A Case of Downhill Esophageal Varices associated with Superior Vena Cava Syndrome due to Mediastinal Mass

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ABSTRACT

Here we report a case of downhill esophageal varices who complained of weight loss, abdominal pain after meals, fatigue, anorexia, dizziness and swollen face for 2 months.

Abbreviation: SVC: Superior vena cava.

Keywrods: Downhill esophageal varice, Superior venacava syndrome, Mediastinal mass.


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INTRODUCTION

Downhill esophageal varices have been described by Israelski and Simchowitz in 1932. The term ‘downhill’ indicates direction of blood flow and should be distinguished from conventional ‘uphill’ varices; those are due to portal hypertension. Various cases of downhill varices have been reported from 1932 till now.1 Isolated proximal esophageal varices (downhill varices) usually develop from obstruction or compression of superior vena cava (SVC). Various benign or malignant causes are known.1,2 ‘Downhill’ varices are usually seen with SVC syndrome and is due to mass effect of lung cancers, intrathoracic goiter, mediastinal lymphoma, thyroid carcinoma, thymoma, mediastinal lymphadenopathy secondary to different head, and neck cancers, such as carcinoma of the tongue.2 Behcet’s disease, systemic venulitis, thyroid disease or a history of thyroid surgery, fibrosing mediastinitis, as a complication of upper extremity hemodialysis access, Castleman’s disease (angiofollicular lymph node hyperplasia), muscular constriction of abnormal extensions of posterior hypopharyngeal veins and venous obstruction as a rare late complication after correction of congenital heart defect and are unusual causes of downhill esophageal varices, as interestingly, a patient with liver cirrhosis have also been reported.2-4 Downhill varices were reported with up to 50% with mediastinal pathologies.5 In downhill varices, bleeding is very rare complication and does not require endoscopic treatment.4 In case of bleeding, endoscopic band ligation or angiographic treatment with systemic embolization through brachiocephalic vein and sclerosing agents injection is recommended.2,7

CASE REPORT

A 53-year-old male patient admitted to internal medicine outpatient service with weight loss (had lost 8 kg within 2 months), abdominal pain after meals, fatigue, anorexia, dizziness and swollen face in morning. His complaints had been for 2 months. There was history of 224 gm/day alcohol consumption and 25 packs/year smoking habit for 15 years. There was no history of jaundice, hepatitis and familial liver disease. In the laboratory investigation, the findings were as follows: hemoglobin: 14.86 gm/dl, white blood cell: 8300/mm3, platelet counts: 162000/mm3. The levels of aspartate aminotransferase, 23 IU/l; alanine aminotransferase, 21 IU/l; gamma-glutamyl transpeptidase, 27 IU/l; total bilirubin: 0.93 mg/dl; alpha-fetoprotein, 1.3 ng/ml; calcium, 19-9: 5.76 U/ml; ferritin, 60.9 ng/ml; transferrin saturation 25% were within normal range. The patient was negative for hepatitis B surface antigen and antibody to hepatitis C virus but expressed antibody to hepatitis B surface antigen. Prothrombin time was 11.5 seconds and albumin level was 4.2 gm/dl. The levels of ceruloplasmin and alpha-1 antitrypsin were 31.5 and 201 mg/dl respectively. Endoscopic examination was performed due to symptom of dyspepsia. Upper gastrointestinal endoscopy revealed proximal esophageal varices (Fig. 1). Distal esophagus, stomach and duodenum had normal appearance. At the physical examination, superficial venous collaterals were obtained in upper extremity and thorax, thought to superior vena cava (SVC) syndrome.

Fig. 1: Endoscopic view of proximal esophagus varices
Computed tomographic angiography revealed $59 \times 47 \times 80$ mm mass at right anterior mediastnum, also left brachiocephalic vein and SVC were not visualized, which may be due to thrombosis (Fig. 2). Azygos veins were dilated. Mediastenal and paravertebral venous collaterals were seen. Collateral venous structure was seen in anterior abdominal wall and anterior part of the liver. Inferior vena cava has been filled with azygos vein. Portal vein was normal with Doppler ultrasonography. Mediastinoscopy was performed. Histopathologic examination of specimen revealed poorly differentiated carcinoma, squamous cell carcinoma.

REFERENCES


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