Thoracic Spine Lesion with Paralysis a rare presentation of Plasmacytoma - a case study

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CASE PRESENTATION

CC: 50-year-old male with bilateral lower extremity weakness

HPI: 50-year-old male with no past medical history who presents to emergency department complaining of worsening bilateral lower extremity numbness and weakness and difficulty ambulating. He reports having a recent upper respiratory infection which has resolved. One week ago, he developed numbness in his lower extremities that progressed to weakness and eventually became worse and the patient as unable to ambulate. He was unable to ambulate the preceding three days before the ER visit. Upon further collecting history, patients reported to have intermittent upper back pain, dull nagging with variation and worse at night for the past month which at time radiated to the front of the chest. Pain was slightly alleviated by using heating pads and with use of ibuprofen as needed for pain control.
The patient denies having any trauma, headache, rash, fevers, chills, nausea, vomiting, or diarrhea.

❑ **Physical examination:**
Vital signs were stable.
Neurological examination: Awake alert oriented to time, place and person
Pertinent positive findings were.
Bilateral Lower extremity Range of motion was decreased, power reduced 2-3/5, and decreased sensation below the waistline.

Investigation:
CBC: Normocytic normochromic anemia slightly reduced Hb
CMP: mildly decreased potassium respectively.
CT scan of the head without contrast shows no acute abnormalities.
Lumbar puncture CSF results: elevated protein level of 121 mg/dL with normal WBC count.

Provisional diagnosis:
Lower extremity paralysis unknown etiology.
Guillain Barre syndrome
Admitted to intensive care unit for close monitoring and with neuro-check to assess the progression of the symptom, neurology was consulted; a detailed neurologic exam showed increased deep tendon reflexes of bilateral knees and ankles.

CT scan of the chest with contrast shows an
- Aggressive appearing 4.2 cm lytic lesion at the T1 spinous process with an osseous erosion and an apparent extension into the spinal canal;
- A left 7th rib mass measuring 6.4 x 3.4 cm; a 1.1 cm lytic lesion at T12.
- Small lytic lesions within the right 2nd, 4th and 5th ribs and the T7 vertebral body;
- A 0.5 cm focal sclerosis at T10. To complete the workup,

CT scan of the abdomen and pelvis shows
diffusely scattered osseous lytic lesions;

MRI of the brain shows
No acute ischemia and minimum of 3 enhancing skull lesions with the largest towards the right occipital bone.

MRI of the entire spine shows:
- Osseous metastatic lesions throughout with the largest along the posterior elements of T1 involving the spinous process and lamina with extension towards the transverse process causing severe spinal canal stenosis from C7-T1 to T1-T2 and cord compression,
- Bone metastases in the pelvis and a grade 1 anterolisthesis of L4 over L5 with moderate bilateral neuroforaminal narrowing with likely contact of the exiting bilateral L4 nerve roots.

❑ **Neurosurgery consulted.**
Patient underwent T1 posterior thoracic laminectomy with resection of a large extradural tumor.

