Mesenteric Ischemia in Giant Cell Arteritis: 6 Cases and a Systematic Review

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ABSTRACT. ***Objective.*** To report the main features of mesenteric ischemia related to giant cell arteritis (GCA).

Methods. We screened 13 French internal medicine tertiary care centers for their cases of patients exhibiting GCA-associated mesenteric ischemia during a 16-year period (1990–2006). Patients were included if they reported newly developed abdominal symptoms associated with histological proof of GCA-associated mesenteric vasculitis and/or radiological abnormalities consistent with GCA-associated mesenteric vasculitis. We performed a Medline search to identify previously reported cases of GCA-associated mesenteric ischemia.

Results. We included 6 original cases and 22 cases identified in the literature (mean age of the 28 patients: 72.4 ± 7.1 yrs; women: 79%). GCA was histologically proven for all patients. In 12 patients GCA diagnosis preceded mesenteric inflammatory arteritis. Mesenteric ischemia occurred either soon after initiation of steroid therapy (n = 6, mean time to onset after starting steroid 12 ± 11 days) or with a low-dose steroid regimen (n = 6, dosage 0–10 mg/day). In 16 other patients, the mesenteric involvement was the first manifestation of GCA. Only 6 patients (21%) reported cardiovascular risk factors. Clinical manifestations of GCA-associated mesenteric ischemia, as well as biological markers (mean C-reactive protein level 91 ± 50 mg/l), were very nonspecific. Imaging explorations were performed for 14 patients and showed specific signs of vasculitis on the mesenteric artery in 10 (71%). Nineteen patients (68%) required laparotomy and 9 patients (33%) died.

Conclusion. Early diagnosis and medical management of mesenteric GCA may ameliorate the severe prognosis of this possibly underdiagnosed complication. (First Release July 1 2007; J Rheumatol 2007;34:1727–32)

Key Indexing Terms:
MESENTERIC ISCHEMIA GIANT CELL ARTERITIS

Giant cell arteritis (GCA) is characterized by inflammatory involvement of large- and medium-size arteries, affecting mainly the aortic arch and its branches. GCA is the most common vasculitis and occurs in elderly patients with an incidence of 17.8/100,000 patients over 50 years old and 46/100,000 patients over 70 years old[1]. GCA clinical symptoms are more often related to ischemic cranial involvement that may be associated with constitutional symptoms such as fever, weight loss, anorexia, or fatigue.

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Extracranial manifestations of GCA are not commonly reported. Only 9% of patients with GCA exhibit involvement of various extracranial sites such as pelvis, thorax, and abdomen[2]. Mesenteric ischemia related to GCA was first described in 1965, and only 22 cases have been reported since then[3-24]. However, the prevalence of mesenteric involvement is probably underestimated during the course of GCA, as suggested by systematic postmortem studies. In studies by Stenwig[21] and Ostberg[25], 20% (13 out of 64) and 33% (4 out of 12) of patients, respectively, had histological mesenteric vasculitis despite the absence of abdominal symptoms. Moreover, it has been reported that large artery involvement is frequent and underrecognized during the course of GCA[26]. Information from a retrospective population-based cohort revealed that any type of large-artery complication occurred in up to 27% of patients with GCA[27].

These data prompted us to conduct this study to identify clinical, biological, and radiological features of mesenteric involvement in patients with GCA.

MATERIALS AND METHODS

Case reports. We screened 13 French internal medicine tertiary care centers for their cases of patients exhibiting GCA-associated mesenteric ischemia during a 16-year period (1990–2006). Patients were included for study if they fulfilled 3 or more of the 5 GCA criteria of the American College of

Rheumatology [age at onset of disease ≥ 50 years, manifested by new headaches, temporal artery abnormality, elevated erythrocyte sedimentation rate (ESR), temporal artery biopsy][28]. The presence of 3 or more of the 5 criteria yields a sensitivity of 93.5% and a specificity of 91.2% in patients with GCA[28]. The criterion for mesenteric involvement was newly developed abdominal symptoms associated with histological proof of GCA-associated mesenteric vasculitis and/or radiological abnormalities consistent with GCA-associated mesenteric vasculitis, as described[29-31].

