Multifocal Colon Cancer in Giant Cell Arteritis

Salem Bouomrani1,2* Marwa Ghribi1,2
1Department of Internal medicine, Military Hospital of Gabes, Gabes 6000, Tunisia
2Sfax Faculty of Medicine, University of Sfax, Sfax 3029, Tunisia

*Corresponding Author: Salem Bouomrani, Department of Internal medicine, Military Hospital of Gabes, Gabes. Sfax Faculty of Medicine, University of Sfax, Sfax, Tunisia. Email: salembouomrani@yahoo.fr

Abstract: Giant cell arthritis (GCA) is a systemic vasculitis predominant in large arteries and in subjects over 50. Several sporadic cases of solid cancers or malignant hemopathies have been reported in association with GCA, and an over-risk of malignancy of this vacuity was demonstrated by several authors but remains very controversial.

We report an original observation of multifocal colic cancer occurring in a 75-year-old Tunisian woman followed for GCA for 5 years.

Synchronous occurrence of the two diseases, paraneoplastic forms of GCA, as well as concomitant relapse of both conditions in certain patients are arguments in favor of the direct causal link between GCA and malignant degeneration. In our observation, the multifocal character of colonic degeneration would be one more argument in favor of the predisposing role of GCA.

Keywords: Colonic cancer, Giant Cell Arteritis, Horton’s disease, Cancer, Vasculitis.

1. INTRODUCTION

Giant cell arthritis (GCA) or Horton's disease is a systemic granulomatous vacuity with segmental and multifocal involvement predominant in large arteries; particularly the divisional branches of the aorta and especially temporal and other cephalic arteries [1, 2]. This angiitis predilectionally affects subjects over the age of 70, and is far more frequent in the Nordic countries with an annual incidence of 1-27/100,000 [1, 2].

The main manifestations of this disease are ophthalmological, neurological, vascular, and rheumatic, including polymyalgia rheumatica (rhizomelic pseudopolyarthritis) [1,2]. More rarely, vascular inflammation can spread throughout the blood vessels and cause serious, and sometimes even fatal, visceral manifestations: gastrointestinal, cardiac, pulmonary, renal and cutaneous [3,4]. These so-called “extra cranial” manifestations remain exceptional: 1.4 to 1.7% of cases in large autopsy series [3]. There was an over-risk of malignancy associated with this vasculitis [5], sometimes even with real paraneoplastic forms of GCA [6,7]. Colorectal cancers remain exceptionally reported during this vasculitis [8].

We report an original observation of multifocal colic cancer occurring in a patient followed for GCA.

2. CASE REPORT

75-year-old Tunisian woman, with a medical history of well treated essential hypertension and mixed dyslipidemia, was hospitalized for left temporal headache resistant to symptomatic analgesic treatment.

The somatic examination noted an apyretic patient, hyperesthesia of the scalp, and an absent left temporal pulse. The ophthalmological, neurological, joint, and skin examination was without abnormalities.

Biologic showed a marked biological inflammatory syndrome with an erythrocyte sedimentation rate at 98mm/H1, a C-reactive protein at 32mg/l, and a polyclonal hypergamma pathy at 24g/l. The other basic laboratory tests were within normal limits (hemoglobin, platelets, leukocytes, creatinine, calcemia, blood sugar, ionogram, transaminases, muscle enzymes, and urinalysis).

The infectious investigation was negative: chest X-ray, sinus X-ray, ENT examination, stomatological examination, blood cultures, urine direct examinations and cultures, and transthoracic cardiac ultrasound. As well as immunological tests (anti-nuclear antibodies, anti-native DNA antibodies, ANCA antibodies, and anti-phospholipid antibodies).
The left temporal artery biopsy confirmed the diagnosis of GCA by showing focal and segmental temporal arteritis with giant cell and fragmentation of the internal elastic lamina.

The patient was treated with systemic glucocorticoids at a dose of 0.5mg/kg/day and salicylated acid at a dose of 100mg/day with good outcome: disappearance of cephalic functional complaints from the second day, and normalization of the biological parameters of the inflammation from the first week of treatment.

Five years after the diagnosis of GCA, the patient had two episodes of low abundance rectorrhagia with abdominal pain. Her disease had been in complete clinical and biological remission for three years and she had not been taking specific treatment for GCA for a year.

The somatic examination was without significant anomalies. The examination of the anal margin showed no tumors or hemorrhoids. Biology objectified a microcytic anemia at 9g/dl and an erythrocyte sedimentation rate at 68mm/H1 without other anomalies. The abdominal ultrasound was normal. Colonoscopy showed multiple colonic polyps, some of which were ulcerated and hemorrhagic. Histological examination revealed several foci of intraepithelial carcinomatous degeneration in several polyps. As well as some foci of in-situ cancer in the non-polypomatous colonic mucosa. The patient was referred to the surgical department for adequate management.

3. DISCUSSION

Several sporadic cases of solid cancers or malignant hemopathies have been reported in association with GCA [8-13]. Likewise, cancers represent one of the main causes of death during this vasculitis [14]. Although this association seems to be, for several authors, far from a mere coincidence [15, 16], it remains very controversial by others [17, 18].

The overall prevalence of cancers (solid cancers and malignant hemopathies) during GCA was estimated at 17% in Liozon E et al series collecting 250 cases of patients followed for this vasculitis (41 cases of malignancies including 25 solid cancers and 16 malignant hemopathies) [16]. Ungprasert P et al, systematic review and meta-analysis of cohort studies reporting malignancy risk in patients with GCA and/or polymyalgia rheumatic, demonstrated a low but statistically significant increased malignancy risk among these patients (pooled risk ratio of malignancy at 1.14) [5]. The risk was particularly higher in the first year after the diagnosis of the GCA (pooled risk ratio of 2.16) [5]. Similarly, in Liozon E et al series, 79% of cancer cases were diagnosed in the year preceding or following the diagnosis of GCA [16].

The pathogenesis of malignancy during GCA is not well understood. It appears to be multifactorial involving, like other chronic inflammatory diseases, persistent chronic inflammation, underlying immune dysfunction, oxidative stress, and vasculitis itself [19].

The synchronous occurrence of the two diseases [12, 17], the paraneoplastic forms [6, 7], as well as the concomitant relapse of both conditions in certain patients [17] are arguments in favor of the direct causal link between GCA and malignant degeneration.

4. CONCLUSION

In patients followed for GCA, a particularly increased risk of malignancy should be kept in mind of the treating physicians. Regular clinical and preclinical monitoring is necessary for the early diagnosis and adequate management of cancers.

In our observation, the multifocal character of colonic degeneration would be one more argument in favor of the predisposing role of GCA.

REFERENCES


