G56 Dissecting Intramural Hematoma of the Esophagus: A Rare Case of Sudden Death

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After attending this presentation, attendees will learn that dissecting intramural hematoma of the esophagus is a rare condition with excellent prognosis when treated conservatively. Spontaneous ruptures of hematoma are rarely described as well as sudden death due to DIHO.

This presentation will impact the forensic science community by presenting the rarity of the fatal events due to DIHO and the autopsy technique performed in visualizing rupture, preserving anatomical relationship between cervic-thoracic organs.

Dissecting intramural hematoma of the oesophagus (DIHO) is a rare condition in which intramural hemorrhage leads to submucosal

dissection of the oesophageal wall. It is usually associated with a rapid increase in intraoesophageal pressure, trauma or a coagulation disorder. The clinical presentation is with chest pain, hematemesis and dysphagia/odynophagia and an accurate history is vital to help distinguish it from other causes of acute chest pain, such as myocardial infarction, aortic dissection or oesophageal perforation. The three different types of acute oesophageal injury are a mucosal tear (Mallory– Weiss syndrome), full-thickness rupture (Boerhaave's syndrome) and dissecting intramural hematoma. Neither the Mallory-Weiss nor the Boerhaave lesions are associated with submucosal hematomas or dissections. In some cases the first event may be hemorrhage into the submucosa with secondary rupture into the lumen. The differential diagnosis includes other causes of central chest pain and it is vital to obtain an accurate history of both gastrointestinal and cardiovascular symptoms. Analysis of the precipitating factors suggests that there are three main subgroups. Firstly, a sudden pressure change in the oesophagus (e.g., swallowing, vomiting) perhaps associated with a temporary disruption in the normal co-ordinated opening mechanism of the upper and lower oesophageal sphincters. Secondly, direct injury following an endoscopic therapeutic intervention (e.g., oesophageal dilatation). Thirdly, about one fifth of patients appear to have a truly spontaneous origin although this may be associated with an underlying predisposition to abnormal pressure changes within the oesophagus (e.g., achalasia) or a bleeding disorder (e.g., due to anti-platelets, anti- coagulants or thrombolytics). The pathophysiology is characterized by submucosal hemorrhage that dissects the submucosa and classically occurs in the distal oesophagus because this region is least supported by adjacent structures such as the trachea or heart.

A rare case is presented of sudden death due to spontaneous rupture of DIHO occurred in a 42-year-old woman presented at local emergency department with a 24 hour history of sudden onset severe central chest and interscapular pain associated with dysphagia and odynophagia. There was no history of vomiting, hematemesis or trauma. There was little previous medical history of note and he was not taking any regular medication. On examination, vital signs were: blood pressure, 104/49 mmHg with no differential between arms; pulse, 125 beats/min; respiratory rate, 24 breaths/min; body temperature was normal. There was no abdominal tenderness and no maelena. EKG was unremarkable as well as cardiac enzymes. Clinical conditions suddenly got worse; the woman collapsed and resuscitation maneuvers were unsuccessful. Autopsy was performed the day after death. Massive hemothorax was recorded. Thoracic and abdominal organs were removed en masse according to Letulle technique and fixed in 10% buffered formalin for a detailed macroscopic examination. All other organs examination was unremarkable except for cerebral oedema. Vessels were poor of blood. Lungs were increased in volume and size, with few subpleural hemorrhagic spots. Mild white foam on the main bronchi was also detected. Heart was normal in size and volume, with conical shape. Coronaries examination was unremarkable. A large bluish/red intramural haematoma of the posterior wall of the oesofagus extending from just below the cricopharyngeous to the gastro-oesophageal junction was recorded with a complete rupture of the oesophagus wall in the proximal third. Mild cerebral odema and focal pulmonary oedema were observed at histological examination with standard H&E staining. Histological examination of heart was unremarkable except for few foci of contraction band necrosis. Sample of oesophagus dissection was collected excluding recognizable abnormality in the muscle layers, except for rupture. A complete immunohistochemical panel has been performed on esophagus samples. Genetic investigations had been performed also.

DIHO, Spontaneous rupture, Sudden Death

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