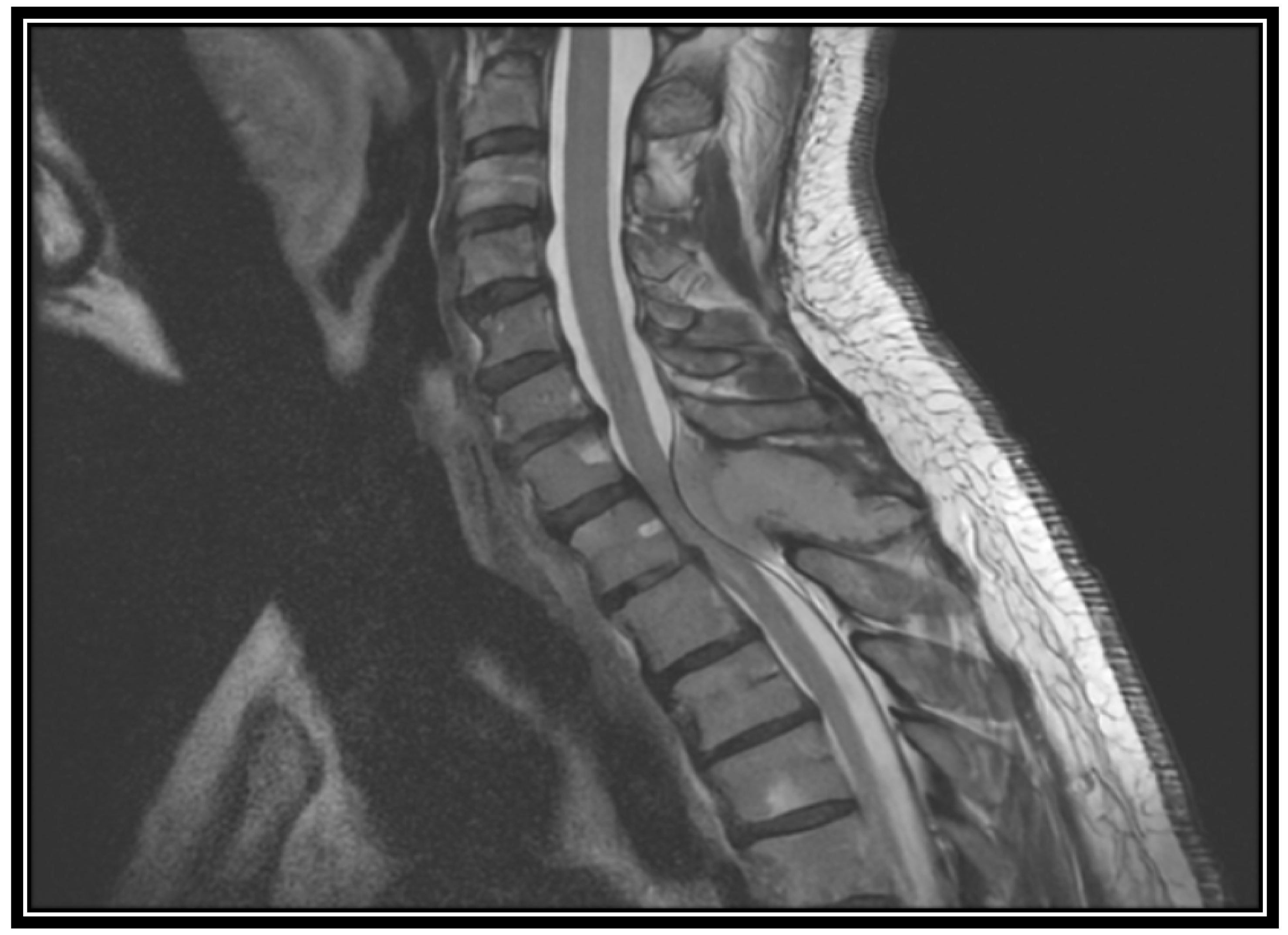
Post surgical course:
Patients tolerate the surgery well and reports improvement in strength and numbness in the lower
extremities.
Post operative day 1: ambulated with physical therapy.
Hospital course: His numbness and weakness continues to improve until discharge several days following surgery

