CASE REPORTS

Base of tongue varices associated with portal hypertension

P Jassar, M Jaramillo, D A Nunez

Abstract

A symptomatic case of tongue base varices in a patient with portal hypertension secondary to liver cirrhosis is presented. There are no previously documented cases in the world literature. Oesophageal varices may not be the only source of expectorated blood in a patient with portal hypertension.

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Keywords: portal hypertension; lingual; tongue; varicose vein

Case report

An 82 year old women with known portal hypertension secondary to cirrhosis of the liver was referred to the otolaryngology outpatient department with a two month history of daily haemoptysis and bloodstained pharyngeal secretions; this occurred mostly on early morning coughing. There was no history of weight loss, dysphagia, dysphonia, or throat pain. She had already been investigated for a pulmonary cause of the haemoptysis and none was found. In keeping with her history, liver function tests and the prothrombin time were abnormal. She also had a history of well controlled essential hypertension. Chest radiography showed cardiomegaly, but the jugular venous pressure was not raised and there was no other clinical manifestation of heart failure.

Indirect laryngoscopy revealed varicose vessels in the tongue base, mainly on the left side (see fig 1). These appeared friable and one area revealed a propensity to bleed on examination. The rest of the ear, nose, and throat examination was normal.

After perioperative cover with fresh frozen plasma, vitamin K, and tranexamic acid she underwent ablation of the varicosities using a 15 watt continuous carbon dioxide laser under general anaesthesia (see fig 2). She had an uneventful recovery and has remained symptom free.

Anatomy

The dorsal lingual veins drain the tongue base through two or more tributaries. These course inferiorly, join together and form the lingual vein, which accompanies the lingual artery. These vessels pass between genioglossus and hyoglossus and the vein empties into the internal jugular vein just above the level of the greater cornu of the hyoid bone.[1]

PORTOSYSTEMIC ANASTOMOSIS

Because the portal system has no valves, portal hypertension results in shunting of blood through anastomotic communications with the systemic venous system. Recognised connections are[2]:

(1) Lower oesophageal veins—the branches of the left gastric vein anastomose profusely with the azygous and hemiazygous veins. Increased shunting results in oesophageal varices within the mucosa of the lower third of the oesophagus.

(2) Periumbilical veins—veins contained in the falciform ligament anastomose with superior and inferior epigastric veins of the anterior abdominal wall. Excessive dilatation of these veins are evident as caput medusa.

(3) Haemorrhoids—the superior rectal vein anastomoses with the middle rectal vein which drains into the internal iliac vein. In addition the middle rectal veins anastomose with the inferior rectal veins which drain into the internal pudendal vein. Increased shunting results in internal and external haemorrhoids.

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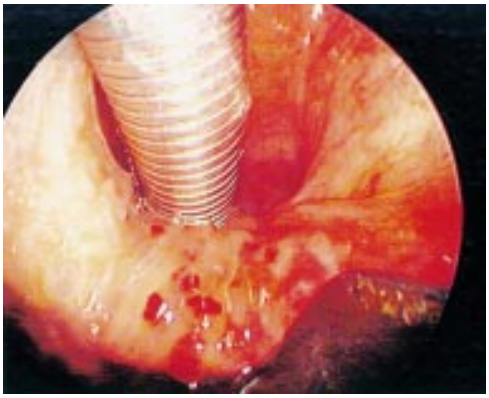

Figure 1 Tongue base varices.

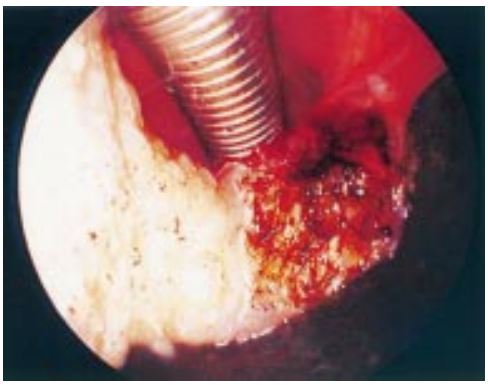

Figure 2 Tongue base varices after laser ablation.

