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(CASE REPORT)

Cirrhosis Revealing Multivisceral Digestive Sarcoidosis: A Case Report

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Abstract

Sarcoidosis is a rare systemic granulomatous disease affecting various organs, predominantly the lungs and mediastinal lymph nodes. Hepatic involvement is common but most often asymptomatic, whereas gastrointestinal involvement remains exceptional. We report a rare case of digestive sarcoidosis involving the stomach, colon/rectum and liver with progression to cirrhosis.

Keywords: Hepatic Sarcoidosis; Gastrointestinal Sarcoidosis; Liver Cirrhosis

1. Introduction

Although sarcoidosis predominantly affects the lung and mediastinal lymph nodes, its capacity to disseminate to less expected organs — the liver, stomach, colon and rectum — may render the diagnosis misleading. Early recognition of digestive forms is essential because they can lead to severe complications, including progressive hepatic fibrosis. This case illustrates such an atypical clinical course and highlights the need for a multidisciplinary approach.

2. Case presentation

We report the case of a 52-year-old man with a three-year history of biopsy-proven mediastinal nodal pulmonary sarcoidosis who was referred for evaluation of diffuse abdominal pain, low-grade fever and a deterioration of general condition that had been evolving for several weeks. Clinical examination found a soft abdomen, a firm non-tender hepatomegaly and mild jaundice.

Laboratory tests revealed moderate hepatocellular injury, cholestasis, polyclonal hypergammaglobulinaemia and an elevated angiotensin-converting enzyme (ACE) level. Viral hepatitis serologies and autoimmune work-up were negative. Abdominal ultrasound and hepatic MRI demonstrated a dysmorphic liver with a nodular parenchymal appearance compatible with cirrhosis, without signs of portal hypertension.

Esophagogastroduodenoscopy showed a diffuse nodular infiltration of the gastric mucosa, while colonoscopy revealed extensive granulomatous lesions of the colon and rectum. Biopsies taken from all involved sites demonstrated non-caseating epithelioid cell granulomas with multinucleated giant cells. Hepatic histology showed portal and periportal granulomatous involvement with architectural remodeling consistent with cirrhosis.

In the absence of other infectious, autoimmune or drug-related causes, a diagnosis of digestive and hepatic sarcoidosis with evolution to cirrhosis was made. Systemic corticosteroid therapy was initiated, with partial improvement of the digestive symptoms.

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3. Discussion

After the lungs and lymph nodes, the liver is the third most commonly involved organ in systemic sarcoidosis. Most cases of hepatic involvement are asymptomatic; only 5–30% of patients present clinical symptoms ranging from nonspecific manifestations such as nausea and vomiting to jaundice, abdominal pain or hepatosplenomegaly [1]. Abnormal liver tests are observed in approximately 20–30% of patients, whereas portal hypertension and cirrhosis occur in fewer than 1% of cases [2]. Although portal hypertension is generally associated with cirrhosis, in patients with sarcoidosis it may occur in the absence of classical cirrhotic hepatic changes [3].

Non-caseating granulomas are the characteristic histopathological lesions of hepatic involvement. Hepatic biopsies frequently show epithelioid granulomas in periportal or sinusoidal locations, sometimes associated with a small lymphocytic cuff and foci of peri-granulomatous fibrosis. These histological changes are common even in the absence of clinical signs, which explains the frequent discordance between positive biopsy findings and a relatively quiescent clinical presentation [4].

Typical laboratory abnormalities are dominated by a cholestatic pattern (elevated alkaline phosphatase and gamma-glutamyl transferase), while transaminase elevations are generally less pronounced. The frequency and degree of hepatic biochemical disturbances vary across series but may reach 20–90% depending on the cohorts and the criteria used; the severity of biochemical abnormalities usually correlates with the extent of granulomatous involvement on histology [5].

