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RESEARCH ARTICLE

Hyperbilirubinemia an Atypical Presentation of Multiple Myeloma

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Abstract

Multiple Myeloma (MM) is a plasma cell malignancy commonly diagnosed in patients over 60. The most common presentations of MM include back pain, fatigue, weight loss, and renal dysfunction. Jaundice, as the initial presentation has rarely been reported. Literature review revealed several cases reporting this unusual presentation and their presumed pathophysiologic mechanisms.

Jaundice in MM can result from pre-hepatic, intra-hepatic, or post-hepatic causes. Autoimmune hemolytic anemia (AIHA) is an example of a pre-hepatic cause. Intra-hepatic causes include hepatic deposition of amyloid or light chains, infiltration of plasma cells, or inflammation due to increased cytokine activity. Post-hepatic processes are most commonly the result of bile duct obstruction by pancreatic plasmacytomas.

In this review, we discuss the various pathophysiologic mechanisms that can lead to jaundice in MM. The aim is to broaden the differential diagnosis of jaundice, especially in an elderly patient with no underlying liver disease.

Timely identification of MM as the cause of jaundice is critical as prompt diagnosis and treatment of underlying MM result in more favourable outcomes.

Keywords: Jaundice, Multiple Myeloma, Hyperbilirubinemia

Introduction & Background:

Multiple Myeloma (MM) is a malignant neoplasm of plasma cells characterized by an increase in monoclonal protein, leading to organ damage. It represents 1.8% of all the new cancer cases in the United States [1] and occurs predominantly in the older population with a mean age of diagnosis of 70 years [1]. Although the most common presentations in MM are bone pain (60%), fatigue (30%), and weight loss (25%) [2], a myriad of other presentations have also been described in the literature (Table 1). Tathineni et al. described uncommon manifestations of multiple myeloma, including proptosis, retinal hemorrhage, vertigo, pancreatitis, abdominal pain, and diabetes insipidus [3]. Although the liver is commonly involved in MM [4, 5], jaundice as the initial presentation is rare.

Table 1: Table describing the common and uncommon presentations of multiple myeloma

Variable Presentations of Multiple Myeloma	
Common	Uncommon
Hypercalcemia	Proptosis
Anemia	Retinal detachment
Bony pain	Mesenteric ischemia
Renal impairment	Vertigo
Fatigue	Cranial nerve palsies
Weakness	Dysphagia
Paresthesias	Jaundice

Jaundice manifests as a yellow discoloration of the skin due to elevated serum bilirubin levels. Normal bilirubin levels metabolism involves the breakdown of senescent red blood cells (80%) and heme containing products in the liver and muscle (20%). MM has a rare association with AIHA, which can also increase bilirubin levels. Once bilirubin is released, it binds to albumin and reaches the sinusoidal surface of the hepatocyte. It then disassociates from albumin and enters the hepatocytes. In the hepatocyte, bilirubin binds with glucuronic acid to form conjugated bilirubin. The deposition of various proteins/light chains associated with MM can impair this process. Conjugated bilirubin is excreted in the bile, which enters the bile duct and then drains into the second part of the duodenum. Extramedullary tumors of the organs surrounding

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the common bile duct can lead to jaundice. This article highlights jaundice as one of the possible presentations of MM, shedding light on potential mechanisms and the need to include MM in the differential of jaundice.

Methods:

We searched "PubMed" for keywords "jaundice" or "hyperbilirubinemia" AND "multiple myeloma". The abstracts were reviewed, which yielded articles on obstructive jaundice, non-obstructive jaundice, liver failure, and concepts related to hyperbilirubinemia and paraproteinemia. The references to these articles were also reviewed for any other presentations.

