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Clinical Presentation of Multiple Myeloma Mimics Giant Cell Arteritis

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Abstract

Introduction: Giant cell arteritis (GCA) is a form of immune-mediated systemic vasculitis affecting large- and medium-sized arteries. GCA and multiple myeloma share the skeletal-related clinical features such as bone pain occurring at multiple sites and fatigue, besides affecting mostly individuals aged ≥ 50 years old. **Case:** We present a 76-year-old lady who presented to the rheumatology department at University Hospital Limerick, Ireland, with temporal headache and right hip pain. A rheumatology review revealed the patient's GCA probability score (GCAPS) is 10, indicating intermediate probability. Her temporal and axillary artery Doppler ultrasound scan was normal. However, the whole-body computed tomography scan of the skeletal survey revealed multiple lytic lesions. Serum protein electrophoresis recorded a high IgG level of 29.9 g/L and a high monoclonal IgG Kappa in the gamma region of 20.6 g/L. A bone marrow biopsy revealed >10% plasma cell proliferation, consistent with Multiple Myeloma (MM). Upon MM diagnosis, the patient is following the treatment protocol at the hematology department. **Discussion:** The presented case, initially pre-diagnosed as GCA, manifested with temporal headache and right hip pain. These symptoms align with the broad, multifaceted nature of GCA, which involves both cranial and large arteries. Literature supports the notion that GCA and MM share overlapping skeletal-related features, such as multifocal bone pain and fatigue in older adults, and reports suggest a possible inflammatory link between the two. Therefore, careful diagnosis is essential to prevent misdiagnosis and avoid unnecessary steroid treatment. **Conclusion:** Medical doctors are recommended to determine MM-specific CRAB features of patients who are suspected to be affected by GCA to rule out MM.

Keywords

Multiple Myeloma, Giant Cell Arteritis

1. Introduction

Giant cell arteritis (GCA) is an immune-mediated vasculitis of unknown etiology characterized by granulomatous inflammation affecting large- and medium-sized arteries [1]. GCA leads to vascular endothelial injury, lymphocyte proliferation, and giant cell formation, causing acute and chronic damage 1. It affects older individuals aged ≥ 50 years, being more common in females at a ratio of 2.5:1 [2].

Classic symptoms of cranial (C-GCA) vasculitis include headache, scalp tenderness, jaw claudication, and visual loss [2]. However, GCA could encompass inflammation of extra-cranial (large-vessel, LV-GCA) arteries leading to aneurysms or arterial stenoses. This results in a shift in the clinical phenotype from classical to systemic symptoms, such as fatigue and weight loss, as well as limb claudication [3].

GCA has a strong association with polymyalgia rheumatica (PMR), with up to 50% of the patients showing comorbidity at the time of presentation with suspected GCA [1]. PMR is an inflammatory disorder of unknown etiology affecting periarticular structures, characterized by the abrupt onset of symmetrical pain and stiffness in the neck, shoulders, and hip girdles [4]. Furthermore, some cancers have been reported to be linked to GCA/PMR due to immune system dysregulation, such as multiple myeloma/monoclonal gammopathy of undetermined significance (MGUS) [5].

We present a case admitted to the rheumatology ward with GCA symptoms, with temporal headache and right hip pain, which ultimately required laboratory investigations, biopsy, and multiple imaging modalities to elucidate the patient's final diagnosis of Multiple Myeloma (MM), initially presumed to be GCA.

2. Case Presentation

A 76-year-old lady was referred to the rheumatology service for suspicion of Giant Cell Arteritis (GCA). Her symptoms started around four months ago with a temporal headache. She then developed an acute onset of right hip pain over three days with no report of trauma. Her medical background revealed several comorbidities, including Monoclonal Gammopathy of Undetermined Significance (MGUS).