More rarely, hepatic involvement progresses to significant fibrosis and portal hypertension. Proposed pathophysiological mechanisms include pre-sinusoidal obstruction by periportal granulomas, periportal and perisinusoidal fibrosis, granulomatous phlebitis, and vascular transformations such as nodular regenerative hyperplasia (NRH), which can produce portal hypertension in the absence of classical cirrhotic architecture. Accordingly, case series and reviews report that portal hypertension may develop without overt anatomic cirrhosis, through pre-sinusoidal block or compression of portal branches and/or granulomatous vascular involvement [6,7].

Symptomatic gastrointestinal involvement is uncommon but described. Gastric sarcoidosis may manifest with epigastric pain, nausea, vomiting or bleeding, whereas colonic involvement can present with diarrhea, rectal bleeding or iron-deficiency anemia. Endoscopic findings are variable (polypoid lesions, irregular ulcers or erythema), and diagnosis relies on demonstration of non-caseating granulomas on biopsy after exclusion of other granulomatous etiologies (infectious causes, inflammatory bowel disease, foreign-body reaction) [8,9].

From a diagnostic standpoint, evidence supporting hepatic/digestive sarcoidosis includes a suggestive systemic context (pulmonary or mediastinal involvement, extragastrointestinal signs), cholestatic biochemical abnormalities, compatible radiological images (hepatomegaly, hypodense or nodular lesions on MRI/CT), and, crucially, histological confirmation by liver and/or mucosal biopsy. It is essential to exclude differential diagnoses such as tuberculosis, other granulomatous diseases, primary biliary cholangitis, primary sclerosing cholangitis and drug-induced granulomatous reactions. Measurement of ACE may be contributory but is neither sensitive nor specific and does not replace pathological confirmation [10,4].

Management is primarily based on systemic corticosteroids for symptomatic forms, progressive cholestasis or complications. Ursodesoxycholic acid (UDCA) is commonly used when cholestasis predominates and may improve liver tests and cholestatic symptoms (pruritus) in some patients. For steroid-refractory or steroid-dependent disease, second-line immunosuppressive agents (methotrexate, azathioprine, mycophenolate) or biologic therapies have been employed; however, available evidence is largely limited to case series and reports and remains heterogeneous [11].

When portal hypertension is established, management follows standard principles (screening and treatment of esophageal varices, bleed prophylaxis with non-selective beta-blockers or endoscopic ligation as indicated, management of ascites and infectious complications). TIPS or liver transplantation have been reported in refractory cases, although outcomes are variable and the prognosis of symptomatic portal hypertension in sarcoidosis can be severe in some series, warranting early multidisciplinary care [12].

Prognostically, most patients with hepatic involvement remain stable or improve with therapy, but a minority progress to cirrhosis or develop complications of portal hypertension. Factors associated with adverse progression include persistent cholestasis, progressive fibrosis on biopsy and the presence of severe systemic manifestations. For these reasons, regular clinical, biochemical and imaging/endoscopic surveillance is recommended, and decisions to escalate therapy should be individualized [5,13].

4. Conclusion

In conclusion, this case highlights the rare but real potential for sarcoidosis to simultaneously involve the gastric, colonic, rectal and hepatic compartments with progression to clinically significant hepatic fibrosis. Diagnosis rests on the systematic search for non-caseating granulomas together with thorough exclusion of other granulomatous causes, and on comprehensive evaluation for systemic involvement. Management is primarily corticosteroid-based for symptomatic or cholestatic forms, with adjunctive use of agents such as ursodeoxycholic acid and immunosuppressants or biologics in cases of steroid dependence or resistance, and with management of portal hypertension complications according to standard practice if they occur.

Such presentations require prolonged multidisciplinary follow-up (hepatology, gastroenterology, pathology, radiology), focused on fibrosis progression, surveillance for complications (portal hypertension, liver failure, hepatocellular carcinoma) and adaptation of therapy, as a small proportion of patients may evolve to severe liver disease requiring interventional strategies or even transplantation.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed

Statement of ethical approval

The present research work does not contain any studies performed on animals/humans subjects by any of the authors.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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