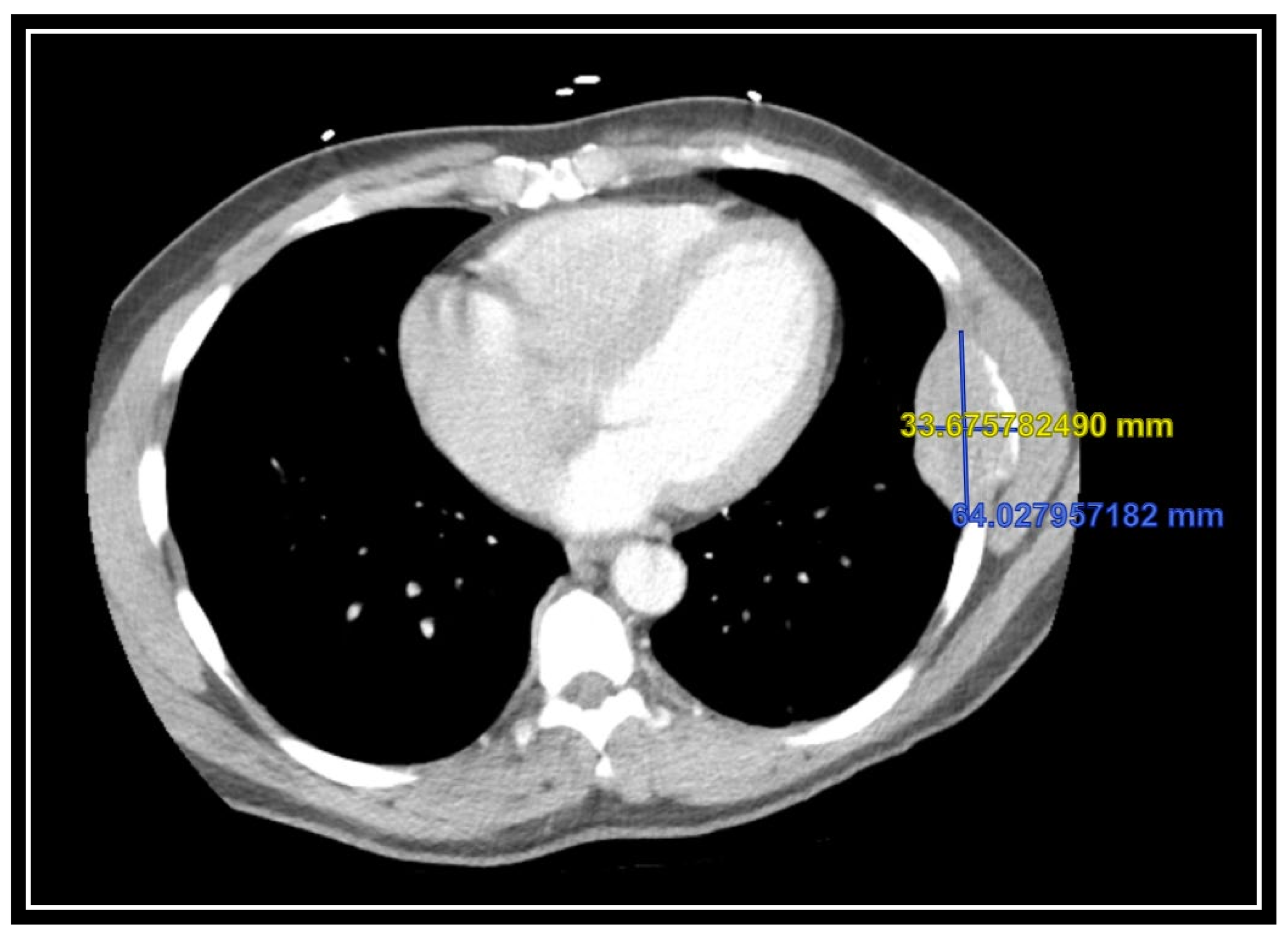
Upon discharge:
Referred to an oncologist for further management.

