Imaging of Multiple Myeloma

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KEYWORDS

• Imaging • Multiple myeloma • Classification • Treatment • Complications

KEY POINTS

- Diagnosis of multiple myeloma is based on a combination of clinical findings, laboratory studies, bone marrow biopsy, and imaging findings.
- Imaging plays an important role in identifying the extent of the disease, disease process, guiding biopsies, and diagnosing associated spinal and intracranial complications. It also plays an important role in the staging, evaluating response to therapy, and monitoring for recurrence.
- Multiple myeloma and related plasma cell proliferative disorders (PCPDs) have a diverse set of clinicopathologic findings and with those present unique and diverse findings on neuroimaging, not only from the disease itself but from complications of the disease and treatment-related complications. Familiarity with these findings is valuable for clinicians and radiologists alike.
- This article describes the imaging findings associated with common neurologic complications seen with multiple myeloma and related PCPDs.

INTRODUCTION

Multiple myeloma is a malignant neoplasm of plasma cells that produces monoclonal immunoglobulins. Each year in the United States, approximately 20,000 new cases are diagnosed with approximately 11,000 deaths. Most patients are older than 60 years with a median age at diagnosis of 66 years old.[1–3]

Multiple myeloma is a cytogenetically and molecularly diverse neoplasm leading to a wide range of clinical disease. Multiple myeloma is believed to be a progression of a premalignant stage called monoclonal gammopathy of undetermined significance (MGUS).[4–9] Patients present at varying stages from the premalignant MGUS, to an intermediate asymptomatic stage termed smoldering myeloma, to the symptomatic

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stage termed multiple myeloma.[4–9] Patients may also present at a stage before multiple myeloma and progress all the way to multiple myeloma at varying rates depending on several cytogenetic and molecular factors.

MGUS is predominantly seen in patients older than 50 years with a prevalence of approximately 3% in the population 50 years and older, 5% in the population 70 years and older, and 7.5% in the population 85 years and older.[1–3]

The purpose of this review article is to describe the imaging findings associated with common neurologic complications seen with multiple myeloma and related plasma cell proliferative disorders (PCPDs). The diagnosis, classification of multiple myeloma and PCPDs, staging and risk stratification, and treatment are briefly described as they pertain to neurologic complications. A brief overview of the imaging of multiple myeloma and then specific imaging findings associated with common neurologic complications seen with multiple myeloma are described.

CLASSIFICATION OF MYELOMA, MONOCLONAL GAMMOPATHY OF UNDETERMINED SIGNIFICANCE, AND RELATED PLASMA-CELL PROLIFERATIVE DISORDERS

The International Myeloma Working Group (IMWG) has set diagnostic criteria for multiple myeloma and MGUS, the most recent revised criteria of 2014 is described in (**Box 1**).[4–9] The IMWG divides the premalignant MGUS and related PCPDs into 6 classes based on several clinicopathologic criteria; non-immunoglobulin (Ig)M MGUS, IgM MGUS, light chain MGUS, solitary plasmacytoma, POEMS syndrome, and systemic amyloid light chain (AL) amyloidosis.[4–9] As previously described, each of these can then progress onto an intermediate stage before becoming multiple myeloma. Also, as described, patients can present at any stage from MGUS to myeloma.[4–9]

IMMUNOGLOBULIN M, NON-IMMUNOGLOBULIN M, AND LIGHT CHAIN MONOCLONAL GAMMOPATHY OF UNDETERMINED SIGNIFICANCE

The first 3 divisions are based on the monoclonal or M-protein, which is secreted by the clonal plasma cells.[4–9] There are the non-IgM MGUS, IgM MGUS, and light chain MGUS. The IgM MGUS is usually compromised of IgG, IgA, and much less frequently IgD and IgE monoclonal gammopathies.[4–9] The PCPD is classified as MGUS when the serum monoclonal protein is present but less than 30 g per liter and clonal bone marrow cells are less than 10%.[4–9] In addition, there must be no end-organ damage from the monoclonal gammopathy such as hypercalcemia, renal insufficiency, anemia, and bone lesions (the so called CRAB features) or amyloidoisis.[4–9] Smoldering myeloma will have a non-IgM serum monoclonal protein more than 30 g per liter

Box 1
The International Myeloma Working Group, 2014

Non-IgM MGUS

IgM MGUS

Light chain MGUS

Solitary plasmacytoma

POEMS syndrome

Systemic AL amyloidosis

Data from Refs.[4–9]