Case Study

Evolutionary and pronostics aspects of Cirrhosis at the University Hospital of Brazzaville (Congo)

Summary

Cirrhosis is a serious pathology that leads to complications whose management remains difficult in our country.

Objective: to evaluate the evolutionary aspects of cirrhosis at the Brazzaville University Hospital.

Patients and methods: this was a retrospective study over a period of 18 months (from January 2015 to July 2016), performed in the gastroenterology department of the Brazzaville University Hospital. All patients regularly followed for decompensated cirrhosis were included. The studied variables were the epidemiological, clinical and paraclinical characteristics, the occurrence of complications, the survival, the causes of death. Univariate analysis was used to determine prognostic factors based on the Child Pugh score.

Results: There were 43 patients, including 32 men and 11 women, with a mean age of 52 ± 9.5 years. Viral hepatitis B was the most common etiology (39.5%). Child Pugh stage C was found in 60.5%. Oesophageal varices were present in 93% of cases. Survival at 18 months was 72%. In univariate analysis, two variables were significantly associated with decreased survival, including Child Pugh stage C and esophageal varices (grade II and III).

Conclusion: cirrhosis remains a worrying pathology because the diagnosis is often made at the stage of often serious complications putting at risk of vital prognosis.

Introduction

The association between chronic fundic atrophic gastritis and iron deficiency anemia has been established for many years, particularly during Biermer’s disease. The association pangastritis with Helicobacter pylori and unexplained iron deficiency anemia has recently been proposed although still discussed [1].

Among the possible causes, the role of Helicobacter pylori infection remains controversial [2,3]. In Congo, the prevalence of H pylori infection is estimated at 47.8% in children [4] and 89% in 2014, in the general population [5]. The main causes of iron deficiency anemia are dominated by parasitosis and malnutrition [4]. We report a clinical case of recurrent anemia secondary to chronic H pylori gastritis.

Observation

This patient was 21 years old, hospitalized in June 2015 for epigastric and syndromeanemic pain. The interrogation did not find any notion of externalized digestive hemorrhage or gastrointestinal transit disorder. She had no pathological digestive history, neither hematological nor gynecological.

The clinical examination revealed signs of intolerance of anemia and sensitivity to palpation of the epigastric region, with no noticeable mass. The pelvic touches were normal.

The hemogram showed normochromic microcytic anemia with hemoglobin at 4 g/dl. Ferritinemia was decreased to 19.5 mg/L (N = 22–321 mg/L) and serum iron to 0.27 mg/L (N = 0.6–1.6 mg/L).

In the emergency the patient was transfused with 4 units of erythrocyte concentrate. Upper gastrointestinal endoscopy showed squamous corporo-fundic mucosa and nodular antropathy without recent bleeding stigma. Multiple antrofundic biopsies were performed for anatomo-pathological examination.

While awaiting the histological results of the biopsies, the
patient was treated with iron. One month after treatment, the hemoglobin count had normalized. Four months after stopping the martial treatment, there was a recurrence of anemia. Colonoscopy was macroscopically normal. Biological and morphological investigations for portal hypertension and hepatocellular insufficiency were normal.

Assay for immunoglobulin A (IgA) anti-transglutaminase antibodies was negative. The parietal cell antibodies as well as the intrinsic anti-factor antibodies were negative. H. pylori serology was positive.

Biopsies from the antrum and fundus showed a large inflammatory infiltrate with many H. pylori.

The patient was undergoing sequential Hpylori eradication therapy with esomeprazole 40 mg twice daily and amoxicillin 1g twice daily for 5 days, followed by esomeprazole 40 mg twice daily, combined with metronidazole 500 mg twice daily and clarithromycin 500 mg twice daily for 5 days. A new iron supplementation for 2 months

Discussion

Iron depletion is one of the major causes of anemia worldwide. It is estimated that iron depletion affects 15% of the world’s population [6]. Endoscopy of the gastrointestinal tract remains essential in the investigation of iron deficiency anemia. However, after a well-conducted exploration, about 35% of anemias remain unexplained [7,8].

This observation reminds us that chronic gastritis with H pylori is a classic and sometimes unknown cause of iron deficiency anemia. Several sero-epidemiological studies carried out, particularly in Africa and South Korea, have also shown that ferritinemia was reduced in patients (children and adults) with positive H pylori serology [8–10].

Several mechanisms have been proposed to explain the relationship between iron deficiency anemia and H pylori; this is occult digestive haemorrhage, reduction of iron absorption and iron sequestration by a receptor located at the level of the H. pylori membrane [11].

Occult bleeding secondary to erosive gastritis has been suggested as one of the mechanisms that may explain iron deficiency in patients with H. pylori infection. However, most published series did not mention endoscopic lesions that may explain occult bleeding [10]. Decreased absorption of secondary iron in the chronic gastric and hypo or achlorhydria is the most likely mechanism [12,13]. Although discussed, iron sequestration and H pylori use have also been suggested in patients with H pylori-associated iron deficiency anemia. Indeed, H pylori needs iron for its growth and has a ferritin-like iron transporter protein [12].

Chronic H pylori infection has no clinical expression in 90% of cases [14]. In our observation, epigastric pain and anemic syndrome were the reason for consultation.

Indeed, the eradication of H pylori was associated with a considerable improvement in symptoms. This suggests that there is a pathophysiological link between H pylori and abdominal pain. But it should be noted that the presence of isolated abdominal pain does not justify the systematic search for H pylori infection. It has been reported that the majority of people, particularly children without H pylori infection who presented with abdominal pain had gastritis [14,15]. It could therefore be hypothesised that the pain would be related to gastritis and that H. pylori would be only one of the etiologies, which would explain why knowledge of the status of H pylori is not sufficient to justify a symptomatology.

The recurrence of anemia in our patient forced us to perform a high digestive endoscopy.

The diagnosis of iron deficiency anemia related to H pylori gastritis was evoked in light of the recurrent nature of anemia and confirmed by the positivity of H pylori serology and especially in the case of antro-fundic biopsies (H pangastritis) pylori).

No recurrence of anemia was observed after treatment combining the eradication of H pylori and iron supplementation in our patient. In general, three months after martial treatment and H pylori eradication, there is a statistically significant increase in hemoglobin and iron [16,17].

Conclusion

This observation shows that it is important to look for H pylori gastritis in patients with recurrent iron deficiency anemia. Digestive endoscopy with biopsies is the first-line examination for the search for etiology.

References