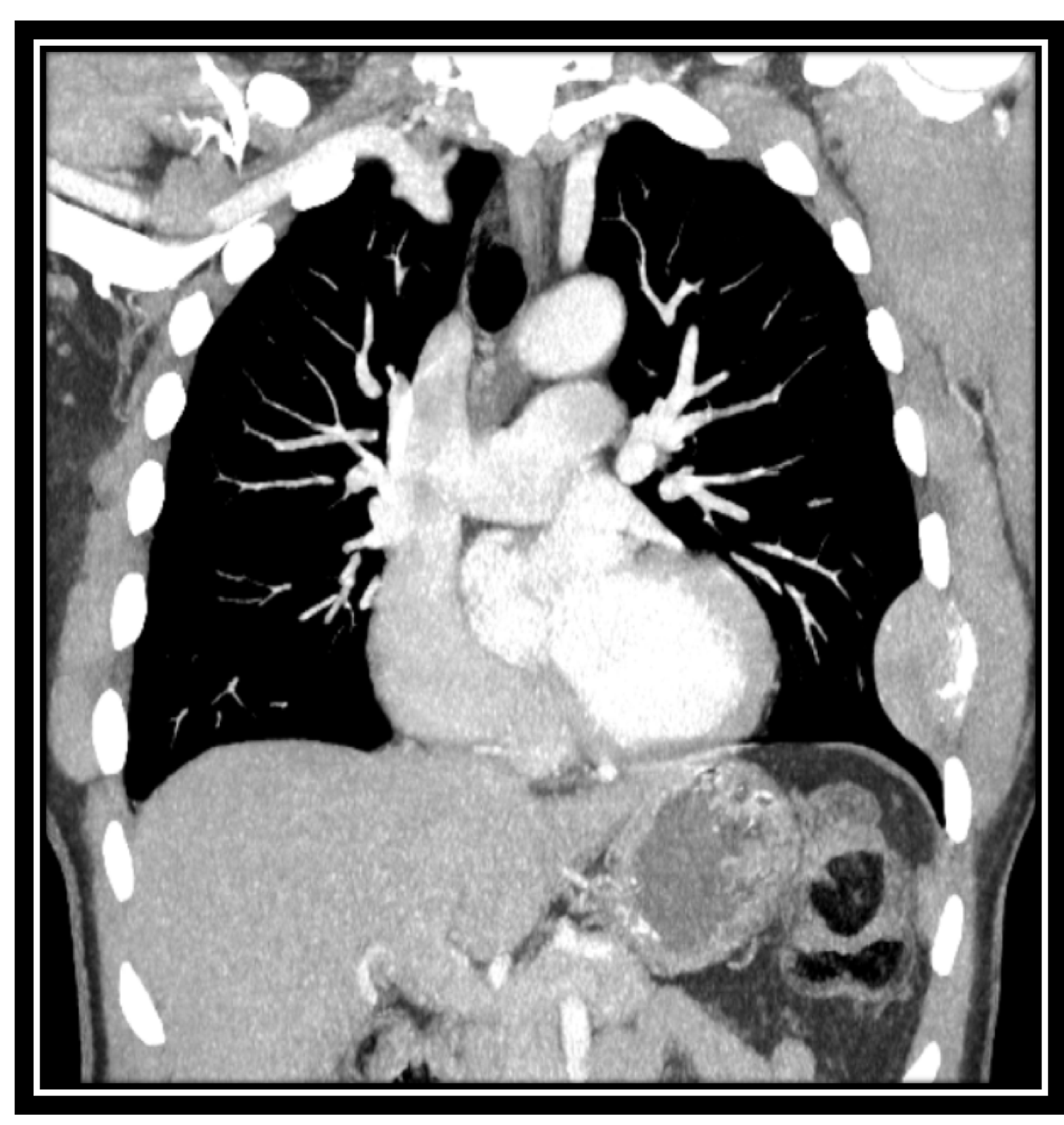
Histopathology report:
The pathology report of the extradural mass indicates that the mass is a **plasmacytoma with cells positive for CD138, negative for AE1/AE3, S100 and CD45,** while in-situ hybridization is positive for Lambda but negative for Kappa. A Serum protein electrophoresis is positive for M-spike with elevated Gamma globulins.

