Perihepatic packing for spontaneous rupture of liver in pregnancy

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ABSTRACT
Spontaneous rupture of the liver is a relatively uncommon but potentially fatal complication of pregnancy generally associated with pre-eclampsia. This case report is of a grand multipara who presented with pre-eclampsia and was subsequently found to have spontaneous rupture of the liver. Bleeding was controlled effectively by the technique of perihepatic packing when other operative measures failed. Perihepatic packing is a well known technique advocated for the management of liver injuries and this may be successfully applied for cases of spontaneous rupture of the liver during pregnancy.

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Case Report. The patient was a 40-year old Saudi woman, gravida 12, who presented at 36 weeks of gestation with complaints of pain in the epigastrium and vomiting for the past 5 hours. On examination, she was fully conscious, febrile, pulse 80/min, blood pressure 230/120 mmHg with bilateral pedal edema. Examination of the chest and CVS was normal. The abdomen was soft with mild epigastric tenderness, no organomegaly. Bowel sounds were normal and there was no clinical evidence of intestinal obstruction. Uterus - 36 weeks size with no detectable uterine contractions. Cervix 1 cm dilated, not effaced, no liquor discharge, no vaginal bleeding. Ultrasound (U/S) examination revealed a normal 36 week pregnancy with single fetus, cephalic presentation with anterior placenta (Fundal Grade II - III).

Results of routine investigations were as follows: WCC 10.5 x 10^9/L, poly 52, lymph 44, EOS 4, hemoglobin 13.5gm/dL, ESR 63 mm/hr, blood group A+, RBS 6.4 mmol/L, urea 8.4 mmol/L, creatinine 71 umol/L, Na+ 137 mmol/L, K+ 4.0 mmol/L serum bilirubin 37.4 umol/L, alkaline phosphatase 148.5 units/L, SGPT 133 units/L, SGOT 138 units/L, serum proteins 61 gm/L, albumin 21g/L, serum amylase 74 units/L. Urinalysis: albumin ++, granular cast +, pus cells 4-5/hpf. Ophthalmoscopic examination: normal fundus, no retinopathy, no papilledema.

The patient was put on conservative management with IV infusion, IV tagamet 400 mg x 12 hrly, adelat cap 10mg PRN, aldomet tab 250 mg x 8 hrly, buscopan 1 amp IV PRN, and IV piperacillin 4gm x 6 hourly. Five hours after admission, she complained of severe pain over the right hypochondrium and abdominal examination revealed marked tenderness with guarding in the right hypochondrium. Repeat U/S showed a fluid collection in the right hepatorenal pouch. Conservative management was continued and four hours later, her blood pressure dropped to 100/80mmHg. This was associated with the disappearance of the right hypochondrial pain. However, she complained of lower abdominal pain and examination revealed tenderness over the uterus and fetal heart sounds were undetectable. Urgent U/S examination revealed no fetal heart activity or movements; no retroplacental bleeding was detected. Investigations carried out showed: Hb 9.6gm, HCT 29, WCC 15.8 x 10^9/L, PT 14"/13", PTT 32"/34", FDP>2000 ngm/mL, fibrinogen 300 mg/dL.

With blood transfusion in progress, artificial rupture of membrane was performed and labor was induced with syntocinon infusion. However, labor failed to progress and in view of evidence of internal hemorrhage, cesarian section was performed and a

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still born baby was delivered. Gross hemo-
peritoneum was present -3.5 L of blood being
evacuated. Further exploration revealed a massive
rupture of the capsule of right lobe of liver and a
smaller capsular rupture over the left lobe of liver.
Hemostasis was secured by pressure with hot packs
followed by diathermy and application of hemostatic
surgical. The abdomen was closed leaving drains in
the right subphrenic region and pelvis. Post-
operatively the patient continued to bleed and she
was re-explored after twelve hours when a large
hemoperitoneum (2.7 liters) was detected and
continuous oozing from the raw surface of the right
lobe of the liver was observed. Diathermy and
packing with surgical was found to be ineffective, so
perihepatic packing using four large abdominal packs
was performed. Subsequently, the general condition
of the patient improved with the sanguineous
discharge from the peritoneal drains decreasing
progressively. The perihepatic packs were removed
after three days when the liver surface was found to
be healing well with no further oozing. She made
good progress, though had mild chest infection post-
operatively and was gradually started on oral fluids
and normal diet. However, on the sixth post-
operative day after removal of the packs, she
collapsed suddenly on the ward and could not be
revived despite cardiopulmonary resuscitation;
presumably having suffered an attack of massive
pulmonary embolism.

Discussion. Abercrombie1 in 1844, first reported a
case of spontaneous rupture of liver and since then
over 130 cases have been described. Spontaneous
rupture of the liver is estimated to occur in 0.4 per
100,000 pregnancies and is associated with a very
high maternal and fetal mortality. Most of these
cases with ruptured liver are associated with severe
preeclampsia or eclampsia and hence occur in the
third trimester of pregnancy though no stage of
pregnancy is immune. In one series,2 out of 225
cases of pre-eclampsia, 7 had spontaneous rupture of
the liver. The triad of preeclampsia, right
hypochondrium pain and features of shock should
alert one to suspect hepatic rupture. Asymptomatic
subcapsular hemorrhage in the liver is found in 80% of
cases of preeclampsia or eclampsia.2

Many authors3 have attributed hepatic rupture to be
secondary to disseminated intravascular coagulation
(DIC) with pathological deposition of fibrin thrombi
in vessels and sinusoids leading to focal and later
confluent subcapsular hemorrhagic necrosis. However,
the association between the development of DIC and hepatic rupture remains unclear as in one
report,4 the development of DIC was seen in a
patient only after surgical repair of hepatic rupture.
The role of minor trauma in initiating, bleeding in
areas of hemorrhagic necrosis in a preeclamptic liver
is unknown. The preeclamptic state causes
generalized cellular derangement and dysfunction in
the parenchyma of different organs and it is possible
that in such situations even the minor trauma of
vomiting, convulsions or parturition may be
sufficient to initiate a hemorrhage. Henney et al5
described the entity of spontaneous hepatic rupture as
a biphasic syndrome. In phase I there is intrahepatic
or subcapsular hematoma formation without rupture
of the capsule. Once the capsule is ruptured the
syndrome enters phase II with life threatening
hemorrhage and surgery becomes mandatory.

These two phases of development of hepatic
rupture were distinctly manifested in the present
patient with progressive deterioration of clinical
symptoms and signs. At laparotomy identification
of the fears and arrest of hemorrhage remain the
primary aims. Various techniques have been
suggested for control of bleeding e.g., diathermy, use
of thrombin, collagen or use of cellulose to promote
hemostasis, hemostatic wrapping with vicryl suture
mesh8 hepatic artery embolization, hepatic artery
ligation.6 Hepatic resection is indicated only when
these conservative measures fail to control the
bleeding. Perihepatic packing with rolls of gauze is
being increasingly advocated for liver trauma.10

Smith et al11 found a survival rate 82% in 27 cases of
spontaneous hepatic rupture secondary to
pregnancy induced hypertension which were
managed by packing and drainage. On the other
hand, survival rate was only 25% in eight patients
undergoing hepatic lobectomy. Their
recommendation was that hepatic hemorrhage with
persistent hypotension unresponsive to blood
products should be managed by evacuating the
hematoma and packing the damaged liver and
draining the operative site. More aggressive surgical
techniques such as hepatic artery ligation or hepatic
lobectomy should be reserved for refractory cases.
In the present case, perihepatic packing was
successfully utilized to combat continuing
hemorrhage from the liver surface when other
conservative measures had failed.

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