Literature review. In August 2006, we performed a Medline® search without time limits using the following subject headings: "giant cell arteritis and mesenteric ischemia," "giant cell arteritis and intestinal infarct," "giant cell arteritis and intestinal angina," and "giant cell arteritis and abdominal pain." We supplemented this search by consulting all the references of interest cited in these articles. For the analysis, we included only references in English and French. We summarized data from selected reports using a standardized data form, and we distinguished definite cases of mesenteric GCA (i.e., those with histopathologic proof) from very probable cases of mesenteric GCA (i.e., those with positive temporal artery biopsy, compatible clinical history of and radiological confirmation for mesenteric arteritis, but without histological proof of GCA in mesenteric arteries).

Statistics. Descriptive statistical methods were used to analyze the data of patients with regard to their outcome. The patients who died were compared to the patients who survived using the Fisher exact test for categorical variables and the Wilcoxon test for continuous variables. Statistical analysis was performed using SAS version 8.2 (SAS Institute, Cary, NC, USA).

RESULTS

Case reports. Seven patients were found to exhibit GCA-associated mesenteric ischemia. One patient [a man with aortic involvement of GCA as shown through magnetic resonance angiography (MRA), who had severe abdominal pain that improved after the daily dosage of steroids was increased] was not included in our study because of the absence of histological or radiological criteria for the diagnosis of GCA-associated mesenteric ischemia.

Illustrative cases. Case 1. A 77-year-old woman presented in our department with a 30-day history of temporal pulsatile pain associated with fever and fatigue. She had a history of breast cancer treated by surgery and radiotherapy 17 years previously. Examination showed evidence of left temporal artery tenderness. Biochemical tests revealed elevated ESR (73 mm/h) and C-reactive protein (CRP; 137 mg/l). Temporal artery biopsy confirmed the diagnosis of GCA. Treatment with steroid was started (prednisone 0.7 mg/kg/day). She was also given prophylactic low molecular weight heparin (enoxaparin 4000 IU/day). Three days later, she presented with acute abdominal pain with occlusion and peritoneal syndrome. Abdominal computed tomography (CT) scan showed extensive small bowel ischemia and severe stenoses of the mesenteric arteries, with a periarterial halo resulting from arterial wall inflammation (Figure 1). Laparotomy revealed extensive necrosis. Resection of 1.8 m of the small intestine was carried out. Pathological findings were consistent with GCA of the superior mesenteric artery. She improved markedly after pulses of steroids (methylprednisolone 250 mg/day, 3 days). CRP level was 8 mg/l after 2 months of steroid therapy. At 1-year followup, she was alive and asymptomatic with prednisone therapy 7 mg/day.

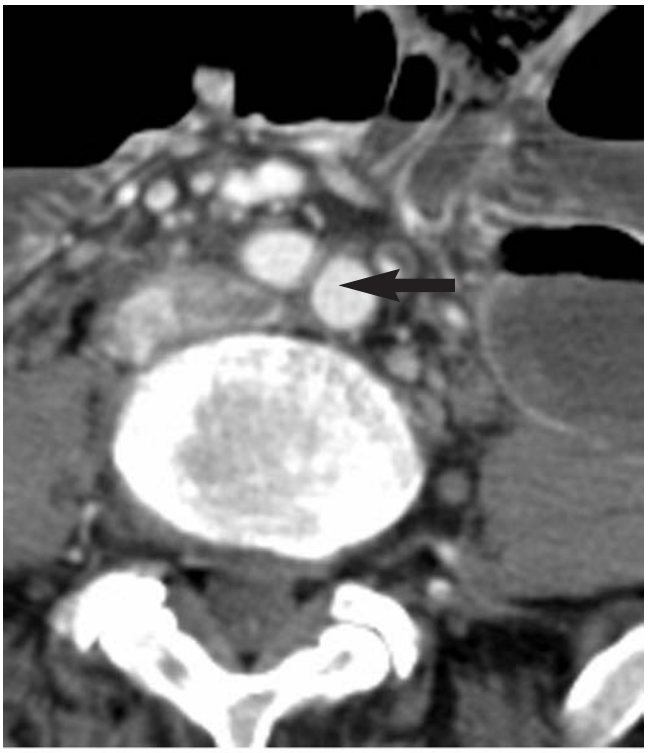

Figure 1. Enhanced CT scan of the abdomen (Patient 3) shows stenosis of the inferior mesenteric artery and circumferential periarterial halo (arrow).