On physical examination, the patient was clinically and vitally stable. Her blood test showed a high adjusted calcium level of 2.65 mmol/L, mild normocytic anemia with a hemoglobin concentration of 9.9 g/dL, progressively deteriorating renal function with creatinine rising from 94 to 303 μmol/L and urea rising from 8.1 to 12.4 mg/dL It has also revealed mildly raised inflammatory markers: an erythrocyte sedimentation rate of 35 mm/hr and C-reactive protein (CRP) of 9 mg/L. On admission, she was started on 60 mg of prednisolone daily, given the possible diagnosis of GCA. A rheumatology review revealed her GCA probability score (GCAPS) was 10, indicating an intermediate probability according to the GCA probability stratification algorithm [6]. This was the sum of the GCAPS weightages: +3 for age 76 years old, +2 for female sex, +3 for onset of symptoms < 6

weeks, +1 for 9 mg/L CRP, and +1 for headache.

Two days after admission and from the start of the prednisolone, the patient had further investigations. She had a temporal and axillary artery Doppler ultrasound scan as per the EULAR recommendation [7], which was normal. The X-ray of her hip showed several circumscribed, punched-out areas of lucency. Her computed tomography (CT) scan of the abdomen and pelvis revealed multiple lytic lesions throughout the bilateral proximal femoral, bilateral iliac, and ribs. A skeletal survey confirmed the lytic lesions (**Figure 1**).

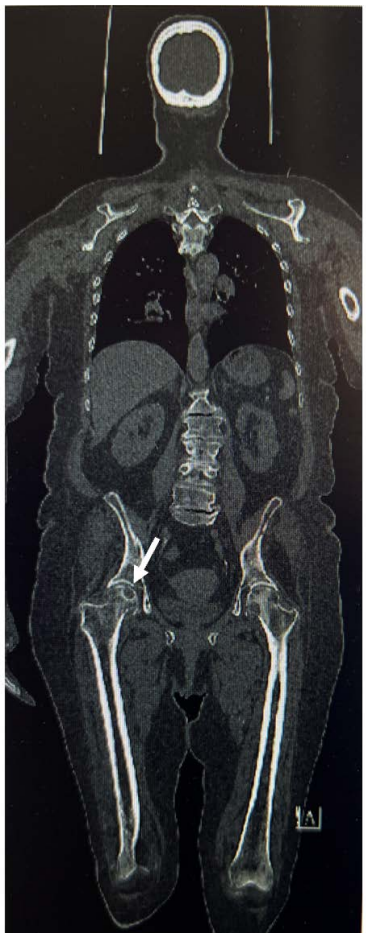

Figure 1. Skeletal survey whole-body CT scan reveals bone lesions (arrow) of the proximal right femur.

Extensive osteolytic lesions identified a more plausible alternative diagnosis, incompatible with vasculitis. These findings were skeptical of the progression of MGUS to Multiple Myeloma (MM). As such, the prednisolone was tapered down after the imaging results. Furthermore, serum protein electrophoresis recorded a high IgG level of 29.9 g/L and a high monoclonal IgG Kappa in the gamma region of 20.6 g/L. She also underwent a bone marrow biopsy, which was suggestive of MM and revealed trilineage hematopoiesis with increased plasma cells consistent with plasma cell dyscrasia (**Figure 2**).

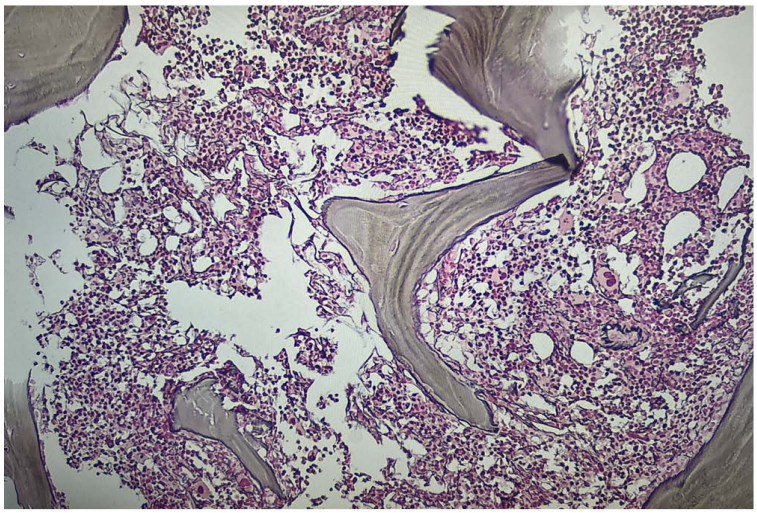

Figure 2. Hematoxylin and eosin-stained bone marrow biopsy demonstrating an increased number of plasma cells with mostly interstitial distribution and extensive amyloid deposition.

