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Peptic Ulcer Associated with Polycythemia Rubra Vera: A Case Report

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1. Abstract

Polycythemia Vera (PV) is a blood disorders known as myeloproliferative disease. Gastric and duodenal ulcer is a frequent disease among population but rarely is a complication of myeloproliferative disorders. Its main etiologies are inbalance of gastric acid secretion and mucosa protection. The complication of PV by an ulcer is a possible but not scientifically well established. The microvascular complications by thrombosis which lead to mucosa alteration are highly suspected. The treatment of gastric ulcer is medical using proton pomp inhibitors but also surgery for perforated ulcer while the phlebotomy is for the PV in emergency to reduce the symptoms and prevent vascular complications.

2. Introduction

Gastric and duodenal ulcer is a frequent disease among population but rarely is a complication of myeloproliferative disorders (MPD) [1]. Polycythemia vera (PV) complicated by an ulcer perforation is unusual and few cases are repported [2]. We report a case of a patient admitted in our service for peptic ulcer perforation confirmed by a chest X ray with pneumoperitoneum and the biology assessment revealed an uknown PV. The aim of this case is to establish the link between the PV and ulcer. This case is presented is presented in line with care checklist 2020 [3].

3. Case Presentation

It is a 37 year old man with tobacco and alcohol intoxication who presented with epigastric pain over 10 hours with vomiting. He has any history of erythromalalgia, pruritis or headache. The physical examine revealed abdominal tenderness with tachypnea at 24 of respiratory rate, pulse of 88 beats/ min, pressure of 12/7 mmHg and temperature of 37.2°C. No splenomegaly was found. Abdominal plain radiograph showed the presence of pneumoperitoneum (Figure 1).

The laboratory blood tests revealed erythrocytosis of 20.4g of Hg/dl, a low thrombocytosis of 424000/ mm3 of platlet and the white blue cells of 4500/mm3.Prothrombine : 95% ; Natremia : 129 mg/l ; kaliemia : 8,6mmol/l ; Urea: 0.23 g/l ; creatininemia : 19.1 mg/l ; aspartate aminotranspeptidase enzyme : 86UI/l ; alanine aminotransferase : 18UI/l ; CRP= 38.5mg/l ; albuminea :45 g/l. he has any history of hematologic disorders such us anemia. The patient was admitted in the service of emergency of visceral surgery of Ibn Rochd UHC for surgery of ucer perforation but the symptoms worsened during his first four hours following his admission and died before the operation.

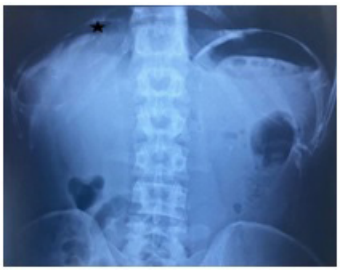

Figure 1: Abdominal plain radiograph of the patient: the asterisk show right pneumoperitoneum

4. Discussion

Polycythaemia Vera (PV) belongs to a group of blood disorders known as myeloproliferative disease (Gilbert, 1973), characterized by haematopoietic proliferation, that is mainly expressed as an inappropriate increase in the number of circulating red blood cells.

