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Demonstration of Esophageal Varices Postmortem by Gastroesophageal Phlebography

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ABSTRACT: The study presents a method for the demonstration of esophageal varices at autopsy by gastroesophageal phlebography. The veins visible on the radiographs were controlled and evaluated by studying the gross appearance of sections of the esophagus at the level of the varicose veins and by histologic examination of the sections. The phlebography was found to be useful in estimating the varicose and normal veins of the esophagus. The study indicates further that the degree of varicosity is not always proportional to the severity of cirrhosis of the liver, and there may be varicose esophageal veins in patients with liver or heart disorders without hepatic cirrhosis.

KEYWORDS: pathology and biology, cardiovascular system, X-ray analysis, phlebography, esophageal varices, postmortem examinations

A trouble at autopsies is the demonstration of varicose veins of the esophagus and of possible ruptures of the veins. Common methods are injection of saline or colored fluid into the varices or examination of the esophagus under water [*1*]. Compressing the esophageal veins by moving one's thumb up and down them may cause blood to seep out from a rupture point [*2*]. In this study, varicose veins of the esophagus and cardia are demonstrated with phlebography at the autopsy. Histological studies of the esophagus and cardia were performed to see whether all or just a few varicose veins are visible on the radiographs, to control the size of the veins and their location in the esophageal wall, and to estimate the changes of the walls of the varicose veins. The study includes subjects with heart and liver disorders without liver cirrhosis to get an idea of the appearance of esophageal varices in those persons.

Subjects and Methods

The study included ten deceased persons, three of whom had hepatic cirrhosis, five had hepatic steatosis without cirrhosis, one had hepatic hemocongestion, and one had a liver of normal appearance. Among the persons with cirrhosis and steatosis, five had a cardiac disorder and three had had hematemesis immediately before their death (Table 1).

The inner organs were removed as a whole. The intestines, heart, lungs, and kidneys were separated from the set which was to be used for phlebography.

For each phlebography, Silicon-Kautschuk RTV (Vergussmasse K, Wacker-Chemie GmbH, München) and Blei (IV)-oxid reinst (Art. 7406), Merck, in proportions of 80 and

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TABLE 1—*Persons subjected to study.*

No.	Age, yrs	Sex	Cir-rhosis	Stea-tosis	Con-gestion	Heart Failure	Hematemesis	Cause of Death
1	85	fem	−	−	−	−	−	femoral fracture
2	58	male	+	+	−	−	−	cerebral hemorrhage
3	38	fem	−	−	+	−	−	overdose of drugs
4	51	male	−	+	−	+	−	myocardial fibrosis
5	56	male	+	+	−	+	−	liver cirrhosis with icterus
6	60	male	−	+	−	+	−	myocardial fibrosis
7	67	male	−	+	−	+	−	coronary heart disease
8	47	fem	+	+	−	−	continuous	gastrointestinal bleeding
9	55	male	−	+	−	+	slight	cardiac infarct
10	42	male	−	+	−	−	sudden massive	gastrointestinal bleeding

20%, respectively, were mixed at high speed. Of this silicone rubber-lead oxide mixture, 480 g was combined with 400 g of silicone oil AK 100 (Wacker Silicone) and 16 g of 2% Haerter T solidifier (Wacker Silicone).

The great veins of the liver, spleen, and mesentery were closed in the vicinity of the root of the left gastric vein. A 2.5-cm-wide 50-cm-long plastic tube on a holder was fastened with a plastic adapter to the left gastric vein. From the tube the contrast mixture was instilled into the venous system with a pressure of 60-mm Hg. The root of the left gastric vein was closed just before the tube ran empty, and the mixture in the veins was left to solidify for 90 min. After that, the liver, spleen, pancreas, and aorta were removed. The esophagus and cardia were opened along the anterior line and greater curvature, respectively, and the gastric contents were set aside for later examination. A radiograph was taken of the esophagus and ventricle at this point, and another was taken after the esophagus and ventricle had been cleaned of all surrounding soft tissue outside the muscular layers. Photographs were taken of the esophageal and gastric lining before the organs were put into a formalin bath. After fixation, sections from the middle and lower thirds of the esophagus and the upper part of the cardia were taken for gross and histological study. In each case, the liver was studied histologically.