The patient was then scheduled within 2 weeks for the outpatient hematology clinic and was planned for the RVd (Revlimid, Velcade, Dexamethasone) protocol.

3. Discussion

Our case has been pre-diagnosed as GCA. However, upon presentation to the rheumatology department with temporal headache, this is consistent with the consensus description of GCA as a multifaceted vasculitis syndrome encompassing inflammation of the temporal arteries and their branches, the aorta and its branches, and girdles, either isolated or combined [1]. Furthermore, LV-GCA and MM share a spectrum of nebulous, nonspecific skeletal-related clinical features, such as bone pain at multiple sites and fatigue, and affect mostly individuals aged ≥ 50 years [1] [8]. The literature presents some case reports describing mimics of clinical features between GCA and MM (**Table 1**) [9]-[10]. Moreover, a hypothetical association between MM and GCA has been reported based on both having a common inflammatory process [9].

The reported association between GCA and MM is based on a limited number of case reports and should be interpreted as a hypothesis rather than a firm, definitive causal connection. In cases where patients present atypically or discordantly with GCA, with abnormal hematologic findings or unexplained systemic features, consideration of evaluating for plasma-cell dyscrasia, such as MM, may be warranted.

4. Conclusion

A thorough anamnesis, clinical instrumental examination, and laboratory investigations are crucial for clinical reasoning in GCA and MM. Since MM and GCA share skeletal-related clinical features, it is recommended to stratify suspected

Table 1. Summary of retrieved case reports on GCA and MM mimicking clinical presentations.

Case Summary	Investigations	Reference
- A 78-year-old lady presented with severe anemia, weight loss, and monoclonal gammopathy. - Clinical features were suggestive of MM. - No typical symptoms of GCA.	- A positron emission tomography (PET) scan showed extensive 2-flourodeoxy-glucose uptake in the vascular tree consistent with arteritis. - A temporal artery biopsy established the diagnosis of GCA.	[9]
- A 78-year-old man presented with a monoclonal peak observed by his general practitioner. - Typical GCA complaints were presented. - No CRAB criteria present. - Suggesting the coexistence of GCA and MM.	- Temporal artery biopsy gave nonspecific findings, yet the diagnosis of GCA was not excluded. - A diagnosis of GCA was confirmed according to the 1990 ACR criteria and the 2018 update of the EULAR recommendations. - A monoclonal IgG/kappa peak was detected on serum protein electrophoresis, and a bone marrow aspirate revealed 26% clonal bone marrow plasma cells, confirming MM.	[10]
- A 60-year-old lady presented with acute monocular blindness and a high erythrocyte sedimentation rate (ESR), initially misdiagnosed as GCA. - No symptoms of headache, jaw claudication, or unexplained fever. - No symptoms of polymyalgia rheumatica.	- Temporal artery biopsy showed no evidence of any inflammatory or granulomatous infiltrate to rule out GCA. - A monoclonal band was detected and immunotyped as IgA Kappa. - A review of the retinal images was performed in retrospect, and this supported the diagnosis of hyperviscosity syndrome. - MM presented atypically in this instance, with a hyperviscosity syndrome causing blindness.	[11]

GCA patients by GCAPS. Patients with an intermediate or high GCAPS with a negative GCA ultrasound should warrant additional tests for an alternative diagnosis. The MM-established CRAB features for diagnosis of hypercalcemia, renal failure, and anemia linked with osteolytic bone lesions should be further considered for differential diagnosis. Clinicians must be aware of the differential diagnoses of MM mimickers to avoid the steroid burden while continuing to treat as GCA.

Informed Consent

The patient has provided informed consent.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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