DEATH DUE TO HEMORRHAGIC SHOCK AS A RESULT OF RUPTURE OF SPLENIC VESSELS – NATURAL DEATH

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ABSTRACT: A continuous rise in the rate of deaths due to pregnancy related causes has been reported in many countries during the past decades. Maternal rupture of splenic vessels during pregnancy seems to be a rare disease whose treatment is not codified. This most feared complication occurs during the third trimester of pregnancy and result in a significant maternal and foetal morbidity and mortality. We examine a case to discuss various factors related to a fatal rupture of spleen vessels in from pregnancy

Keywords: Forensic pathology, autopsy, splenic vessels, hypovalemia and gamma gandy bodies

Brief History
A 21 year old full term primi complained of weakness while climbing the stairs and collapsed, was immediately rushed to a hospital but was declared brought dead. Her ante natal check ups were uneventful.

Autopsy Findings:
The dead body is that of a female aged about 21 years, moderately built and nourished. Post mortem staining was present over the back and rigor mortis was well appreciated over the body. The conjunctivae and nail beds were pale.

Eyes and Conjunctivae: Normal and pale.
Ears & Nose: Normal.
Neck: Normal
Trunk: Normal
Abdomen: Normal
Upper Limbs: No demonstrable Injuries
Lower Limbs: No demonstrable Injuries
External Genitalia: Intact

INTERNAL EXAMINATION

HEAD:
Skull: Intact.
Brain and Membranes: 1100 g. There was no intracranial haemorrhage. Section revealed no evidence of injury.
Spinal Cord: Intact
Mouth: Normal.
Tongue: Normal.
Pharynx: Normal.
NECK AND CHEST:
Walls & Ribs: Intact
Pleura: Intact
Larynx & Trachea: Intact
Hyoid Bone and Laryngeal Cartilages: Intact
Thyroid Gland: Intact

THORAX
Ribs and Sternum: Intact.
Pericardial Sac: Normal.
Heart: Intact and weighed 280 g. All the chambers were empty and there was no evidence of any abnormality or disease.
Pulmonary trunk: Healthy
Aorta: Healthy.
Pleural Cavities: Normal.
Larynx, Trachea and Main Bronchi: Normal.
Lungs: Both the lungs are intact and pale.

The left weighed 240 g. and the right lung 306 g. They were pale in colour. Dissection of the lungs revealed - Exudes scanty blood.

Oesophagus: Normal.

ABDOMEN:
Abdominal Cavity: An extensive blood clot was found in the lesser sac and across the upper abdomen.
Peritoneum: An extensive blood clot was found in the lesser sac and across the upper abdomen. Peritoneum contained about 2500ml of blood and blood clots, the clots being conspicuous over the left paracolic gutter tracking down from the hilum of the spleen into the mesentery.
Gastrointestinal Tract: Normal

Mouth, Pharynx & Esophagus: Intact
Stomach and its contents: Contains 500ml of partially digested food particles. Mucosa is pale. No unusual smell is present.
Small Intestine & its contents: Contains gas and its Contents
Large Intestine & its contents: Contains gas and its Contents
Liver: Intact
Spleen: The spleen was enlarged, measured 10 cm x 7 cm and weighed about 1000 grams. The surface showed multiple irregular grayish white scarring with a dense dark clot at the hilum. Upon dissection the splenic vein was ruptured and showed clots while distal part was torturous and dilated. (Figure No: 5)
Gall Bladder, Pancreas, Adrenal Glands: Normal.

GENITO-URINARY ORGANS
Kidneys: Intact and pale.
Urinary Bladder: Intact and empty
Organs of Generation
External organs of generation: Intact
Uterus: Uterus was gravid with a dead fetus of 9 months gestation.

Histopathology Report:
Macroscopy- Enlarged spleen with irregular scarring of the surface cut- section shows a single infarcted area measuring 3x3 cm.
Microscopy- Splenic tissue with enlarged and congested red pulp and relatively obliterated white pulp, with large areas of fibrosis in red pulp and fibrosiderotic nodules with calcification (Gamma Gandy bodies) and hyalinization was observed. Areas of healed infarct replaced by fibrous scar were also noted. An impression of congestive splenomegaly and acute tubular necrosis was made.

The cause of death

was opined based on the autopsy findings and histopathology report as due to hemorrhagic shock consequent upon rupture of splenic artery aneurysm. (Natural death).

COMMENTARY

The dead body had an exsanguinated look, conjunctivae and nail beds were pale, faint post mortem staining over the back, and all the internal organs were intact and pale. The uterus was gravid with a dead fetus of 9 months gestation. Peritoneum contained about 2500ml of blood and blood clots, the clots being conspicuous over the left paracolic gutter tracking down from the hilum of the spleen into the mesentery. Figure No 1. An extensive blood clot was found in the lesser sac and across the upper abdomen. The spleen was enlarged, measured 10 cm x 7 cm and weighed about 1000 grams; the surface showed multiple irregular grayish white scarring with a dense dark clot at the hilum. Upon dissection the splenic vein was ruptured and showed clots while distal part was torturous and dilated. (Figure 2). The cause death was ascertained as hemorrhagic shock as result of spontaneous rupture of splenic vein associated with massive splenomegaly during pregnancy (Figure-3-5).
Figure No. 3 showing tracking of clot from the hilum of the spleen to the lesser sac

