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ALECT2 Amyloidosis of the Spleen With Concurrent Pancreatic Amyloid Deposition Incidentally Identified in a Patient With Symptomatic Mucinous Cystadenoma

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Abstract

ALECT2 amyloidosis is a systemic form of amyloid deposition that most often involves the kidneys and liver and is frequently identified incidentally on surgical pathology. Splenic involvement is relatively common, whereas pancreatic deposition is rare, and both are typically asymptomatic. A woman in her early 60s presented with right flank and left upper abdominal pain and was found to have a 7-cm cystic lesion in the pancreatic tail. She underwent laparoscopic distal pancreatectomy and splenectomy, and pathology revealed a multiloculated mucinous cystadenoma involving both organs. Congo red staining demonstrated amyloid deposition in the pancreas and spleen. Mass spectrometry confirmed ALECT2 amyloid in the spleen. Postoperative evaluation showed no evidence of systemic involvement. The patient recovered well following surgery. This case highlights that ALECT2 amyloid identified in the spleen or pancreas is usually incidental and that accurate amyloid typing is essential to guide management and avoid unnecessary therapy.

Categories: Oncology, Gastroenterology

Keywords: alect2 amyloidosis, amyloid typing, leukocyte chemotactic factor 2 amyloidosis, mass spectrometry, mucinous cystadenoma, pancreatic amyloidosis

Introduction

ALECT2 amyloidosis, also known as leukocyte chemotactic factor 2 amyloidosis, was first described in 2008 when LECT2 was identified as a novel amyloidogenic protein responsible for systemic amyloid deposition [1]. Since its initial recognition, ALECT2 has been increasingly reported and is now known to be a significant cause of renal and hepatic amyloidosis especially among Hispanic communities [2]. Extrarenal involvement also occurs, with the spleen among the more frequently affected secondary organs. Despite this, splenic ALECT2 deposits are typically asymptomatic and identified incidentally. In one dedicated study of splenic amyloidosis, ALECT2 accounted for 43.5% of cases, underscoring its prominence among splenic amyloid subtypes [3]. Pancreatic involvement is far less common and has been documented only rarely in retrospective studies, with no established association with pancreatic dysfunction [4]. Amyloid deposition can be identified using histologic staining, while definitive identification of the amyloid precursor protein is most reliably performed using mass spectrometry [5]. Accurate subtype identification is essential, as treatment strategies differ substantially from those used in immunoglobulin light-chain (AL) amyloidosis, and misclassification can lead to unnecessary chemotherapy [4,6]. In this report, we describe incidental splenic ALECT2 amyloidosis, confirmed by mass spectrometry, with concurrent pancreatic amyloid deposition of undetermined subtype in a patient undergoing surgery for a symptomatic mucinous cystadenoma. This unusual coexistence highlights the importance of accurate amyloid typing to distinguish ALECT2 from AL amyloidosis and prevent unnecessary chemotherapy.

Case Presentation

A Hispanic woman in her early 60s with a history of obesity, type 2 diabetes mellitus, and hypertension presented to the emergency department with a two-day history of sharp right flank pain radiating to the left upper quadrant. A computed tomography (CT) scan of the abdomen and pelvis with contrast demonstrated a 7-cm cystic lesion along the tail of the pancreas. Surgical Oncology was consulted based on these findings.

A multiphase CT of the pancreas with and without contrast showed a 7.3-cm thin-walled cystic lesion at the pancreatic tail with minimal enhancement of thin-walled septations on delayed post-contrast imaging. These findings were nonspecific but raised concern for a pancreatic cyst or cystic neoplasm. The patient later followed up with Surgical Oncology as an outpatient and underwent a laparoscopic distal pancreatectomy and splenectomy due to splenic hilar involvement of the mass noted intraoperatively.

Final pathology described a multiloculated mucinous cystadenoma involving the spleen and pancreas. Histopathologic examination demonstrated amorphous eosinophilic deposits within the pancreatic islets

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(Figure 1), with thioflavin T fluorescence confirming pancreatic amyloid deposition (Figure 2). Similar amorphous eosinophilic deposits were identified within the splenic red pulp (Figure 3), with corresponding thioflavin T fluorescence confirming splenic amyloid deposition (Figure 4). Focal pancreatic ductal findings were also described as consistent with intraductal papillary mucinous neoplasm (IPMN). Liquid chromatography-tandem mass spectrometry of splenic tissue confirmed ALECT2 amyloid deposition. There was insufficient remaining pancreatic tissue to complete mass spectrometry on the pancreas.