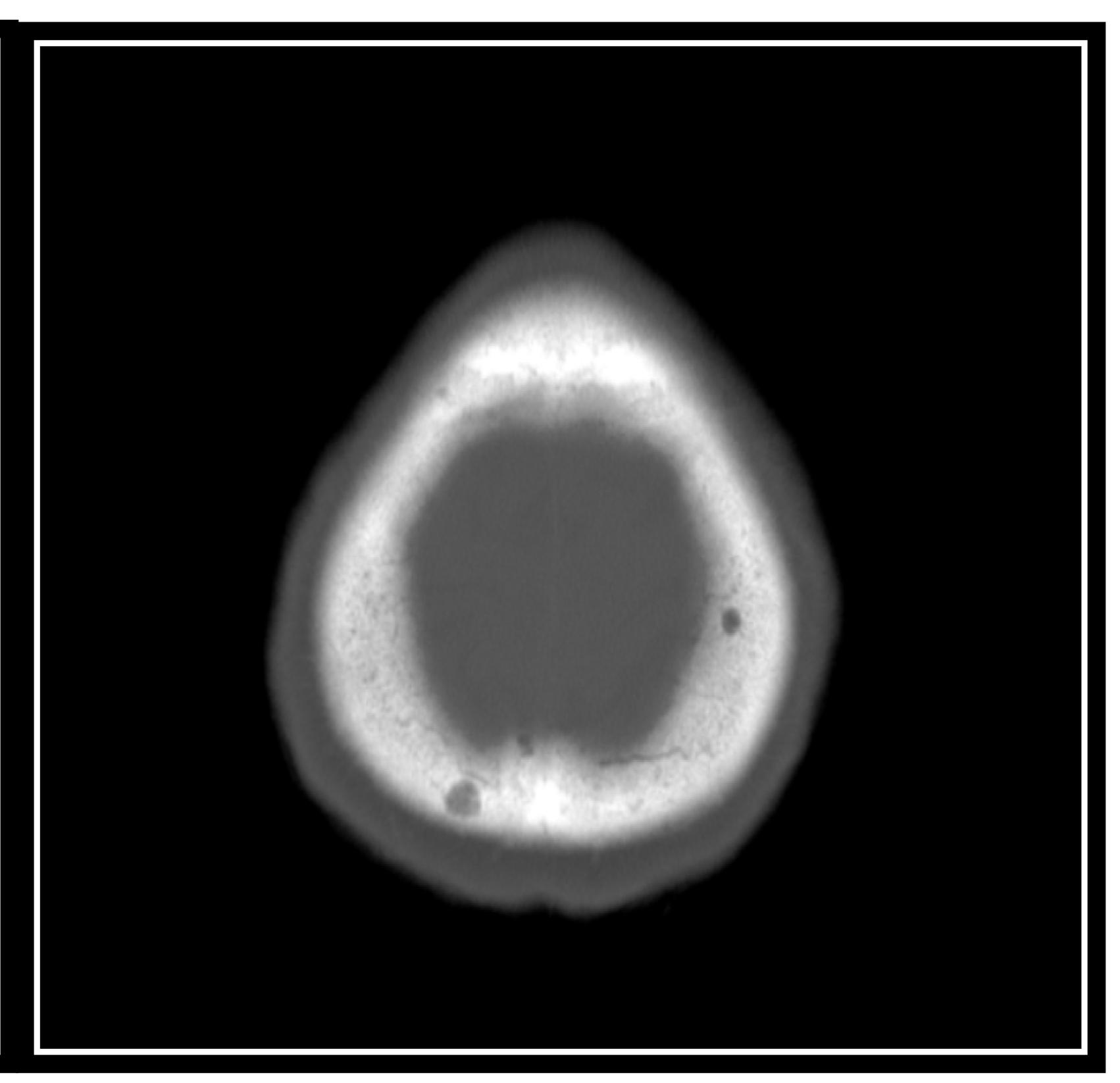
Histopathological diagnosis: plasmacytoma