Case 2. A 73-year-old woman with no relevant history was referred to the hospital because of recently developed asthenia and anorexia; her admission examination was unremarkable. Laboratory studies disclosed a severe inflammatory syndrome (ESR 110 mm/h and CRP 137 mg/l). A temporal artery biopsy showed damage consistent with GCA. She was given high-dose steroid therapy (1.5 mg/kg/day), in combination with prophylactic anticoagulation with low molecular weight heparin. On Day 4, she was admitted to the intensive care unit because of a severe shock state and on Day 5 she developed abdominal pain. A CT scan was interpreted as normal. In particular, there was no evidence for mesenteric impairment. A laparotomy revealed an infarction involving the whole colon and a large part of the small bowel. Pathological examination of the resected bowel showed GCA of the mesenteric vessels. She died despite intensive steroid therapy and supportive measures.

Literature review. We identified 27 patients with mesenteric GCA in both English and French literature[3-25,32-34]. Two were excluded because the GCA diagnosis was uncertain[32,33]. Moreover, 3 patients were also excluded because the mesenteric involvement was insufficiently documented[25,33,34]. Among the 22 remaining patients, 16 had definite mesenteric GCA and 6 had very probable mesenteric GCA.

Characteristics of the 28 patients. Counting the 22 patients from the literature and including the 6 cases reported above, we reviewed 28 GCA patients with mesenteric involvement (Table 1). They were 22 women (79%) and their mean age at

Table 1. Characteristics of the 28 patients. Specific signs of mesenteric vasculitis include stenosis or periarterial halo. Nonspecific signs of mesenteric vasculitis include bowel ischemia or peritoneal reaction.

Reference	Sex	Age, yrs	CV Risk Factors	GCA Diagnosis Prior to Mesenteric Ischemia	ESR, mm/h	CRP, mg/l	Radiological Data	Surgery	Survival
8	F	75	Diabetes mellitus	Yes	58	NM	NM	Yes	No
21	F	77	None	Yes	97	NM	NM	Yes	Yes
10	F	78	NM	Yes	120	NM	NM	Yes	No
20	F	69	None	Yes	111	107	NM	Yes	No
9	M	60	NM	Yes	68	NM	NM	Yes	Yes
7	F	75	None	Yes	100	NM	NM	Yes	Yes
24	M	63	None	Yes	NM	132	NM	Yes	No
Patient 1	F	73	Hypertension	Yes	110	137	CT scan: nonspecific	Yes	No
Patient 2	M	56	Smoking	Yes	76	139	CT scan: nonspecific Arteriogram: specific	No	Yes
Patient 3	F	77	None	Yes	73	137	CT scan: specific	Yes	Yes
Patient 4	M	64	Hypertension	Yes	50	29	Ultrasonography: normal CT scan: specific	No	Yes
Patient 5	F	73	None	Yes	100	80	CT scan: specific	No	Yes
18	NM	NM	NM	No	NM	NM	NM	Yes	No
19	F	68	None	No	82	NM	NM	Yes	Yes
17	F	67	None	No	22	NM	NM	Yes	Yes
14	F	82	None	No	NM	NM	NM	Yes	No
12	F	65	None	No	113	NM	CT scan: nonspecific	Yes	Yes
15	F	73	None	No	103	NM	NM	Yes	Yes
6	F	78	NM	No	NM	NM	NM	Yes	No
22	F	75	None	No	71	NM	CT scan: nonspecific Arteriogram: normal	Yes	Yes
11	F	83	None	No	90	NM	CT scan: nonspecific	Yes	Yes
3	F	72	NM	No	38	NM	Arteriogram: specific	No	Yes
4	F	67	None	No	100	27	CT scan and arteriogram: specific	No	No
23	M	87	NM	No	90	NM	NM	Yes	Yes
5	F	80	Diabetes, hypertension	No	45	NM	CT scan, ultrasonography, MRA, and arteriogram: specific	No	Yes
16	F	72	None	No	96	NM	CT scan: nonspecific Ultrasonography and arteriogram: specific	No	Yes
Patient 6	F	72	Hypertension, cholesterol	No	100	35	CT scan: specific	No	Yes
13	F	72	NM	No	NM	NM	Per-operative ultrasonography and CT scan: specific	Yes	NM