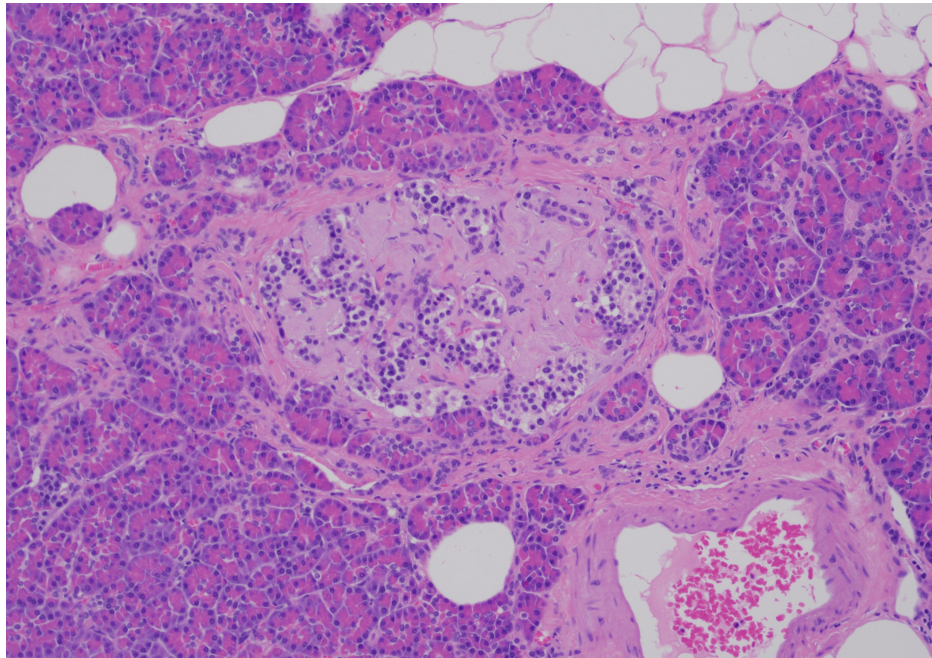


FIGURE 1: Pancreatic amyloid deposition on H&E staining

Pancreatic islet showing amorphous eosinophilic deposition at $\times 100$ magnification on H&E staining.

H&E: hematoxylin and eosin

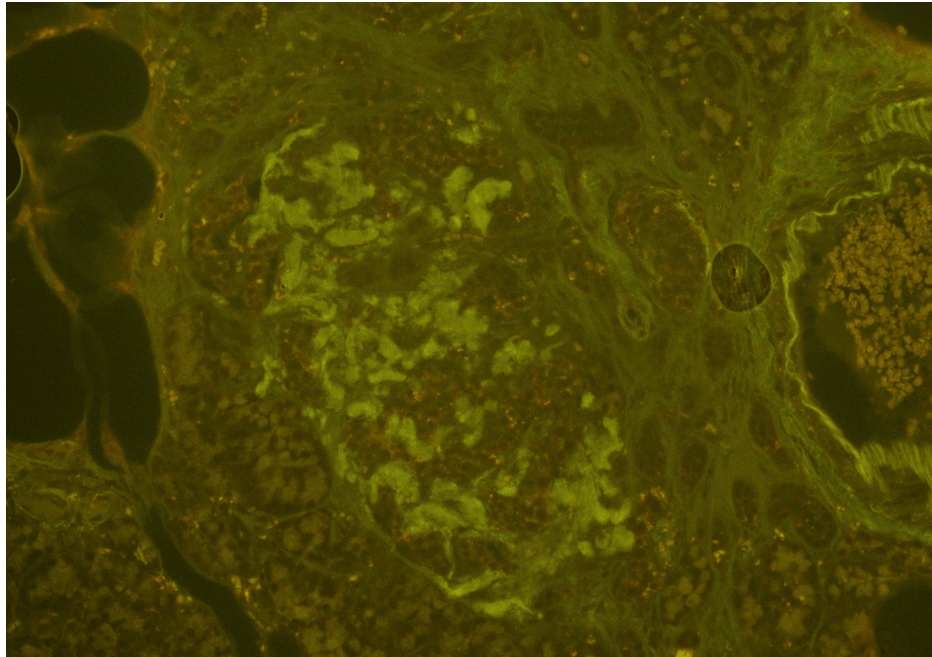


FIGURE 2: Pancreatic amyloid deposition on thioflavin T staining

Pancreatic islet showing thioflavin T fluorescence under ultraviolet light, confirming amyloid deposition at $\times 100$ magnification.