TABLE 2—*The appearance of esophageal and cardiac varices in the subjects.*

No.	Intrinsic and Periesophageal Varices, Their Diameter (mm) and Length (cm) on Radiographs	Paraesophageal Varices, Diameter (mm) on Radiographs	Cardiac Varices, Their Diameter (mm) on Radiographs
1	none	none	none
2	none	none	single, 1 mm
3	none	none	none
4	none	single, 2 mm	single, 1.5 mm
5	none	single, 2.5 mm	multiple, 2 mm
6	single, 1.5 mm, 11 cm	single, 2 mm	single, 2 mm
7	none	single, 1.5 mm	single, 1.5 mm
8	multiple, 2 mm, 12 cm	multiple, 2 mm	multiple, 2.5 mm
9	multiple, 1.5 mm, 6 cm	multiple, 2 mm	single, 2 mm
10	multiple, 2 mm, 13 cm	multiple, 1.5 mm	multiple, 2 mm

Results

Table 2 shows the presence of esophageal and cardiac varices in the subjects studied. The terms intrinsic, periesophageal, and paraesophageal veins have been used in accordance with Hoevels et al. [*3*], that is, intrinsic veins are those in the subepithelial and submucosal layers, periesophageal veins are those in the connective tissue surrounding the esophagus, and paraesophageal veins refer to the mediastinal veins overlying the esophagus.

On the first radiograph taken in each case, the intrinsic, periesophageal, and paraesophageal veins are pictured. On the second radiograph the paraesophageal layers have been removed (Fig. 1).

In this study, straight or slightly sigmoid veins with a diameter below 1 mm were not regarded as varices. All the varices were of Grade 1 or 2 in the five-grade classification suggested by Dagradi et al. [*4*].

By comparing the radiographs with histological sections of the esophagus, it was revealed that esophageal veins with a diameter well below 1 mm were invisible on several radiographs at any level of the esophagus.

At sections obtained from the upper aspects of the middle third of the esophagus, the veins that were filled with contrast mixture constituted 0 to 13% of all veins. None of these veins was 1 mm or more in diameter. At sections from the lower aspects of the middle third of the esophagus, 55 to 80% of the veins were filled with contrast fluid, and at sections from the lower third of the esophagus, 90% of the veins or more were filled. All of the unfilled veins were below 1 mm in diameter. In the lower third a maximum of 25% of all veins were located in the muscular layer (Case 8, Figs. 2 to 4). In the same case, all the veins with a diameter of 1 mm or more were located in the submucous or subepithelial layers.

At sections from the esophagogastric junction, 90% or more of the veins were filled with contrast mixture. In all cases with varicose veins, the varices were located in the submucous or subepithelial layers at this level.

