CLINICAL VIGNETTE

Multiple Myeloma Presenting as Abnormal Liver Tests

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A 75-year-old female presented to gastroenterology with non-specific upper abdominal discomfort, mild nausea, decreased appetite and abnormal liver tests. She was seen in urgent care and her labs included AST 247 mg/dl, alkaline phosphatase 140 (nl 113), ALT 356 mg/dl, Hgb 11.4 g/dl, PLT 145, total bili 1.1, total protein 10.0 (nl 8.2), albumin 3.6, MCV 96, calcium 9.6. Ultrasound of abdomen was reported to show mild fatty liver. Gastrointestinal Review included: one bowel movement per day with no melena or rectal bleeding. No heartburn or weight loss. She never had a screening colonoscopy, but had annual FOBT by her primary care doctor that were negative. Past medical history was negative other than femoral fracture 40 years ago. She has no other medical problems and denies alcohol use, smoking or illicit drugs. She is on no medications and no supplements. Physical exam included: BP 124/68, pulse 78, height 5 foot 2, weight 120 pounds, BMI 23. Skin: no jaundice or spider angiomata. Abdomen was soft, nontender without hepatosplenomegaly. Additional labs were requested including: ANA 1:160, alpha-fetoprotein 17.8, ferritin 885 (NL <180), iron 156, percent saturation 60, TIBC 259. Total protein 10.2 and albumin 3.6. Patient is carrier for hemochromatosis H63D. Celiac serology was negative other than markedly elevated IgA of 4,630 (nl <426). CT of the abdomen pelvis and MRI of abdomen were unremarkable. Upper endoscopy showed a 1 cm hiatal hernia with normal biopsies of the esophagus stomach and small bowel. Colonoscopy showed mild sigmoid diverticulosis with normal random biopsies of the colon.

She was referred to oncology for the abnormal IgA pattern with a monoclonal gammopathy and plasma cell disorder.

Hematology evaluated for amyloid versus multiple myeloma versus smoldering underlying myeloma. Results included: LDH 330 (nl <256), beta-2 microglobulin 2.9 (nl 2.10), kappa light chains elevated at 13 (nl,1.94). UPEP normal, SPEP: Monoclonal IgA, Sed rate 76 (nl <25). Bone marrow showed sheets of plasma cells 70% and no amyloid on Congo red stain.

Multiple myeloma often presents with bone pain from lytic lesions on x-ray. Labs show increased total serum protein concentration in the presence of a monoclonal protein in the serum or the urine. Systemic signs or symptoms suggestive of malignancy include unexplained anemia, hypercalcemia, and acute renal failure with bland urinalysis or nephrotic syndrome. Multiple myeloma assessment includes serum protein electrophoresis (SPEP) with immunofixation and quantitation of the immunoglobins, in addition to routine urinalysis and 24-hour urine collection for electrophoresis (UPEP) and immunofixation. Other studies include: Bone marrow aspiration and biopsy with immune phenotyping, conventional cytogenics, and fluorescent in situ hybridization (FISH).

Teaching points: The value of celiac serology in the evaluation of abnormal liver tests. Celiac serology with IgA oftentimes detects abnormally low IgA, but in this case found very high IgA. About 40% of patients with celiac disease have abnormal liver tests. This case also emphasizes the importance of screening for multiple myeloma in patients with increased total protein.

There are multiple case reports describing liver function abnormalities as the presenting sign of multiple myeloma.[1-3] Although liver disease as the presenting symptom of myeloma is relatively rare, overt liver involvement has been described in up to 40% of cases of myeloma at autopsy.[1] Other liver pathology associated with myeloma includes splenomegaly, ascites, or even acute liver failure from either amyloid deposition or plasma cell infiltration.[3] In our case, the presenting symptoms was a slight elevation of liver transaminases, which prompted her evaluation.

After diagnosis, she was fully staged, and PET/CT and MRI of liver showed no overt liver involvement. We reviewed treatment option with her, and recommended standard front line therapy with combination Lenalidomide, Dexamethasone, and Bortezomib. However, she elected to participate in a clinical trial, and was assigned to an arm of therapy with Liposomal Doxorubicin, Dexamethasone, and Bortezomib. After 3 months of therapy, her liver transaminases normalized, and the only remaining abnormality was a slight elevation in Alkaline Phosphatase. Her monoclonal protein also significantly improved. She opted to transition to maintenance therapy, and has remained clinically stable, now two years from diagnosis. If she does progress again, she can consider 2nd line therapies with immunotherapy options, or even CAR T-Cell therapy.

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