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## SUBCAPSULAR HAEMATOMA OF THE LIVER IN A CASE ODISSEMINATED INTRAVASCULAR COAGULATION SECONDARY TO A BLEEDING PLACENTA PREVIA

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### ABSTRACT

The following case report is on a 43 year old lady who had presented with profuse antepartum haemorrhage due to a placenta praevia Type 4 anterior. Following hysterectomy she developed DIVC that was corrected with platelets and FFP. Subsequently on 7<sup>th</sup>. Post-op day she had complains of abdominal distension and right hypochondrium fullness. An ultrasound and CT scan was done which showed a large subcapsular haematoma of the liver. This was then aspirated.

Formation of a subcapsular haematoma following DIVC is a rare event. No case of subcapsular haematoma has been reported following DIVC from a bleeding placenta praevia. This case illustrates the pathophysiology of DIVC as well as the imaging modalities in the diagnosis of a subcapsular haematoma.

Spontaneous subcapsular haematoma of the liver is a rare event. In most of the reported cases it was most often associated with toxaeimias of pregnancy (2,4,5,6). No cases of spontaneous subcapsular haematomas have been reported following haemorrhage due to placenta praevia. The following report is of a case of Type 4 anterior placenta praevia who suffered excessive blood loss from ante and postpartum haemorrhage which eventually led to disseminated intravascular coagulation.

DIVC = Disseminated Intravascular Coagulation

FFP = Fresh Frozen Plasma

### INTRODUCTION

Spontaneous subcapsular haematoma of the liver is a rare complication of pregnancy. The underlying factor is hepatic necrosis, the most likely cause being vascular disease (particularly toxaeimias) and Disseminated Intravascular Coagulation (DIC). The incidence of liver

haematoma in pregnancy is not known. In most of the reported cases during pregnancy, it was most often associated with toxaeimias of pregnancy, other causes being fatty metamorphosis, hepatoma, diabetes and hypertension. Spontaneous subcapsular haematomas following hemorrhage

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secondary to a placenta previa has not been reported before. The following report is of a case of Type 4 anterior placenta previa who suffered excessive blood loss from ante and postpartum haemorrhage which eventually led to disseminated intravascular coagulation, and developed a subcapsular haematoma of the liver.

### CASE HISTORY

A 43 year old Gravida 8 Para 7 lady with a period of amenorrhoea of 26 weeks was admitted with sudden profuse per vaginal bleeding. She was having episodes of slight per vaginal bleeding for the past one month, though fetal movements were normal. There was no rupture of membranes nor was there fever. Her previous pregnancies were uneventful. On examination the patient was pale was otherwise stable with normal blood pressure and pulse. There was no evidence of pre-eclampsia of pregnancy.

Abdominal examination showed a uterus of 26 weeks. An ultrasound abdomen was done at admission and showed a single normal foetus with a Type 4 anterior placenta previa. Patient had an emergency Lower Section Caesarian Section (LSCS) on the 26th day of admission for persistent and profuse antepartum haemorrhage. However, immediately after the LSCS, she had massive postpartum haemorrhage and a haematological examination done showed prothrombin time to be raised i.e. 2.06, a low platelet count i.e. 87,000 E9/L and low fibrinogen levels i.e. 0.9G/L, consistent with that of DIVC. A total abdominal hysterectomy was done and the DIVC was corrected with platelets and fresh frozen plasma. On the 7<sup>th</sup> post-operative day, patient started to complain of abdominal distension and fullness over right hypochondrium. Chest radiograph revealed plate atelectasis in the right lung base. Ultrasound of abdomen showed a mixed echogenic mass inferior to the right lobe of the liver and anterior to the right kidney (Fig.1). This measured 14 x 10.5 x 8 cm. and was compatible with a large subcapsular haematoma of the liver.

A computed tomogram (CT scan) of the abdomen showed a large hypodense, well-defined, peripheral, subcapsular collection situated at the inferior aspect of the right lobe of liver. No enhancement of this collection was seen following intravenous contrast media. The CT value of this mass was 30 Hounsfield units (HU) which was consistent with that of blood (Fig.2). The haematoma was causing deformity of the liver. A diagnosis of a subcapsular haematoma was made.