(4) Veins of Retzius—the veins of a secondary retroperitoneal structure may anastomose with the veins of the dorsal body wall, forming veins of Retzius.

Thus, there is no recognised anastomosis between lingual venous drainage and the portal circulation.

Discussion

A varicosity is a condition indicating an enlarged and tortuous vein. Previously described lingual varices referred to sublingual varices on the ventral surface of the tongue or floor of mouth. These were ascribed to the use of dentures, and vitamin C deficiency in an elderly population.[3]

Burket associated the occurrence of lingual varices with cardiorespiratory disease,[4] however this assumption was shown to be unsubstantiated in a double blind study by Kleinman.[5]

This represents the first reported case of dorsal tongue base varices in a patient with portal hypertension. We propose three explanations for this association. Firstly that the varices represent a spontaneous entity unrelated to systemic disease. Secondly, they are a manifestation of cardiopulmonary disease, an association endorsed by the known anatomic connection, but improbable given that there was no other clinical evidence of heart failure such as a raised jugular venous pressure. Lastly, that there is an, as yet, unrecognised anastomotic connection between lingual venous drainage and the portal circulation.

Tongue base varices should be considered in cases of blood expectoration and portal hypertension.

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Cefuroxime induced lymphomatoid hypersensitivity reaction

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Abstract

An 84 year old women developed erythematous blotchy erythema and purpuric rashes over the lower limbs three days after being started on intravenous cefuroxime for acute diverticulitis. A skin biopsy specimen showed a mixed infiltrate of lymphoid cells and eosinophils; many of the lymphocytes were large, pleomorphic, and showed a raised mitotic rate. Immunohistochemistry showed the infiltrate to be T cell rich, with all the large cells being CD30 positive. Typical mycosis fungoides cells, marked epidermotropism, and Pautrier's abscesses were not seen. The rash disappeared 10 days after cessation of cefuroxime and the patient remained asymptomatic 15 months later. This apparent cutaneous T cell lymphoma-like reaction is best described as lymphomatoid vascular reaction. The drug induced immune response with an atypical cutaneous lymphoid infiltrate mimics a cutaneous pseudolymphoma.

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Keywords: cefuroxime; atypical cutaneous lymphomatoid infiltrate; cutaneous T cell lymphoma

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Case report

An 84 year old women, with established diverticula disease, presented as an emergency with a short history of fever, acute abdominal pain, and diarrhoea. On examination she was found to be toxic, febrile, dehydrated, with a tachycardia of 110/min and blood pressure 80/40 mm Hg. The left iliac fossa was tender. Haemoglobin, full biochemistry profile, including serum amylase, chest radiography, plain abdominal radiography, and an abdominal ultrasound scan were normal. Her white cell count was raised at 20.4×10^9/l with marked neutrophilia at 14.2×10^9/l.

A provisional clinical diagnosis of acute diverticulitis with septic shock was made and she was started on intravenous (IV) fluids, IV metronidazole 500 mg eight hourly, and IV cefuroxime 750 mg three times a day in a sequential manner. Three days later and after five doses of IV cefuroxime, purpuric and erythematous macular rashes developed over the lower limbs. Cefuroxime was considered to be responsible and was discontinued; metronidazole was continued for another week. The rashes were treated with flucinolone acetonide 0.00625% cream. Further investigations at this stage showed normal immunoglobulins, antinuclear factor, antineutrophilic cytoplasmic antibodies, and autoantibody profile. A haematoxylin and eosin stained section of representative skin biopsy showed a mixed dermal infiltrate of lymphoid cells and eosinophils, marked red blood cell extravasation indicative of ongoing small vessel damage, minimal focal vacuolar interface