Result:

False Hyperbilirubinemia

Paraproteins represent non-functional monoclonal immunoglobulins and fragments produced in increased quantities by malignant plasma or B cells. They can interfere with automated chemistry assays, including serum bilirubin [6–9]. Dutta et al. [10] described a case of a 60-year-old patient who presented with fatigue and was found to have highly elevated bilirubin in the setting of an unremarkable physical exam. The test was repeated with a different bilirubin assay kit revealing normal bilirubin. It was hypothesized that elevated bilirubin was due to a precipitation reaction between the paraproteins and solubilizing agent. Further workup with serum and urine electrophoresis demonstrated a prominent M band, and bone marrow biopsy confirmed the diagnosis of MM. Overall, the evidence for the proposed precipitation reaction is limited, and the exact reason behind falsely elevated bilirubin levels remains dubious. The case mentioned above underscores the need for a visual examination of serum colour during the assay in discordant hyperbilirubinemia cases. It is also vital to remember the possible interference of different biochemical parameters with paraproteins to avoid diagnosis and subsequent management errors (Table 2).

Pre-Hepatic Causes

Pre-hepatic causes of jaundice in multiple myeloma are typically rare. One of the mechanisms is through autoimmune hemolytic anemia. Although AIHA is known to be associated with B-cell lymphoproliferative disorders [11], the association with MM is not well understood. Some studies propose that about 4% of MM patients [12] [13] will develop AIHA, while other studies have reported no association [14]. Kashyap et al. [15] observed AIHA in 7/66 patients with MM. These patients achieved remission of AIHA with the therapy of the underlying MM. It is suggested that MM being a B cell malignancy disturbs the immune system causing usually suppressed clones to produce antibodies against red blood cells (RBC) [15].

Table 2: Table describing pathophysiologic mechanisms of jaundice in Multiple Myeloma

Causes	Hypothesized Mechanisms	How to diagnose?	Relationship with multiple myeloma
False Hyperbilirubinemia	Precipitation reaction between paraprotein and solubilising agent	Serum showing precipitates on visual examination	Multiple myeloma is associated with increased paraproteins
Pre-hepatic Causes			
1. Autoimmune hemolytic anemia	Breakdown of RBC's leading to increased indirect/unconjugated bilirubin	Laboratory examination will show hemolytic anemia, positive antiglobulin test	Multiple myeloma disturbs the immune system leading to auto-immunity causing Normally suppressed immune cells to produce anti-RBC antibody
Hepatic Causes			
1.Amyloid deposits	1.Amyloid deposits in the liver distort the architecture/ parenchyma 2.Direct toxicity to cholangiocytes 3.Disruption of bile flow	Liver biopsy will show amyloid deposits which when stained with congo red and seen under polarised light will give apple green birefringence.	Multiple myeloma leads to increased production of monoclonal plasma cells secreting light chains which deposits in the various organs as amyloid.
2.Plasma cell infiltration	Plasma cell infiltrate the sinusoids in the liver damaging the liver parenchyma	Plasma cell infiltration seen on histology of the liver	Overproduction of malignant plasma cells in multiple myeloma which may spread by hematogenous route to the liver
3. Light Chain deposition disease (LCDD)	Alteration in the canalicular conjugate export pump	Light chains in the urinalysis	Myeloma is underlying condition in 50- 60% of patients with LCDD
4.Increased activity of Osteopontin (OPN)	A Pro-inflammatory cytokine leading to fibrosis	Osteopontin plasma levels	1. Multiple myeloma (MM) can increase the osteopontin levels which may lead to rapid progression of fibrosis. 2.OPN levels correlate with the severity of MM.
Post-Hepatic Causes			
1. Extramedullary plasmacytomas	Plasmacytomas around the bile ducts can lead to obstructive jaundice	CT scan will reveal the site of obstruction, follow up with a CT guided biopsy	Extramedullary plasmacytomas can happen anywhere in multiple myeloma
2. Amyloid deposition	Exclusive amyloid deposition in bile duct can lead to obstructive jaundice	Liver (?) biopsy	Multiple myeloma leads to increased production of monoclonal plasma cells secreting light chains which deposits in the various organs as amyloid.

Although the studies have shown mixed results, MM should be included in the differential for a patient presenting with AIHA in certain clinical contexts.

Hepatic Causes

Amyloid Deposition: Amyloid light chain (AL) amyloidosis is the most common type of systemic amyloidosis in developed countries, with 9 cases/million every year reported [16]. The clonal expansion of plasma cells in MM leads to significant monoclonal light chain immunoglobulin production, which deposits in various tissues via amyloid fibrils leading to organ damage. Approximately 30% of MM patients have subclinical amyloid deposits in multiple organs [17, 18]. Data regarding the specific pathophysiologic mechanism is limited. Proposed mechanisms include the possibility of the direct toxic effect of amyloid to cholangiocytes, as well as the deposits of amyloid fibrils disrupting bile flow through intrahepatic and extrahepatic ducts [19].