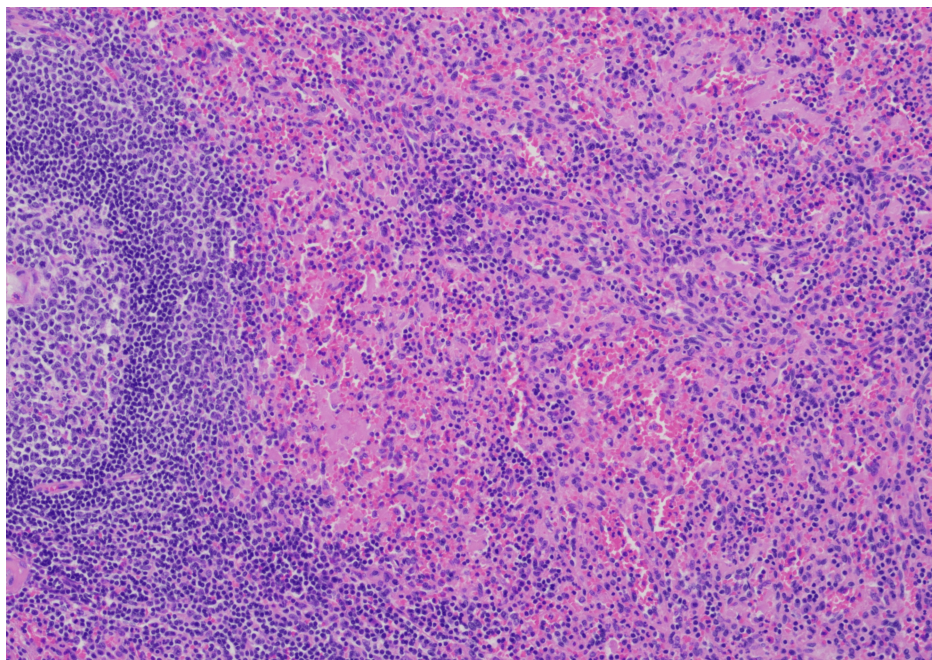


FIGURE 3: Splenic amyloid deposition on H&E staining

Splenic tissue showing amorphous eosinophilic deposition within the red pulp with relative sparing of the white pulp at $\times 100$ magnification on H&E staining.

H&E: hematoxylin and eosin

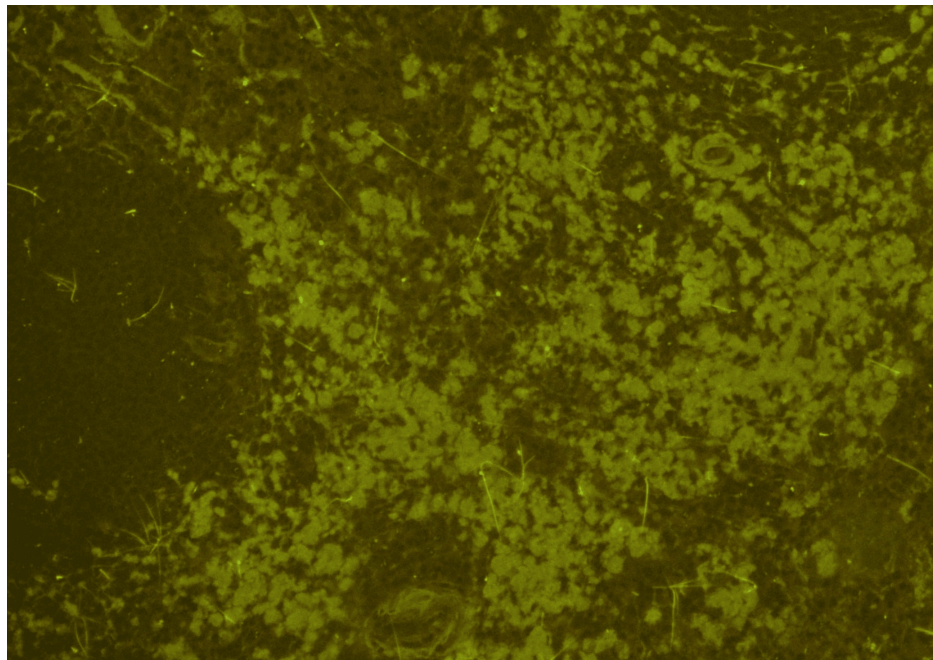


FIGURE 4: Splenic amyloid deposition on thioflavin T staining

Splenic tissue showing thioflavin T fluorescence under ultraviolet light, confirming amyloid deposition at $\times 100$ magnification.

Postoperative evaluation included magnetic resonance imaging (MRI) of the abdomen and transthoracic echocardiography, both of which were unremarkable. Serum protein electrophoresis, urine protein electrophoresis, serum immunofixation, and urine immunofixation were all negative for monoclonal protein. A positron emission tomography (PET) scan showed no lytic bone lesions. Bone marrow biopsy demonstrated normocellular marrow with trilineage hematopoiesis, approximately 5% polytypic plasma cells, and no amyloid deposition.

At several outpatient follow-up visits, the patient reported doing well without recurrent abdominal symptoms. Ongoing monitoring of liver and kidney function was recommended.