NM: not mentioned; CV: cardiovascular; MRA: magnetic resonance angiography; CT: computed tomography.

GCA diagnosis was 72.4 ± 7.1 years. GCA was histologically proved for all patients. Except for age, one or more cardiovascular risk factors was present in 6 patients (21%). Patients presented both constitutional and cranial ischemic signs of GCA (n = 17, 61%), isolated constitutional symptoms (n = 8, 29%), and isolated cranial ischemic signs (n = 3, 10%). The mean ESR was 83 ± 26 mm/h and mean CRP was 91 ± 50 mg/l. In 12 patients, GCA diagnosis preceded the onset of mesenteric ischemia. In these patients, mesenteric ischemia occurred either soon after the initiation of steroid therapy (n = 6, mean time since introduction of steroid 12 ± 11 days) or at the end of the therapy (n = 6, steroid dosage 0 to 10 mg/day). In the other 16 patients, the mesenteric involvement revealed the GCA. Mesenteric ischemia occurred despite treatment with preventive low molecular weight heparin (n = 2), clopidogrel (n = 1), and aspirin (n = 1) in 4 patients. For the other 24 patients, neither anticoagulation nor antiaggregant therapy was mentioned.

Mesenteric ischemia. At diagnosis, all patients presented with abdominal pain. Thirteen patients (46%) presented with occlusive syndrome and 4 others (14%) complained of typical abdominal angina after meals. Digestive bleeding was uncommon (7%). Malabsorption features were not reported. Results of abdominal CT scan, MRA, Doppler ultrasonography, or angiography were described for 14 patients. Ten of them presented radiological evidence of mesenteric vasculitis (i.e., stenosis of the superior or inferior mesenteric artery and/or periarterial halo resulting from arterial wall inflammation) and 5 had involvement of other arteries (e.g., aorta, hepatic artery).

Nineteen patients (68%) required laparotomy. Four of the 9 patients who were treated medically (i.e., with steroids) required a complementary intervention (mesenteric bypass or

angioplasty). The overall mortality rate was 33%. Compared to patients who died, the survivors tended to present (1) more often with constitutional symptoms of GCA, (2) less often with ischemic signs of GCA, and (3) less often with occlusive syndrome (Table 2). Finally, the survivors did not require surgery as often as the patients who died, perhaps because the diagnosis of mesenteric GCA was more frequently done by imaging (Table 2).

DISCUSSION

We describe case histories of 28 patients with GCA-associated mesenteric ischemia. The clinical signs of mesenteric GCA were very nonspecific. Abdominal pain was common. Only 46% of the patients presented with occlusive syndrome, and digestive bleeding or typical abdominal angina after meals was reported by less than 15% of the patients. Imaging explorations were very helpful in the diagnosis of the GCA mesenteric involvement, showing specific abnormalities in 10 of the 14 patients whose symptoms were investigated. The overall mortality rate was 33% and it was lower in patients for whom the diagnosis was performed by imaging.

The occurrence of GCA-associated mesenteric ischemia is probably underestimated, as suggested by systematic postmortem studies[21,25]. Several causes may result in this underestimation. First, before it becomes symptomatic, intestinal ischemia requires involvement of several vessels because of the extensive mesenteric collateral circulation. Second, symptoms of intestinal angina are very nonspecific, resulting in both overlooked diagnosis and misdiagnosis in patients with GCA. Abdominal pains in a patient with GCA may be misinterpreted by physicians, who may think at first of a gastrointestinal complication of the steroid therapy. Third, performing a necropsy leads to diagnostic bias because the majority of GCA patients are alive.