Figure No. 4 showing enlarged intact spleen with multiple grayish scarring on the surface (Gamma Gandy Bodies)

Figure No:5 Enlarged spleen with irregular scarring of the surface
DISCUSSION

Infection, malignancy, metabolic disorders, as well as vascular and hematological diseases are known to cause splenomegaly which may rupture pathologically or due to trauma. Several reports have been published over the last century describing patients from tropical areas with massive splenomegaly. After excluding known causes of splenomegaly, tropical splenomegaly syndrome was defined as a separate entity. This condition was later defined as hyper reactive malarial syndrome (HMS). Genetic factors, pregnancy, and malnutrition may play a role in the etiology of HMS. [1,2, 3]. The common causes of hemoperitoneum in pregnancy are ruptured ectopic or abruptio placenta or ruptured uterus. However one of the rarest causes could be a rupture of splenic vein. Its consequences can be devastating for both mother and fetus.[4]. Sudden death from spontaneous rupture of the splenic vein is rare but few cases have been documented. [5]. Most commonly associated with pregnancy and are usually a catastrophic event. Splenic vein flows in the portal vein, portal hypertension would increase splenic venous pressure and thus increase intrasplenic microvascular pressure and fluid extravasation.[5]

Although the exact mechanism for hyper reactive malarial syndrome (HMS) is uncertain, evidence suggests that exposure to malaria elicits exaggerated stimulation of polyclonal B lymphocytes, leading to excessive and partially uncontrolled production of immunoglobulin M (IgM) as the initiating event resulting in splenomegaly. In this case the cause for massive congestive splenomegaly could be attributed to HMS as she was resident of a tropical malaria endemic area and further other causes for splenomegaly were ruled out. [6]. In spite of increasingly common reliance on abdominal ultrasonography and other radiological investigations during the antenatal checkup, clinicians uncommonly recognize any of the pathology with spleen and splanchnic circulation, which is further confounded by the absence of premonitory signs or symptoms which are most likely to be missed. Accordingly, rupture of a splenic vein and visceral aneurysm, including splenic artery aneurysm, typically presents as sudden, unexpected death. As a consequence, the initial recognition and diagnosis of bleeding from splenic vein rupture due to portal hypertension take place only at autopsy. As far the histopathology of congestive splenomegaly, the splenic tissue was with enlarged and congested red pulp with relatively obliterated white pulp with large areas of fibrosis in red pulp. It showed fibrosiderotic nodules with calcification (Gamma Gandy bodies) and hyalinization. An impression of congestive splenomegaly and acute tubular necrosis was made. These fibrosiderotic nodules with calcification (Gamma Gandy bodies) represent arease of organized hemorrhage in the spleen that is caused by portal hypertension. Gamma-Gandy bodies contain hemosiderin, fibrous tissue, and calcium. [7]

In the histopathological analysis of 23 (65%) cases of congestive splenomegaly, varied morphologic changes were found. There were hemorrhages, congestion of sinuses and fibrosis along with hemosiderin laden macrophages and gamma-gandy bodies. These changes in literature have been characterized as sclero-congestive or fibro congestive, implying that there is progressive fibrosis of the splenic cords due to the prolonged congestion and ischemia.8 Congestive splenomegaly may be accompanied by signs of hypersplenism along with fibrous thickening of the capsule, which is frequent. There is marked dilatation of veins and sinuses, hemorrhages, fibrosis of the red pulp and accumulation of hemosiderin containing macrophages. The lymphoid follicles become inconspicuous. Iron incrustation of the connective tissue and sclerosiderotic nodules Gamma–Gandy bodies develop as a result of focal hemorrhages. [8]. Figure no 4 shows the uterus was gravid with a dead fetus of 9 months gestation. Pregnancy especially third trimester is often associated with many vascular changes such as an increase in cardiac output and blood volume. These vascular changes along with the dilation of the veins and changes in venous pressure seem to be involved in the pathogenesis of rupture of the splenic vein. In this case, she had features of congestive splenomegaly and with advanced pregnancy could have caused vascular alterations which finally lead to weakening of the vein wall. Although rupture of spontaneous rupture can occur at any time during pregnancy and during the immediate postpartum period, 69% of cases are found in the third trimester. [9].

The cause death was ascertained as hemorrhagic shock as result of spontaneous rupture of splenic vein associated with massive splenomegaly during pregnancy. Living in tropical malaria endemic area, portal hypertension, congestive splenomegaly and advanced pregnancy are considered to be potential risk factors. In most cases, the exact cause of a spontaneous rupture splenic vein is unknown. However, whatever the etiology, the ultimate pathology is local failure of the connective tissue of the vessel wall to maintain the integrity of the vessel. [9]. While Potential risk factors include portal hypertension, congestive splenomegaly, congenital abnormalities of the vessels, inherited vascular and connective tissue disorders, vascular trauma, inflammatory processes, and degenerative arterial disease. [10, 11].
CONCLUSION
In this case the woman was in term pregnancy, collapsed and died suddenly, splenomegaly was attributed to hyper reactive malarial syndrome (HMS). The cause for massive hemoperitoneum was due to the spontaneous ruptured splenic vein. It is essential that obstetricians are alert to the prodromal and catastrophic symptoms of splanchnic circulation. A high index of suspicion, early recognition, and prompt management are vital to the survival of mother and fetus.

REFERENCES