Imaging









Discussion

Multiple myeloma

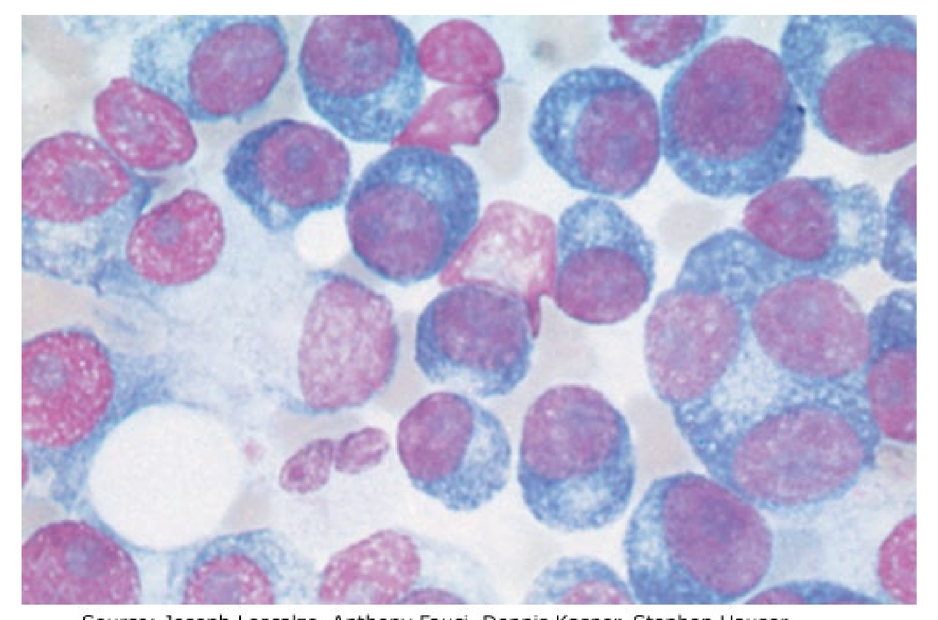
DEFINITION

Multiple Myeloma represents a malignant proliferation of plasma cells derived from a single clone. The tumor, its products, and the host response to it result in a number of organ dysfunctions and symptoms, including bone pain or fracture, renal failure, susceptibility to infection, anemia, hypercalcemia, and occasionally clotting abnormalities, neurologic symptoms, and manifestations of hyper viscosity.

ETIOLOGY

The cause of myeloma is not known. Myeloma occurred with increased frequency in those exposed to the radiation of nuclear warheads in World War II after a 20-year latency. Myeloma has been seen more commonly than expected among farmers, wood workers, leather workers, and those exposed to petroleum products.

INCIDENCE AND PREVALENCE: In 2021 in the United States, 34,920 new cases of myeloma were estimated to be diagnosed, and 12,410 people were estimated to die from the disease. Myeloma increases in incidence with age. The median age at diagnosis is 69 years; it is uncommon under age 40. Males are more commonly affected than females, and blacks have nearly twice the incidence of whites.



Source: Joseph Loscalzo, Anthony Fauci, Dennis Kasper, Stephen Hauser, Dan Longo, J. Larry Jameson: Harrison's Principles of Internal Medicine, 21e
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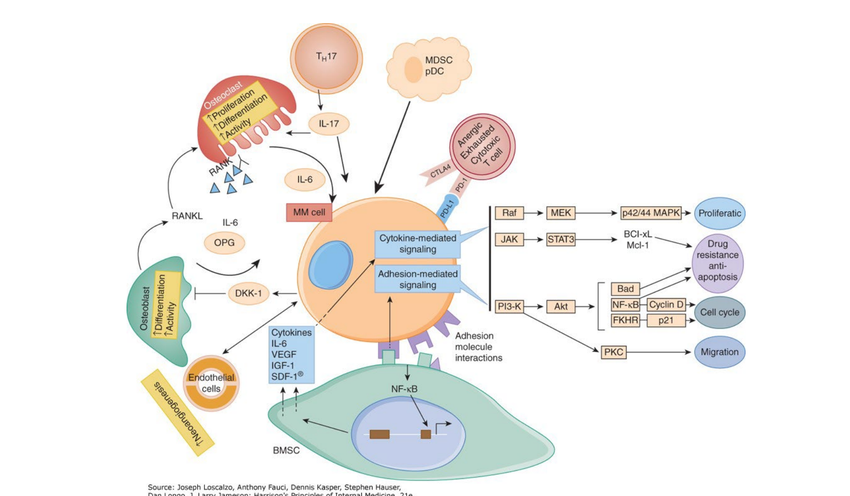