Classic traditional risk factors were present in 21% of patients. Previous studies highlighted the importance of traditional atherosclerosis risk factors in the incidence and development of severe ischemic complications of GCA. In 1998, Duhaut and colleagues found an independent association between smoking or previous arterial disease and GCA in women[35]. In a population-based study from Northwestern Spain, the presence of traditional risk factors of atherosclerosis at the time of GCA diagnosis was found to significantly increase the risk of developing at least one of the severe ischemic complications associated with this vasculitis[36]. Due to this, we cannot exclude a role of these cardiovascular risk factors in the development of mesenteric ischemia.

Because clinical signs and biological abnormalities are very nonspecific, diagnosis of GCA-associated mesenteric ischemia is difficult. Conventional angiography remains the best method for visualization of distal structures, which is helpful in 2 situations: in cases where revascularization is considered, or for the diagnosis of distal vasculitis (e.g., Behçet disease or Churg-Strauss angiitis). By contrast, in proximal stenosis (which is the case for GCA-associated stenosis) less invasive techniques such as ultrasonography, MRA, and CT may be as effective as conventional angiography[29,31,37]. These techniques not only allow study of the lumen but also enable examination of the arterial wall, which is fundamental for the etiologic diagnosis. They distinguish typical irregular atherosclerotic intimal changes with calcified plaques from the inflammatory enlargement of the arterial wall seen in GCA. The reported sensitivity and specificity of duplex ultrasonography are 96% and 92%, respectively, for stenosis of the superior mesenteric artery secondary to atherosclerotic origin[38]. For GCA-associated large-vessel involvement it has been shown that a dark halo around the lumen of the temporal artery had a specificity of 79% to 93%, but a sensitivity of only 40%[39]. The halo confirms a diagnosis of temporal arteritis, but its absence does not rule it out[39]. To our knowledge, the performance of ultrasonography for the diagnosis of mesenteric GCA remains unknown.

Among the 28 patients described here, stenosis of the superior mesenteric artery was observed for 2 out of 3 patients who

Table 2. Characteristics of patients with regard to outcome. Constitutional symptoms include fever, weight loss, anorexia, myalgias, malaise, and fatigue. Ischemic symptoms include headaches, scalp tenderness, jaw or tongue claudication, temporal artery tenderness, and visual disturbances. Specific signs of mesenteric vasculitis include stenosis or periarterial halo. Signs of bowel ischemia or peritoneal reaction were not considered as specific.

Characteristic	Death (n = 9)	Alive (n = 18)	p
Percentage of women	88	78	0.63
Mean age ± SD, yrs	73 ± 6	72 ± 8	0.72
Mean ESR ± SD, mm	100 ± 24	79 ± 26	0.06
Patients with constitutional symptoms, %	67	94	0.09
Patients with other ischemic symptoms, %	75	61	0.67
Patients presented with occlusive syndrome, %	88	27	0.004
Patients presented with abdominal angina, %	11	17	0.85
Patients with specific radiological lesions of mesenteric vasculitis, %	11	45	0.19
GCA diagnosis anterior to mesenteric ischemia, %	56	39	0.45
Patients who required laparotomy, %	89	61	0.20

were examined by abdominal ultrasonography (no halo was described). The main drawback of this technique is that it is operator-dependent. CT scan of the abdomen may be more reliable when evaluating abdominal pain in patients with GCA. The CT scan can show nonspecific signs of bowel ischemia (i.e., enlargement of the bowel wall, absent or poor enhancement of the bowel wall, parietal pneumatosis or peritoneal fluid[13]). Usually, mesenteric vasculitis demonstrates a relatively long length of bowel involvement and a nonsegmental distribution. CT scan may also show more specific signs of mesenteric vasculitis, including the regular concentric stricture leading to narrowing or occlusion of the arterial lumen[40]. This periarterial halo (Figures 1 and 2) is one of the hallmarks of vasculitis. Disadvantages of this technique include nephrotoxicity of the contrast media and limitations of axial, coronal, and sagittal imaging.