Aspiration of this collection was done using a 16G branula under ultrasound guidance. This was done to relieve the patient of pain and discomfort. A total of 250cc of stale blood was aspirated. Following the aspiration, she was discharged well, and remained well on follow up 1 month later.



Fig.1. Ultrasound liver shows a large mass of mixed echogenicity inferior to the right lobe of liver.



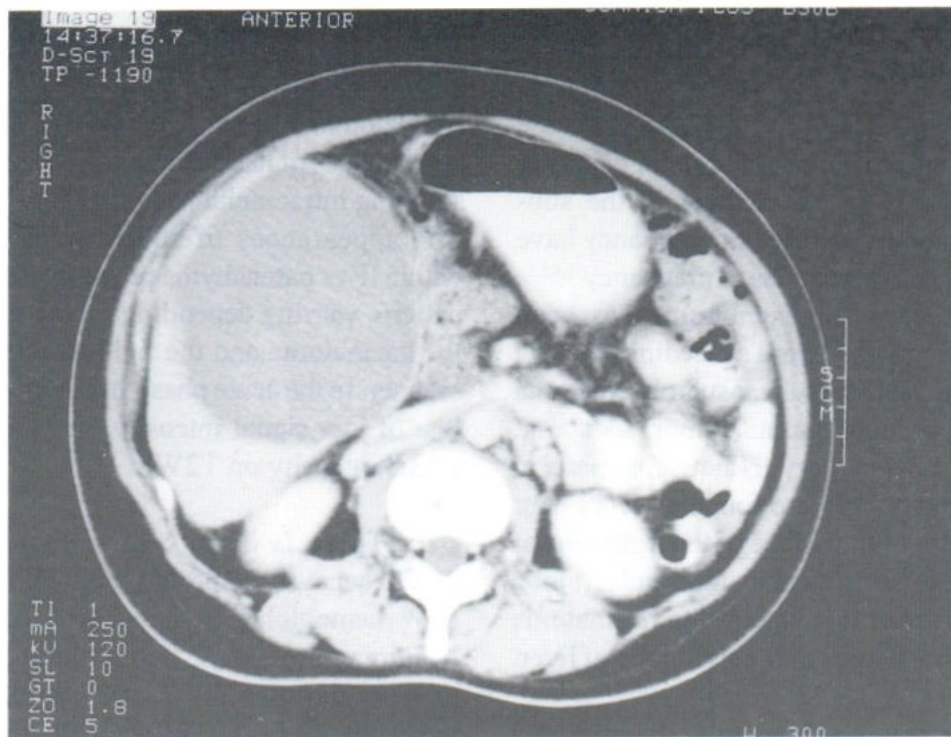


Fig 2. CT abdomen (axial) shows a non enhancing homogenous collection. The liver appears deformed.

## DISCUSSION

Disseminated Intravascular Coagulation is defined as defective haemostasis due to the activation of the coagulation system by recognised triggers leading to the consumption of clotting factors and platelets<sup>1</sup>. Common causes of consumptive coagulopathy during pregnancy are intrauterine fetal demise, sepsis, amniotic fluid embolism, induced abortion, pre-eclampsia or eclampsia, excessive bleeding and acute fatty liver.

The key pathophysiological derangement in most obstetric cases is hypofibrinogenemia as in this case. Activation of the coagulation system by recognized triggers leads to the consumption of clotting factors and platelets. Coagulation inhibitors are swamped and large amounts of fibrin are formed and deposited in the small vessels of multiple organs, including the kidney, lung and brain. Ischaemic tissue damage and micro-angiopathic haemolysis may result. In the liver,

tissue damage usually occurs at the periphery of the hepatic lobule especially involving the right lobe<sup>3</sup>. This accounts for the subcapsular haematoma that occurs.