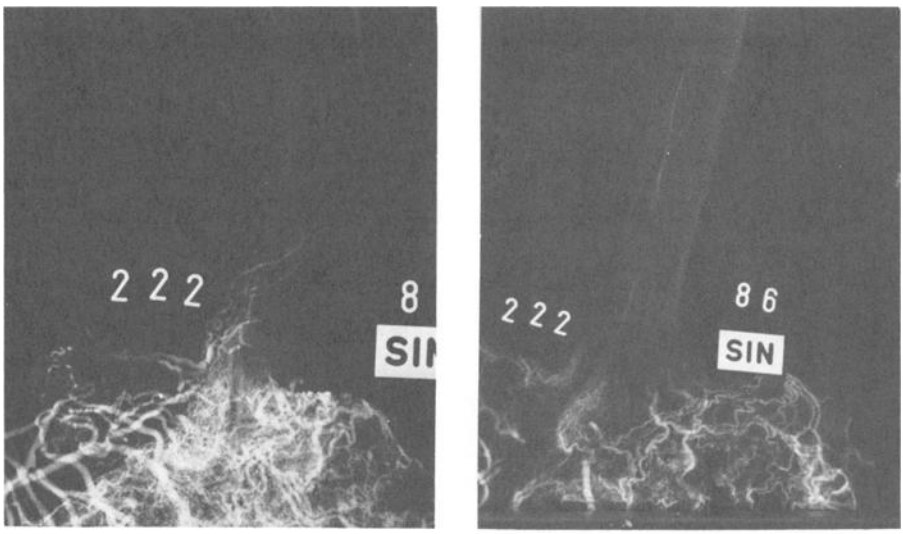

FIG. 1—(a) *The intrinsic, periesophageal, and paraesophageal varices of Case 6.* (b) *An intrinsic varix. The paraesophageal layers have been removed. The subject had steatosis of the liver and a hypertrophic heart, but no cirrhosis.*

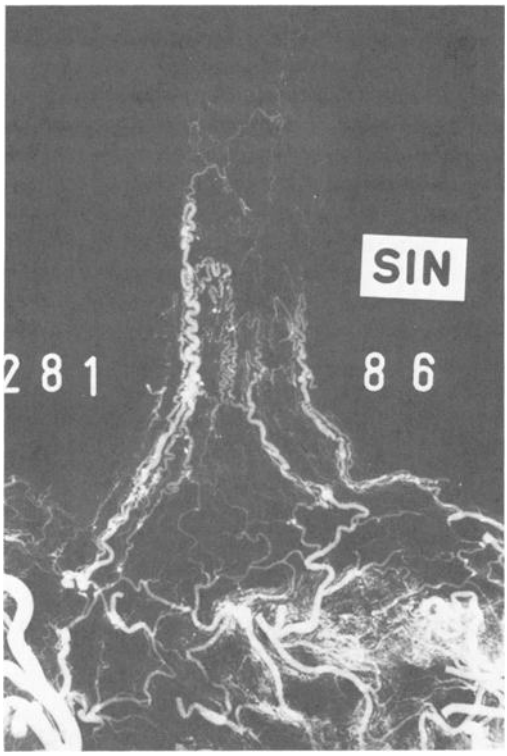

FIG. 2—*Esophagogastric varices of Case 8. The paraesophageal layers have been removed before X-raying.*