The middle-aged and elderly population is frequently concerned of PV, with a slight male preponderance. PV is defined by an acquired increase in hemoglobin andhematocrit level above 16.5 gm/dL/49% in men and 16 g/dL/48% in women, associated with JAK2 mutation and bone marrow morphology characteristics. The 2016 WHO classification system for hematopoietic tumors recognizes the almost constant association between PV and a JAK2 mutation, for JAK2V617F is detected in 50–70% of patients with either ET and PMF. The formal diagnostic requires three major signs (Hb/Hct or level above 16.5 g/dL/49% in men and 16 g/dL/48% in women or red cell mass >25% above mean normal predicted value; consistent bone marrow morphology; and presence of a JAK2V617F or exon 12 mutation) and one minor (subnormal serum erythropoietin (Epo) level) criteria; WHO-qualified diagnosis requires the presence of either all three major criteria or the first two major criteria plus the minor criterion [4]. The differential diagnosis are secondary PV due to hypoxemia in patients with chronic obstructive bronchopneumopathy, renal tumor with erythropoitin secretion idiopathic PV [5]. Clinical manifestations of PV include gastrointestinal disorders well known, represented by peptic ulcers found with a notably higher frequency than in the normal population ranging from 7% to 23% [6]. Gastroduodenal ulcer is the ultimate loss of mucosal integrity as the result of an inbalance between aggression and defense. No steroid anti-inflammatory drugs, epithrial and micrivascular injuries are the maim etiologies of ulcer. Obliteration of the microvascular blood supply may well contribute a component of focal ischemia responsible for the production of a crater. Ulcers, no matter what the cause, are often called peptic because they almost always develop in an acid-peptic microenvironment, specific for the gastroduodenum [7]. It is now well known that H. pylori infection is the main etiology of chronic gastritis and ulcer disease [8]. The most extensively studied of such strains are those expressing the cagA gene [9]. And several studies have shown that CagA was present in 80–100% of H. pylori isolates from patients with duodenal or gastric ulceration compared with 50–60% in patients from superficial gastritis alone [6] (Crabtree et al, 1991). Other vascular complications in patients with polycythemia vera are microvascular circulatory disturbances typical of thrombocythemia including erythromelalgia, peripheral ischemia, atypical cerebral ischemic attacks, and major arterial and venous thrombotic events [10]. Nomura et al. in their study evaluated the prevalence and severity of gastroduodenal lesions in a group of patients with PV compared with a group of age and sex-matched controls undergoing endoscopic examination for dyspeptic symptoms and found the interaction between H. Pylori infectious and PV [2]. The pathophysiology and mechanisms of peptic ulcers in PV are altered mucosal blood flow due to increased plasma viscosity, and/ or increased histamine release, which was found to be related to high blood basophil counts which lead to mucosa ischemia and ulceration [11]. In their experimental kowaleski et al. study found an increased gastric secretion associated with gastric ulcer in rats induced polycythemia vera. There is a great relation and interaction between erythrocytosis and ulcer independing of its etiology [12]. Whether primary or secondary, the increase of blood Packed Cell Volume (PCV) is always associated with reduced splanchnic circulation and high risks of thrombosis which leads to mucosa ischemia and ulcer. The complication of perforation then warsens the state and the bresults is death for patient.

Torgano et al. in their study found a higher prevalence of gastroduodenal lesions in PV patients, with respect to age- and sexmatched controls. In fact, such prevalence was 74% in PV versus 19% in controls, with gastric or duodenal ulcers being observed in 29% and 7% respectively. The prevalence of H. pylori was also significantly higher in PV patients than in controls (83 versus 57%), suggesting that PV may predispose to H. pylori infection. Whether blood hyperviscosity or other factors play a role in facilitating H. pylori infection in PV patients remains to be determined. The underlying pathogenetic mechanism has not been thouroughly studied, and could possibly involve an impairment of blood mucosal flow, due to blood hyperviscosity. For both the two diseases, the diagnosis and treatment are known but their association still not well established till now. The medical treatment of gastric ulcer is proton pomp inhibitors and surgery for perforated ulcer. The treatment of PV is essentially phlebotomy to reduce symptoms. For our case, the patient presented with symptoms of ulcer perforation evolving 5 hours and the clinical worsened quickly after hospitalisation. The patient died despite of rescussitation methods after respiratory distress and hemodynamic instability. The blood count analysis found 60% of hematocrit and 20g/ml of Hg. Then, we concluded for a possible uknown and complicated PV with perforated gastroduodenal ulcer. The laboratory tests shaw a high suspicon of PV with the 60% of hematocrit and 20 mg/ml of Hg. Although However, we do not perform autopsie or genetic test for JAK2 mutation to confirm a myeloproliferative syndrome, we choose to share this case for learning purpose and to allert health practitionners for an actually fatal ulcer complication in patients with PV which need a high surveillance.

5. Conclusion

Although gastric ulcer is a benign pathology, it can be a life threatening when it is associated with other pathologies. The gastroduodenal disorders complications of PV especially gastric ulcer are severe, can appear after diagnosis or reveal the disease.

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