Pathogenesis of multiple myeloma. Multiple myeloma (MM) cells interact with bone marrow stromal cells (BMSCs) and extracellular matrix proteins via adhesion molecules, triggering adhesion-mediated signaling as well as cytokine production. This triggers cytokine-mediated signaling that provides growth, survival, and antiapoptotic effects as well as development of drug resistance. Additional bidirectional interactions lead to inhibition of osteoblast and increase in osteoclast activity which leads to bone-related issues in myeloma. Similar interactions with immune microenvironment lead to augmentation of tumor promoting immune responses and suppression of tumor protective immune responses, overall allowing myeloma cell growth. (Adapted from G Bianchi, NC Munshi: Blood 125: 3049, 2015.)

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Bone pain is the most common symptom in myeloma, affecting nearly 70% of patients. Persistent localized pain usually signifies a pathologic fracture. The bone lesions of myeloma are caused by the proliferation of tumor cells, activation of osteoclasts that destroy bone, and suppression of osteoblasts that form new bone. **Others:** renal failure, Normocytic and normochromic anemia. Although neurologic symptoms occur in a minority of patients, they may have many causes. Hypercalcemia may produce lethargy, weakness, depression, and confusion. Hyperviscosity may lead to headache, fatigue, shortness of breath, exacerbation or precipitation of heart failure, visual disturbances, ataxia, vertigo, retinopathy, somnolence, and coma. Bony damage and collapse may lead to cord compression, radicular pain, and loss of bowel and bladder control. Infiltration of peripheral nerves by amyloid can be a cause of carpal tunnel syndrome and other sensorimotor mono- and polyneuropathies.

DIAGNOSIS AND STAGING

The diagnosis of myeloma requires marrow plasmacytosis (>10%), a serum and/or urine M component, bone marrow plasma cells are CD138+ and either monoclonal kappa or lambda light chain positive. Two important variants of myeloma are solitary bone plasmacytoma and solitary extramedullary plasmacytoma. These lesions are associated with an M component in <30% of the cases, they may affect younger individuals, and both are associated with median survivals of ≥10 years. Solitary bone plasmacytoma is a single lytic bone lesion without marrow plasmacytosis. Extramedullary plasmacytomas usually involve the submucosal lymphoid tissue of the nasopharynx or paranasal sinuses without marrow plasmacytosis. Both tumors are highly responsive to local radiation therapy. If an M component is present, it should disappear after treatment. Solitary bone plasmacytomas may recur in other bony sites or evolve into myeloma. Extramedullary plasmacytomas rarely recur or progress. **Serum protein electrophoresis and measurement of serum immunoglobulins and free light chains are useful for detecting and characterizing M spikes, supplemented by immunoelectrophoresis.** A 24-h urine specimen is necessary to quantitate Bence Jones protein (immunoglobulin light chain) excretion. The serum M component will be IgG in 53% of patients, IgA in 25%, and IgD in 1%; 20% of patients will have only light chains in serum and urine. Chest and bone radiographs may reveal lytic lesions or diffuse osteopenia. Magnetic resonance imaging (MRI) offers a sensitive means to document extent of bone marrow infiltration and cord or root compression in patients with pain syndromes.

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