

As all conventional means of controlling haemorrhage had failed, the patient was referred to a supra-regional liver unit for consideration of transplantation as a means of controlling bleeding. She was accepted and transferred using the intensive care transport system. The patient remained stable throughout the journey, requiring only 3 u. of blood. On arrival the patient remained stable for 24 h when a further laparotomy was performed to remove the intra-abdominal packs, evacuate the haematoma and to administer argon diathermy to the raw surface of the left lobe of the liver. At this time the vaginal packs were removed and the uterus and external genitalia examined; there was no further oozing of blood from these sites. After operation she was haemodynamically stable, her INR remained in the range 1.2–1.5 and renal function was satisfactory, with a urine output of 1 ml kg\(^{-1}\) h\(^{-1}\). Under these circumstances liver transplant was considered unnecessary.

Over the subsequent 5 weeks the patient continued to improve despite requiring treatment for intermittent sepsis and mild ARDS. The patient returned to her original hospital where she was reunited with her baby.

**Discussion**

Our patient developed eclampsia without any preceding features of pre-eclampsia. Douglas and Redman reported recently that 38% of eclamptic cases were un heralded by hypertension and proteinuria.\(^1\) They stated that convulsions are one of a range of clinical features caused by endothelial cell damage secondary to placental ischaemia. The manifestations of the syndrome depend on the site and extent of endothelial cell damage and are not the result of a predetermined hierarchy. Convulsions may precede hypertension or proteinuria, as demonstrated in this case.

The aetiology of the pre-eclampsia complex of hypertension, proteinuria and convulsions is unknown. DIC may occur and its severity is thought to be proportional to the degree of pre-eclampsia. DIC is a result of unregulated release of free thrombin into the circulation.\(^2\) Tissue factor is released locally at the site of tissue damage and this complexes with factor VII which results in a cascade of enzymic reactions required for thrombin generation. Damaged placental endothelium is unable to concentrate antithrombin on its surface which normally would prevent build up of thrombin. This results in widespread microvascular thromboses leading to organ damage. To maintain vascular patency, excess plasmin is generated causing fibrinolysis and fibrinogenolysis. Generation of free thrombin and plasmin results in thrombotic and haemorrhagic manifestations, respectively, of DIC.

Additionally, free thrombin results in a change in platelets such that their surfaces bind and concentrate all the components of the haemostatic system. Platelet survival and numbers are reduced. Haemorrhage is the commonest manifestation of DIC because of generation of free plasmin and depletion of coagulation factors and platelets.

Eclamptic patients are prone to spontaneous haemorrhages and frequently show fibrin deposition on the basement membrane of hepatic sinusoids and arterioles. It has been postulated that sensitization of the reticuloendothelial system of the liver by previous subclinical pre-eclampsia may render it unable to clear fibrin thrombi from the circulation. As a result, infarction with vascular disruption may occur leading to intrahepatic haemorrhage and parenchymal destruction. Rarely the periportal necroses that result coalesce and form subcapsular haematoma and ultimately rupture of Glisson’s capsule with intraperitoneal haemorrhage.\(^1\) This may be exacerbated by diaphragmatic and abdominal muscular contractions but the underlying hepatic parenchyma is abnormal and a history of significant trauma can rarely be elicited.

Concerning the early diagnosis of spontaneous hepatic rupture, Howard and Jones claimed that the most important presenting complaint is right upper quadrant pain and that ultrasound scan is the quickest means of diagnosis, although computerized tomography is more sensitive.\(^4\) Our patient complained of lower central chest pain and an ultrasound scan demonstrated the presence of intraperitoneal fluid with mixed reflectivity around the hepatic area. Howard and Jones also stated that in such cases termination of pregnancy is the first step in the treatment and that surgery may not be required. They also stated that although elevated transaminase concentrations, decreased platelets and DIC occur
acutely, they resolve rapidly. This was not found to be the case with our patient who, despite aggressive medical and surgical treatment, continued to show signs of bleeding and DIC.

Loevinger, Lee and Anderson reported a patient in whom transcatheter gelfoam embolization of the hepatic artery was successful in arresting intrahepatic haemorrhage after hepatic rupture. They claimed that this is an alternative to surgery if the patient is clinically stable. We believe that our patient would not have been.

In a review of four cases between 1973 and 1976, Nelson, Archibald and Albo found that all four continued to require significant blood replacement of 4–25 u. after laparotomy for hepatic rupture. Erhard and colleagues reported the case of a 30-yr-old primigravida who developed hepatic rupture as a complication of the HELLP syndrome (haemolysis, elevated hepatic enzymes, low platelets) who successfully underwent urgent hepatic transplantation after a hepatectomy for bleeding and hepatic necrosis. Nelson, Archibald and Albo claimed that the lesion most often described is subcapsular haematoma of the right hepatic lobe with free rupture into the peritoneal cavity and resultant exsanguinating haemorrhage.

Hepatic rupture, although a rare complication of eclampsia, carries a high mortality rate. In some review series this was between 60 and 70%. Hence when the triad of pre-eclampsia, right upper quadrant pain and sudden hypotension is present in pregnancy or shortly afterwards, prompt recognition is essential. Ultrasound scan should be performed to aid diagnosis, and transplantation should be considered if medical and surgical intervention fails to control bleeding. Any woman who develops DIC during or after labour must be admitted to an intensive care unit or a high dependency unit for monitoring.

References