Discussion

Amyloidosis refers to a heterogeneous group of disorders characterized by the deposition of misfolded protein fibrils within tissues. The clinical manifestations depend on the specific amyloid precursor protein and the organs involved [7]. AL amyloidosis, derived from immunoglobulin light chains, is typically aggressive and often leads to significant renal, cardiac, or neurologic dysfunction [8]. Serum amyloid A (AA) amyloidosis, associated with chronic inflammatory conditions, frequently results in progressive renal impairment [9]. Transthyretin (ATTR) amyloidosis, caused by misfolded transthyretin, commonly produces cardiomyopathy or neuropathy and may be associated with substantial morbidity [10]. In contrast, ALECT2 amyloidosis represents a more indolent and recently recognized form of systemic amyloid deposition derived from leukocyte chemotactic factor 2 [1]. It is most often identified incidentally, is typically clinically silent, and when symptomatic may manifest only as slowly progressive renal impairment [11,12].

Although some amyloid subtypes, such as ATTR amyloidosis, are hereditary, the genetic basis of ALECT2 amyloidosis is less clearly defined. Sequencing studies have identified a shared non-synonymous polymorphism in the LECT2 gene (rs31517) among affected patients, suggesting a genetic susceptibility that may increase the tendency of the protein to misfold and deposit but is not sufficient on its own to cause disease. This supports a multifactorial pathogenesis rather than a purely inherited mechanism [13].

Additionally, while ALECT2 most frequently involves the kidneys and liver, population-based studies demonstrate notable variation in distribution by ancestry [2,13]. Early cohorts identified a strong association with Hispanic ethnicity, with 88-92% of cases occurring in individuals of Hispanic background [11,12]. A large 2023 autopsy study from Mexico further highlighted this pattern, identifying ALECT2 in over half of all amyloidosis cases and demonstrating deposits across several organs, including the kidneys, liver, spleen, and adrenal glands [14].

These findings highlight that extrarenal involvement is not uncommon and the spleen appears to be one of

the more frequently affected secondary sites [3]. In a study of splenic amyloidosis, ALECT2 accounted for 43.5% of cases, a frequency comparable to AL amyloidosis, yet splenic involvement is almost always asymptomatic [3]. Pancreatic involvement, however, is significantly less common and has been reported only rarely in the literature [4]. To date, published evidence suggests that pancreatic ALECT2 does not produce characteristic clinical symptoms and is generally found only when tissue is examined for other indications.

In our patient, this pattern was evident. The presenting abdominal pain was ultimately attributable to a multiloculated mucinous cystadenoma requiring surgical resection. Thioflavin T staining demonstrated amyloid deposition in both the spleen and pancreas. Although the pancreatic amyloid subtype could not be confirmed because insufficient tissue remained for mass spectrometry, the concurrent splenic ALECT2 deposition raises the possibility of the same subtype in the pancreas. Consistent with prior literature, the amyloid deposits were incidental and were not considered responsible for the patient's presenting symptoms.

Accurate amyloid typing is essential in such cases because treatment varies substantially by subtype [5]. Mass spectrometry allows the identification of the amyloid precursor protein and, in this case, confirmed ALECT2 in the splenic tissue [5]. This distinction is imperative because treatment differs markedly by subtype and misdiagnosis as AL amyloidosis could lead to unnecessary plasma cell-directed chemotherapy and potentially harmful interventions. On the other hand, ALECT2 amyloidosis is typically managed with supportive care and lacks disease-specific therapy [4,6]. Long-term management of ALECT2 centers on monitoring renal function, and prognosis is generally favorable in the absence of significant renal involvement [4,11].

In patients with ALECT2 amyloidosis who develop progressive renal failure, renal transplantation may be considered, as outcomes are generally favorable in the absence of systemic involvement [4]. Cardiac involvement is uncommon in ALECT2 amyloidosis, which further distinguishes it from other amyloid subtypes and contributes to its relatively indolent clinical course [4].

Conclusions

This case highlights incidental mass spectrometry-confirmed splenic ALECT2 amyloidosis with concurrent pancreatic amyloid deposition of undetermined subtype in the setting of a symptomatic mucinous cystadenoma. While splenic involvement in ALECT2 amyloidosis is recognized relatively frequently, pancreatic involvement remains uncommon and is usually clinically silent. Accurate identification of the amyloid subtype using mass spectrometry is essential, as distinguishing ALECT2 from other amyloid subtypes, particularly AL amyloidosis, has substantial implications for prognosis and management.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Moza A. Hamoud, Andrew Hwang, Charity Huang

Acquisition, analysis, or interpretation of data: Moza A. Hamoud, Andrew Hwang, Charity Huang

Drafting of the manuscript: Moza A. Hamoud

Critical review of the manuscript for important intellectual content: Moza A. Hamoud, Andrew Hwang, Charity Huang

Supervision: Charity Huang

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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