Yamamoto et al. [20] described a case of a 79-year-old Japanese female who presented with ascites, edema, and jaundice. Her initial workup revealed anemia, elevated total and direct bilirubin, and elevated liver enzymes in a cholestatic pattern with a negative viral hepatitis screen. Serum protein electrophoresis showed a monoclonal gammopathy. The patient died on the 16th day due to complications from hepatic failure. Liver biopsy performed post-mortem showed diffuse deposition of amyloid with the disappearance of liver cell cords. Elgouhari et al. [21] also described a case of a 59-year-old woman who presented with abdominal distension and weight loss and was found to have a diffusely enlarged liver and ascites on CT scan. Liver biopsy revealed amyloid deposition, distorting the liver architecture. Urine protein electrophoresis showed marked elevation of the light chains, and bone marrow aspirate revealed smoldering MM. The patient died two months later from multi-organ failure. Sadeghi et al. [22] also reported a case of MM who presented with liver dysfunction due to amyloidosis and improved with a standard chemotherapy regimen for high-risk MM. The first two cases highlight the fact that hepatic amyloidosis is associated with poor prognosis [23], suggesting that hepatic involvement with MM is a marker of advanced disease and is often related to poor outcomes if not caught early [23- 24]. The third case shows that initiation of therapy even in the setting of liver disease can lead to clinical improvement if the diagnosis is made promptly and treatment is started before further disease progression. Given the potential for worse outcomes in this population, additional data is needed to support specific chemotherapy regimens for MM and hepatic involvement.

Plasma Cell Infiltration: Plasma cells are white blood cells that originate in the bone marrow and secrete large quantities of proteins. Hepatic plasma cell infiltration has been observed in about 45 percent of MM patients during autopsy [4, 5]. However, only rare cases of jaundice have been reported in the literature, most of them presented as non-obstructive cholestasis with subsequent progression to liver failure. Overall, cases with reported plasma cell infiltration of the liver are associated with a high mortality rate. Arebi et al. [25] described a case of a 66- year-old Nigerian woman who presented with jaundice, pale stools, and dark urine. Laboratory tests showed elevation of ALT, ALP, and GGT. Viral hepatitis serologies and autoimmune workup were negative. CT scan showed three small attenuation lesions in the liver. Liver biopsy revealed plasma cells and plasmablasts in the sinusoids without any amyloid deposition. Serum electrophoresis confirmed the presence of a broad monoclonal band in the beta/gamma globulin region, and bone marrow biopsy confirmed plasma cells. This patient's liver function deteriorated during admission and she refused treatment for myeloma and died within a few days.

In regards to the pathophysiologic mechanism, it is proposed that plasma cells flood the hepatic sinusoids to varying degrees, which can damage liver parenchyma [26]. Although hepatic infiltration by plasma cells occurs more frequently in the setting of advanced stages of MM, it can also be the first presentation of this disease. Liver biopsy is useful and can lead to an expedited diagnosis.

Light Chain Deposition: Light Chain deposition disease (LCDD) characterized by the deposition of immunoglobulins' fragments in various organs, is seen in about 5% of MM cases [31]. LCDD most commonly involves the kidneys and presents as nephrotic syndrome and renal failure [27]. Hepatic deposition of light chains alone has been known to occur rarely [28]. Montialoux et al. [29] described a case of a 77-year-old man presenting with jaundice, pale stools, dark urine, and weight loss. Workup was remarkable for markedly elevated ALP and GGT (in the 2000s) with mildly elevated ALT and AST. Workup was negative for acute/chronic hepatitis.CT scan was unremarkable. A trans-parietal biopsy revealed PAS-positive cells without amyloid or plasma cells. A subsequent electrophoresis and BM aspiration revealed kappa chain myeloma. The deposition was hypothesized to be due to light chain deposits, which appeared as a granular amorphous material in perisinusoidal spaces. The mechanism of associated cholestatic jaundice can be explained by alteration of the canalicular pump leading to abnormal bilirubin. Although the pathophysiology is unclear, it has been postulated to alterations are consequences of an inflammatory response due to pro-inflammatory cytokines versus atrophy of hepatocytes adjacent to light chain deposits [29]. In cases of unexplained cholestasis and other clinical findings of MM, clinicians should keep LCDD as a differential.

