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Case Report

Forensic Section

The Portal Hypertensive Gastropathy: A Case and Review of Literature

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ABSTRACT

Upper gastrointestinal bleeding is a cause of high risk for morbidity and mortality. It has been debated in alcoholic cirrhosis, if alcohol exerts an exclusive and causal role upon gastropathy or whether it is linked to cirrhotic portal hypertension. The authors describe an autopsy report regarding mortality caused by gastric bleeding in a 53-year-old patient who suffered from cirrhosis. Literature has evidence of direct, marked damage of alcohol upon the gastric mucosa and there is noteworthy statistical data implying the revaluation of the pathogenesis of the bleeding.

Keywords: Alcoholic cirrhosis, Cirrhotic portal hypertension, Gastrointestinal haemorrhage

CASE REPORT

Autopsy was conducted on a 53-year-old man who died as the result of cardio-circulatory insufficiency caused by an acute gastric bleeding, ulcer and alcoholic cirrhosis. The patient was a bricklayer and was unemployed since about six months. He had a history of about 10 years of alcoholism. He also had complaints of gastric ulcer and was on pharmacologic treatment since the last year, but he did not have any respite. His family members had reported of irrational behaviour, often aggressive and violent, in the last month of his life. He lived alone in a house as a tenant.

He was 178cm tall and weighed approximately 73kg. His general health condition was registered as poor. It was noted that his adipose tissue showed a regular distribution and a diminished muscle trophic condition.

The skull did not show any appreciable preternatural motility of the bone surface. Patches of ECG recordings on the chest showed no preternatural motility of the bone surface, suggesting the absence of trauma. The examination of the thanatological phenomena showed pink hypostasis in dorsal surface of the body and upto the posterior axillary line due to the haemorrhagic blood loss and complete resolution of the stiffness in all joints. No putrefactive phenomenon was observed.

After dissecting the soft tissue, the rib cage was found to be fully intact, diaphragm and bowel loops were normal, with a paucity of limpid liquid in the abdominal cavity.

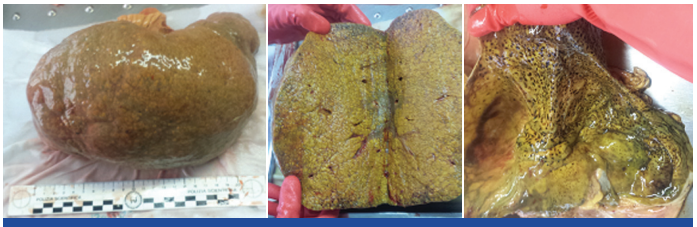
Upon removal of the sternal plastron, the lungs were expanded, without liquid in the pleural cables and adhesions in the basal sides. The pericardium was intact.

The heart had a globular form and an increased size (520 g weight, 11.2 cm longitudinal diameter, 12.6 cm transverse diameter, 4.1 cm anteroposterior diameter). The thickness of the wall of the left ventricle was approximately 1.4 cm whereas the interventricular septum wall was 1.1 cm. In the longitudinal section, myocardium had a pale appearance.

The lungs showed increased dimensions and consistency (right lung weight 785g - left 672g) and were cracked. The large, medium and the small bronchi had mucosal hyperaemia. In the full-thickness section of the lungs, the parenchyma was congested, an observation corroborated by squeezing that generated of frothy bleeding. This was indicative of pulmonary hypertension due to heart failure.

The liver weight was increased (2200g) and had a nodular surface. The cut surface was plainly cirrhotic [Table/Fig-1,2].

The internal surface of the gastric mucosa was not perfectly discernable, however it appeared as "snake-skin" (putrefactive changes) and there were dark red spots (not ‘cherry red’ as in the living individual) [Table/Fig-3]. The other organs did not show any significant of pathology.



[Table/Fig-1]: Macroscopic view of the liver. **[Table/Fig-2]:** Cut surface of the liver. **[Table/Fig-3]:** The internal surface of the stomach.

The gastric bleeding may be explained by the direct influence of the alcohol on the gastric mucosa as well as the cirrhosis and the subsequent Portal Hypertensive Gastropathy (PHG). The aggressive gastrointestinal bleeding seems to have caused cardio-circulatory failure to the extent of involving the lungs, which presented as typical oedema and congestion leading to the pulmonary hypertension.