Simultaneously, the fibrinolytic mechanism is activated to lyse the fibrin deposits. Thrombocytopenia, the depletion of coagulation factors, and the anticoagulant effect of fibrin degradation products all contribute to the continued bleeding that occurs in DIVC. Consumptive coagulopathy is usually manifested clinically by visible signs of disruption of normally intact haemostasis. These signs may include bleeding from venipuncture of intravenous access sites, gums, nose, vagina or rectum.

Haematuria, ecchymoses or petechiae and bleeding from surgical wounds or laceration may be evident. Subcapsular haematoma of the liver



and its rupture into the peritoneal cavity have been reported before<sup>3</sup>. However the incidence of subcapsular haematoma is not known. This case is unique as, a subcapsular haematoma occurring following DIVC is rare and most of the subcapsular haematomas reported in pregnancy have been associated with toxaeemias of pregnancy<sup>3,4,5,6</sup>. No case of subcapsular haematoma has been reported following DIVC from a bleeding placenta previa. Other causes of a subcapsular haematoma are penetrating or blunt trauma, tumor, aneurysm or abscess and post surgery. Ninety-five percent of hepatic haematomas were found to occur over the anterior or superior surface of the right lobe of liver<sup>3</sup>, other areas being caudate lobe and left lobe of liver. However in this patient the haematoma was at the inferior aspect of the right lobe of liver.

On ultrasound subcapsular haematoma appears sonographically solid with heterogenous echogenicity. Over time, cystic evolution of a subcapsular haematoma occurs. The haematoma retracts in size and becomes more cystic in appearance, often with multiple septations<sup>7</sup>. US is cheap, fast and has no radiation and its portability makes it ideal in the patient in Intensive Care Unit as well as for follow-up.

On axial CT of the abdomen subcapsular haematomas usually appear as crescent-shaped or lenticular well-marginated collections located just beneath the hepatic capsule. The density of the collection depends on the age of the haematoma, being of higher attenuation than non-contrast-enhanced liver early when clotted blood is present and decreasing in attenuation over time to low attenuation.

Most haematomas appear hypodense when compared with contrast-enhanced liver parenchyma, although freshly clotted blood usually remains hyperdense with enhanced liver parenchyma. It is also possible to quantify the amount of blood present in the pelvis as well as the amount of blood surrounding the liver<sup>8</sup>.

There has been no reported experience with magnetic resonance imaging (MRI) in subcapsular haematoma. Most knowledge concerning the MR appearances of haematomas has been from reports studying intracranial haemorrhage. Therefore the MR appearances of subcapsular haematomas within liver parenchyma probably follow the same pattern, varying depending on the time course of the haematoma and the field strength of the MR scanner. In the acute phase haemorrhage is usually that of low signal intensity on T1WI and higher signal intensity on T2WI.

There is often central hypointensity on T2WI because of preferential T2 shortening of deoxyhemoglobin. In the subacute phase, typically beginning around 72 hours, the methemoglobin present shortens the T1 of blood, resulting in hyperintensity on T1WI as well. This is initially seen at the periphery of haematomas and increases centrally over time. In the chronic haematoma a peripheral low signal rim may be seen as a result of formation of hemosiderin.

Other causes of post-operative right hypochondrium pathology that must be considered include liver abscess, Budd - Chiari syndrome, biloma, aneurysm and cholecystitis. However the US, CT scan and MRI features would be useful to differentiate between them.

The treatment of subcapsular haematomas of the liver is somewhat controversial. Left alone they may resolve spontaneously, expand and burst with delayed intraperitoneal bleeding, cause a hepatic abscess or decompress into the biliary tree and cause hematemesis<sup>1</sup>. With the imaging facilities available i.e. ultrasound, computed tomography and magnetic resonance imaging, nonoperative management of subcapsular or intraparenchymal haematomas has become popular i.e. aspiration of the haematoma is preferably done under CT or US guidance. The CT scan allows one to accurately assess the severity of the liver subcapsular haematoma.



In summary, this case illustrates a rare complication of DIVC induced by haemorrhage from a placenta previa. Clinical findings and laboratory investigations strongly raise the suspicion of DIVC. The diagnosis of a subcapsular haematoma was then established by ultrasound and CT scan following which treatment was initiated by aspiration of the haematoma under ultrasound guidance.

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