Rapid Progression of NASH to Cirrhosis: Osteopontin is an extracellular pro-inflammatory cytokine involved in bone resorption, angiogenesis, and fibrosis in various tissues [30]. Osteopontin (OPN) levels are increased in patients with MM

[31]. The degree of increase in OPN is linked closely to the severity of MM [32]. There is also growing evidence that increased osteopontin is related to an increased fibrosis rate [39].

Syn et al. [33] in 2016 described that Nonalcoholic steatohepatitis (NASH) is associated with hedgehog activation, which induces osteopontin and promotes liver fibrosis. They studied liver sections from 11 patients with nonalcoholic fatty liver disease (NAFL), NASH, and NASH-cirrhosis. They analyzed that the expression of OPN was lowest in NAFL and highest in patients with NASH Cirrhosis, thus highlighting the relationship between osteopontin levels and liver fibrosis.

Akashi et al. [34] described a case of a 63-year-old female in whom the NASH progressed to cirrhosis due to rapid fibrosis within three years. Since the fibrosis progression was quick, they investigated for the presence of another condition. The liver biopsy was negative for amyloid disease or autoimmune disease. Immunoelectrophoretic study showed the absence of M protein, but an immunofixation analysis confirmed Bence Jones protein's presence. Bone marrow aspiration revealed plasma cells. They hypothesized that MM derived increase in OPN caused rapid progression of fibrosis in their patient. The measured osteopontin level was six-fold higher as compared to the average population.

This association between OPN and fibrosis could be a potential target for the treatment of NASH cirrhosis. More research in this field could yield promising solutions for better management of liver fibrosis.

Post-Hepatic Causes

Plasmacytomas: Pancreatic plasmacytoma is a common cause of obstructive jaundice in multiple myeloma. Williet et al. [35] conducted a systematic literature review identifying 63 case reports of pancreatic plasmacytoma. Jaundice was the most common presentation. The most common site of involvement was the head of the pancreas, with only 2 cases in the body and tail. Forty-one percent of these patients had MM. Whether jaundice was the initial presentation remains unclear

Utsumi et al. [36] reported a case of an 83-year-old male presenting with obstructive jaundice and CT scan, revealing a mass in the pancreatic head and retroperitoneum. CT guided biopsy of the retroperitoneum showed atypical plasma cells. Immunochemistry revealed plasmacytoma, which was followed by bone marrow biopsy showing MM. It is challenging to diagnose plasmacytoma before MM; however, it should be considered as a differential in patients presenting with obstructive jaundice, especially in the elderly. Other sites of plasmacytoma in MM include duodenum, gastrohepatic ligament, and gastroduodenal ligament.

Amyloid Deposition: Sasaki et al. [37] did an Autopsy study in patients with systemic amyloidosis. They hypothesized that amyloid deposition in the biliary duct correlates with amyloid deposition in the hepatic arteries concluding that biliary amyloid deposition is a part of hepatic amyloidosis. However, Terada et al. reported a patient presenting with jaundice found to have amyloid deposition exclusively in the biliary system on autopsy. This case is unique, and to the best of our knowledge, it is the only case reported with Jaundice in MM due to exclusive deposition in the bile duct [38]

Conclusion:

Jaundice is a rare presentation of MM. Lack of awareness of this presentation in the differential diagnosis of jaundice may lead to delays in diagnosis and treatment of underlying MM. Recent advances in treatment of MM make it possible to extend the duration and improve the quality of life of MM patients. It is crucial for the clinicians to be aware of this atypical presentation. Further research into the mechanisms of jaundice in MM is needed. We want to encourage physicians to continue to report these cases to add to the literature for better understanding of this rare association.

Conflict of Interest

We have no conflicts of interest to disclose.

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