Three-dimensional MRA is the noninvasive method of choice to assess neck and aortic arch branch vessel pathology due to its capacity for oblique-plane imaging. It has been suggested that T2-weighted magnetic resonance images are particularly helpful in determining wall thickness. MRA may be capable of detecting wall edema, an indicator potentially important in monitoring response to therapy[31]. The major caveat of this technique is that no studies have been performed to prospectively correlate histomorphology, laboratory measures of active vasculitis, and clinical signs of ongoing vascular disease with imaging findings. Because isolated mesenteric involvement may be the first event in patients with undiagnosed GCA, these imaging techniques may be very helpful in patients with mesenteric ischemia who present with constitutional symptoms or a marked biological inflammatory syndrome. For these patients, the radiologist must be clearly informed of the possible GCA diagnosis, in order to look for subtle signs of abdominal vessel inflammation.

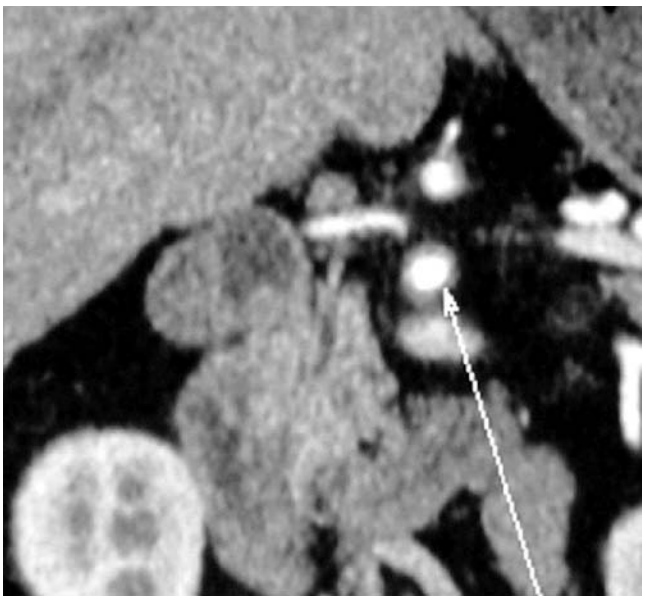

Figure 2. Enhanced CT scan of the abdomen (Patient 4) shows circumferential periarterial halo around the superior mesenteric artery (arrow).

Because mesenteric GCA has been rarely described, there is no prospective study and no consensus about its management. The efficacy of steroid therapy in GCA has been demonstrated. Nevertheless, although it is well known that steroid therapy is very effective in treating constitutional symptoms, it does not fully protect against ischemic complications, which can develop after starting steroid therapy[41,42]. The most studied ischemic complication of GCA was visual loss, and this occurred or deteriorated in 1% to 13% of cases after the start of steroid therapy[42-45]. In our study, mesenteric ischemia occurred soon after the initiation of steroids in 6 out of 28 patients (21%). In this context, low-dose aspirin treatment may be effective in preventing ischemic complications in GCA. In a retrospective study, Nesher and colleagues[46] proved that GCA patients receiving low-dose aspirin treatment were 5 times less likely to experience cranial ischemic complications, despite the fact that these patients had more vascular risk factors. Even if there are few clinical data corroborating this, recent progress in understanding the physiopathology of GCA tends to corroborate this assumption. Indeed, GCA is a T cell-dependent disease[47] and experimental and clinical data[48-54] strongly support a model of GCA with 2 kinds of T cell cytokine production not exclusive of each other (i.e., interferon-γ or interleukin 2), leading to 2 different clinical presentations (i.e., constitutional or ischemic symptoms), with specific treatment for each pathway: steroid and low-dose aspirin. Only 2 out of the 28 reported patients mentioned having been treated with antiaggregant therapy when the mesenteric infarct occurred. Even if these data may not have the relevance of a prospective study, a risk-benefit analysis strongly suggests the use of low-dose aspirin in addition to steroid therapy in order to prevent the ischemic complications of GCA. However, to date, no definite conclusion can be drawn and these hypotheses warrant further investigations.

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