DISCUSSION

Upper gastrointestinal haemorrhage affects 100-150/100,000 adults each year, resulting in the death in 6-14% of cases [1]. The risk of morbidity and mortality is related to the initial loss of blood, causative disease and the patient's age [2]. It causes about 300,000 hospitalizations/year in the USA and the causes can be many and even the gravity, from insignificant forms of bleeding to life-threatening forms: peptic ulcer and esophageal varices are the most common causes in the USA. UK accounts for up to 70,000 acute hospital admissions each year and gastritis, Mallory Weiss tears, peptic ulcers, and gastroesophageal varices are the common causes [3].

Esophageal varices are the most frequent causes in the patients with liver cirrhosis and may also be jejunal, [4], ileal [5] and in cecum [6]. Nevertheless, the literature indicates that the stomach and gastritis are an important source of bleeding in these patients [7].

PHG represents a group of gastric mucosal lesions that cause gastrointestinal bleeding in patients with Portal Hypertension (PH) of any aetiology. The incidence is very variable (4-80%) [8]. There is a lack of consensus concerning endoscopic diagnostic criteria,

especially with regard to the role of the risk factors [9]. In the case of alcoholic aetiology of cirrhosis, for example, the question arises as to whether or not alcohol can be the exclusive inducer and have a direct causal role on gastropathy or whether it requires a concomitant cirrhotic PH.

The authors have presented an autopsy report of expiration by gastric bleeding in a patient presenting cirrhosis.

The traditional hypothesis, published in an interesting article by Geraghty et al., is that the increased pressure in the portal vein, due to the increased resistance to portal venous flow secondary to hepatic fibrosis, is associated with a stagnant gastric microcirculation which renders the mucosa of the organ most susceptible to injury by alcohol [10].

However, the PH concept maintains that the stomach is an organ passively congested is at odds with haemodynamic studies. These show that in PH there is a hyperdynamic circulation state with an increase in the gastric flow and, hence, a reduction of susceptibility of mucosa to damaging agents [11].

In a study of the factors associated with PHG and gastritis, there are no associations between these two clinical conditions to be found although it is reported in the literature, thus it may be argued that the development of PHG cannot be linked exclusively to cirrhosis [12].

Just as controversial is the reference to the potential relationship between the severity of liver disease (cirrhosis) and the development of PHG, many authors have found no co-relation between PHG and severity of liver disease. For example, in a study of Curvello et al., no association between hypertensive gastropathy and severity of liver disease (assessed according to the classification of Child) could be found [13].

In a further study on 1,500 patients who underwent gastroscopy, no statistically significant co-relation was found between the injury and alcohol intake, but an association was between the incidence and severity of gastritis with age [14].

The gastric ectasia is probably the first lesion that appears in alcoholics as a direct consequence of the insult of alcohol on the gastric mucosa. In fact, alcohol excess affects the stomach through different mechanisms. The first mechanism to be taken into account is oxidative stress, which affects the gastric cells following an acute bout of alcohol intoxication, about which there is still much to be understood. In fact, under normal conditions the production of Reactive Oxygen Species (ROS) is well-balanced due to the presence of antioxidants. Alcohol abuse disrupts this balance with consequential damage to the mucosa [15]. Furthermore, the presence of alcohol promotes neutrophils infiltration of the stomach with accompanying synthesis and release of myeloperoxidase, which, through the oxidation of chloride by hydrogen peroxide, results in the formation of hypochlorous acid (HClO), a weak acid, which is another ROS- damaging agent for mucosa [16]. Another mechanism is linked to the mitochondrial activation: in response to an oxidative stress with an increase in heat shock protein, there is an increase in the permeability of the outer mitochondrial membrane which releases a caspase-activating factor triggering the apoptosis [17]. Finally, alcohol exerts the antagonist systems of ROS causing a consequently increased antioxidant activity (the inhibition of the synthesis of prostaglandins known as gastric protectants).

Many authors agree that the erosive gastric disease is a short term effect of alcohol abuse while the natural evolution is towards the atrophy of the stomach mucosa by the degenerative action on cells [18]. The bleeding, lastly affects the heart as in other haemodynamic causes [19,20].

CONCLUSION

The cause of acute haemorrhagic gastritis in alcoholic cirrhosis should not only be attributed to PH, but the role of alcohol on the mucosa should also be considered.

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