Upper gastrointestinal bleeding secondary to vigorous physical exercise☆

Hemorragia digestiva alta secundaria a ejercicio físico intenso

The appearance of digestive symptoms in athletes is relatively frequent, and according to published case series, varies from 30-81%. Gastroesophageal reflux, nausea, vomiting, diarrhea, or transitory abdominal pain linked to exercise are the most frequent manifestations. Since the 1980s, numerous observational studies have shown that intense exercise can cause iron-deficiency anemia due to the appearance of gastritis, gastroduodenal ulcers, or erosions in the small bowel and colon. Depending on the degree of exercise, 7 to 85% of athletes can present with a positive fecal occult blood test upon finishing their training. Marked gastrointestinal bleeding is much less frequent and only anecdotal cases are found in the literature.1 2 The first and only death due to gastrointestinal bleeding in a jogger was reported in 1982.1

A 30-year-old man with an unremarkable personal and family history came to the emergency department complaining of 3 bowel movements consistent with melena within the past 24 h. He stated that he had not taken any drug or gastrototoxic agent, no nutritional supplement, and no tobacco, alcohol, or other toxic substance. When asked about his usual physical activity, the patient said he rode a bicycle 4-5 times a week for approximately 45 min. When questioned about his activity in the morning before coming to the emergency department, he stated that 6 h before presenting with melena he had finished a 5-h bicycle ride at a much greater speed than usual. Physical examination revealed a heart rate of 105 bpm and the rest of his vital signs were normal. The laboratory work-up showed urea of 56 mg/dl (normal: 15-45 mg/dl) and hemoglobin of 12.7 g/dl (normal: 13-17.5 g/dl). The remaining results were normal. An emergency upper gastrointestinal endoscopy was performed that identified 10 linear ulcers in the gastric body of approximately 10-15 mm in length and 2 mm in width, following the direction of the gastric folds. The ulcers had irregular edges and were covered with hemat (Forrest IIc) and fibrin (Forrest III) (fig. 1). Two biopsies were taken from the antrum, two from the gastric body, and one from each of the two largest ulcers. The patient remained under surveillance for 24 h, presented with no other episodes of melena, and was released under treatment with omeprazole 20 mg/12 h for 8 weeks. The results of the gastric biopsies were negative for H. pylori and there were no foci of dysplasia or metaplasia. Only some alterations consistent with the base of an ulcer and a mild, nonspecific, acute inflammatory infiltrate were documented. Upper gastrointestinal endoscopy was repeated at 12 weeks, confirming the endoscopic and histologic cure of the lesions. During follow-up, two 13C-labelled urea tests were carried out (with no previous PPI or antibiotic ingestion) to detect H. pylori, and were negative. The laboratory work-up was extended to include PTH and gastrin determination, and both were normal. Abdominal ultrasound had no significant findings. One year after the initial episode the patient is asymptomatic.

In 2001, Choi et al. conducted one of the most demonstrative studies, despite its small sample size,
on upper gastrointestinal lesions secondary to vigorous physical activity. Theirs was a prospective study that included 16 long-distance runners (20 km) that underwent endoscopy, laboratory tests, and fecal occult blood testing. At endoscopy after the race, all the participants had developed gastritis, six presented with esophagitis, and one patient had a new gastric ulcer. It is postulated that the reduction of splanchnic flow during exercise, the activation of the sympathetic nervous system, and the inhibition of the parasympathetic nervous system are the main pathophysiologic mechanisms implicated in the damage of the gastrointestinal mucosa.  

Regarding the two studies and the existence of confusion factors not included in the analysis, for example, economic status and its association with given patterns of consumerism or the heterogeneity in measuring the physical activity, limited the conclusions that the authors could make.

The use of anti-H2 drugs has not shown efficacy in the prevention of gastrointestinal blood loss in these patients. Regarding PPIs, there is only one blinded and randomized clinical trial with pantoprazole 20 mg/24 h/3 days on 37 persons that competed in an ultra-marathon (246 km). Pantoprazole significantly reduced the rate of positive fecal occult blood tests (difference of risk: 0.86; 95% CI: 0.45-0.96), but no clinically significant events were detected in any of the groups. Due to the sparsity of data on the pharmacologic prevention of these types of lesions, recommendations cannot be established, beyond moderation in physical activity and avoiding the use of NSAIDs.

In our patient, we can assume that there was a causal relation between intense physical exercise and the gastrointestinal bleeding, because he did not ingest gastrotoxic drugs, the temporal relation between the physical activity and the presentation of symptoms was 6 h, three tests for H. pylori were negative, there were no clinical and laboratory data suggesting an alternative etiology, and he has had no new episodes during the one-year follow-up, since moderating his physical activity.

This case illustrates an infrequent cause of gastrointestinal bleeding and underlines the importance of a thorough and directed anamnesis in patients with gastrointestinal bleeding of unknown origin.

Conflict of interest

The authors declare that there is no conflict of interest.

References


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