Acute respiratory insufficiency after endoscopy for bleeding oesophageal varices

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Abstract

Two patients with alcoholic liver disease and gross ascites underwent endoscopic injection and compression by Sengstaken tube of oesophageal varices under general anaesthesia. Postoperatively both patients developed acute respiratory failure, which resolved after air had been aspirated from the stomach via the Sengstaken tube.

All air should be aspirated at the end of the procedure in patients with ascites who undergo endoscopy, and respiration should be carefully supervised postoperatively.

Introduction

Diaphragmatic excursion is the principal muscular movement in achieving inspiration. In the presence of mechanical obstruction vital capacity and functional residual capacity fall appreciably. We report on two patients with alcoholic liver disease and gross ascites who developed acute respiratory failure after endoscopic injection of oesophageal varices under general anaesthesia.

Case reports

CASE 1

A 51 year old woman was admitted to this unit with bleeding oesophageal varices, gross ascites, and hepatic encephalopathy. Anaesthesia was induced with etomidate 12 mg and suxamethonium 50 mg and maintained with intermittent positive pressure ventilation together with 0-6% enflurane, oxygen, and 60% nitrous oxide. Her varices were injected with 5% ethanolamine oleate endoscopically and then compressed by a Sengstaken tube.
Postoperatively, she rapidly recovered spontaneous respiration. Within minutes after extubation she developed acute respiratory failure followed by cardiovascular collapse. There was no evidence of further bleeding. She was rapidly reintubated and resuscitated, and on her return to the unit her girth had increased to 1-4 m and her abdomen was tympanic on percussion. Air was aspirated by suction on the Sengstaken tube and her cardiovascular system restabilised. She was extubated and returned to the ward.

CASE 2

A 40 year old man with alcoholic liver disease was admitted after a massive haematemesis. Anaesthesia was induced with etomidate 10 mg and suxamethonium 100 mg and maintained with alcuronium 15 mg, 0-6%, enfurane, oxygen, and 60%, nitrous oxide and intermittent positive pressure ventilation. At emergency endoscopy his varices were injected and a Sengstaken tube left in place. Postoperatively, residual neuromuscular blockade was antagonised by atropine 1-2 mg and neostigmine 2-5 mg and spontaneous respiration returned. He was extubated in the left lateral position but, when turned on to his back, became apnoeic with circulatory collapse. He was again turned to the left lateral position, reintubated, and ventilated. Air was aspirated from the stomach via the Sengstaken tube and intermittent positive pressure ventilation was continued for two days before he could maintain adequate spontaneous respiration.

Discussion

Many abdominal conditions restrict diaphragmatic movement. These include simple obesity, pregnancy, ovarian cysts, and peritoneal dialysis, as well as ascites and gastric distension. Our two patients were already severely compromised by their ascites and the reduction in functional residual capacity that occurred in the supine position and after anaesthesia. Air insufflated by the endoscopist during variceal injection was retained in the stomach, which was already distended with the gastric balloon of the Sengstaken tube containing 400 ml air. This may have tipped these patients into acute respiratory failure. The clinical improvement after gastric decompression would tend to support this hypothesis. A pilot study investigating respiratory function in the presence of gross ascites has shown a reduction in vital capacity by up to 50% of predicted values, and we propose to study this further.

High intra-abdominal pressures are known to reduce venous return and impair myocardial performance.1 The cardiovascular collapse seen in these two patients may have been due partly to compression of the inferior vena cava, the so-called “supine hypotension syndrome” of pregnancy. Certainly the second patient (case 2) showed some clinical improvement when he was turned to the left lateral position before resuscitation.

Patients with ascites who undergo endoscopy should have all air aspirated at the end of the procedure, and careful respiratory supervision should be continued in an intensive care unit during the early postoperative phase.

References


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Pseudoporphyria associated with consumption of brewers' yeast

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Abstract

A case of pseudoporphyria associated with excessive consumption of brewers' yeast was studied. Detailed analysis of the yeast tablets by high performance liquid chromatography showed the presence of dicarboxylic deuteroporphyrin, mesoporphyrin, and protoporphyrin; coproporphyrin I and III isomers; and uroporphyrin I and III isomers. The faecal porphyrin concentration of the patient taking yeast tablets was significantly increased, resembling the excretion pattern in variegate porphyria.

Any patient showing an unusual porphyrin excretion pattern on high performance liquid chromatography should be investigated for a possible dietary cause.

Introduction

Brewers' yeast is sold as vitamin enriched tablets with added thiamine, riboflavin, and nicotinic acid. Although certain strains of yeast are known to accumulate porphyrins,2 there have been no reports of porphyrins in commonly taken vitamin enriched yeast tablets. Diagnosis of the porphyrrias requires the routine identification and determination of porphyrins in blood, urine, and faeces. In recent years this has been carried out increasingly by high performance liquid chromatography.2,4

We report a case of pseudoporphyria associated with consumption of brewers' yeast and describe the detailed analysis of porphyrins in these tablets and their relation to the biochemical identification of the porphyrrias.

Present study

CASE REPORT

A 24 year old man suffered persistently from episodic abdominal pain. Since no other abnormalities were found, screening tests for porphyrias were performed. He had normal urinary concentrations of 5-aminolaevulinate (15 μmol/l [201 μg/100 ml]; normal range 0-40 μmol/l [0-536 μg/100 ml]), porphobilinogen (1-6 μmol/l [36 μg/100 ml]; normal range 0-8 μmol/l [0-181 μg/100 ml]), and total

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