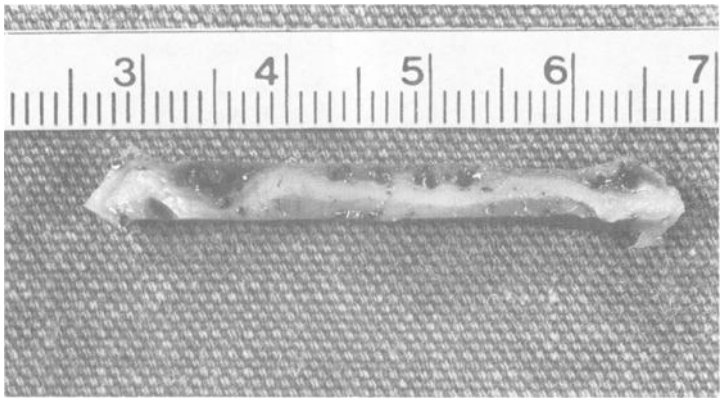

FIG. 3—*Sections from the lower third of esophagus of Case 8.*

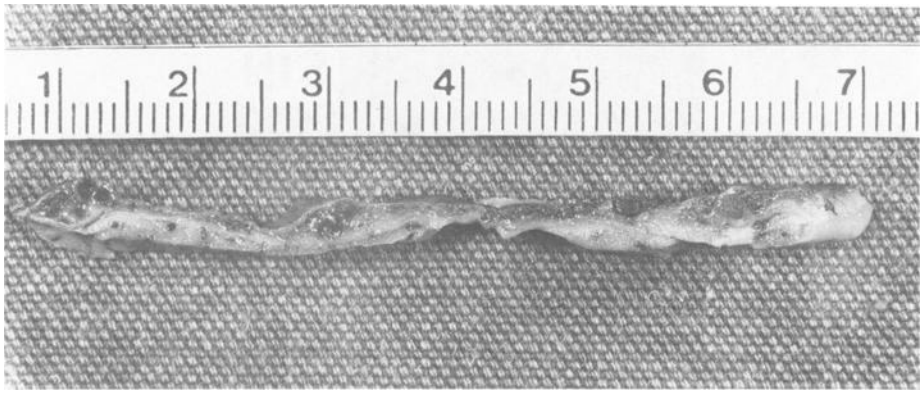

FIG. 4—*Esophagogastric junction shows the location of varices (subepithelial and submucosal layers) and undistended veins (all layers of vessel wall) of Case 8.*

The rupture of a varicose vein could easily be demonstrated in one case (Case 9, Figs. 5 to 7). In the two other cases with hematemesis, contrast mixture did not seep out through the ruptures, but the ruptures could be demonstrated at close examination of the esophagogastric lining. The histological examination of the walls of the varices revealed varying degrees of fibrosis of the subendothelial layers, with loss of elastic fibers in the walls.

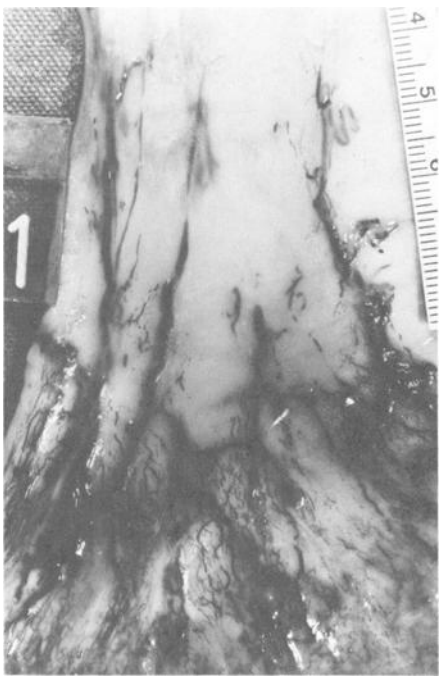

FIG. 5—*Appearance of varices of Case 9 on photograph of esophagogastric mucosa with contrast medium in veins. A rupture is located in the left varix next to the frosted upper part of the glass carrying the digit 1.*

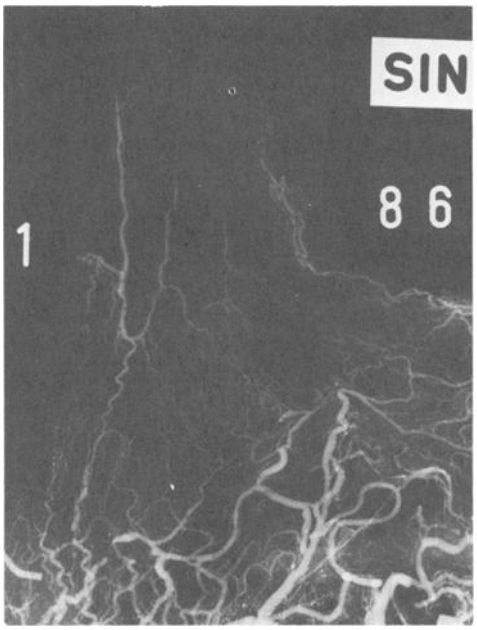

FIG. 6—*Appearances of varices of Case 9 on radiograph of esophagogastric mucosa with contrast medium in veins.*

FIG. 7—*Section from lower third of esophagus of Case 9.*

Among the subjects, Case 2 had hepatic cirrhosis but apparently only one small cardiac varix. Case 6 had a 40% hepatic steatosis and a hypertrophic heart (400 g) without coronary disease. With him, single varices were found in the esophagus, paraesophageal tissue, and cardia. Case 9 had a 70% steatosis of the liver, a hypertrophic heart (625 g) with some 90% stenosis of the coronary arteries. He had multiple esophageal varices. Case 10 had a 50% steatosis but no cirrhosis and no heart disease. He had multiple esophageal varices and his cause of death was apparently gastrointestinal bleeding (Tables 1 and 2, Figs. 1 through 8). The degree of varicosity seems not always to be positively related to the severity of cirrhosis of the liver, and there may be esophageal and cardiac varices in patients with liver or heart disorders without hepatic cirrhosis.

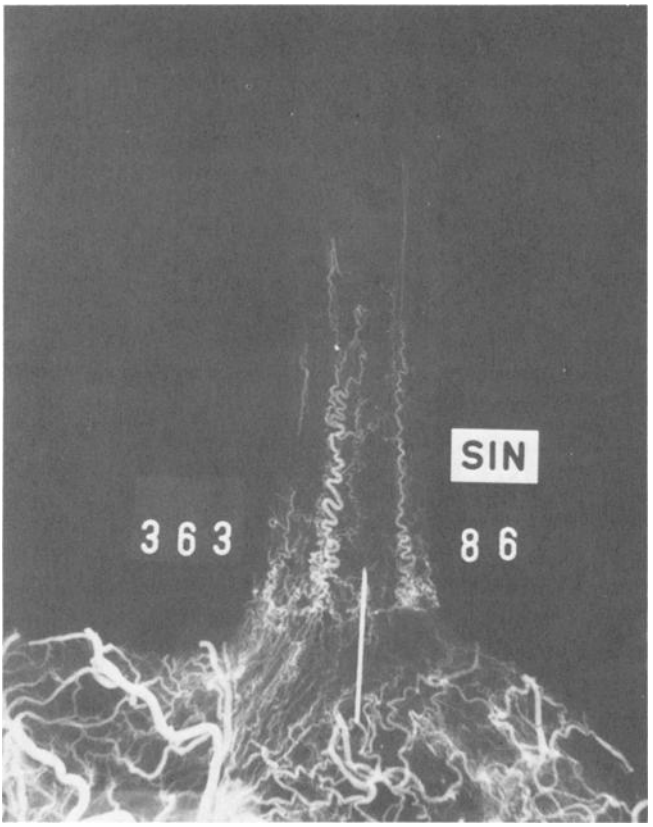

FIG. 8—*Large tortuous varices of Case 10. A rupture was found at the esophagogastric junction. The upper point of the needle indicates the site of a small vessel pouch with blood clot which may have interfered with passage of contrast medium in the vein.*

Discussion

The causes of esophageal varices are the causes of portal hypertension. Rezek and Schwartz [*1,5*] list causes of portal hypertension. Among these, the intrahepatic causes other than cirrhosis and the suprahepatic causes with heart failure and myocardial fibrosis are common disorders and the patients suffering from these may be a risk group worth attention in regard to esophagogastric varices. Sometimes no apparent cause of portal hypertension and bleeding esophageal varices can be found.

Burcharth et al. [*6*] found that the size of esophageal varices was related to the size of the coronary and short gastric veins and to the portal pressure. The size of gastric varices was related to the cephalad collaterals from the spleen and splenic vein, but not to portal pressure.

With living patients there are hemodynamic problems such as hepatofugal and hepatopetal blood flow and portal pressure to pay attention to in estimating the reliability of the number of varices visible on the radiographs [*3*]. Thrombosis of the varices occurs [*1*] and may make the varix difficult or impossible to recognize at phlebography. Thus, also in postmortem examinations, negative findings at esophageal phlebography do not rule out varices.

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