



Review Article

Gastrointestinal Myeloid Sarcoma a Case Presentation and Review of the Literature

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Abstract. Myeloid sarcomas can be detected in up to 30% of acute myeloid leukemia cases or occur de-novo without bone marrow involvement. The most frequent localization of myeloid sarcomas in the abdominal cavity is the small intestine, and gastric presentations are infrequent, frequently misdiagnosed, and a high level of suspicion should exist when the characteristic histomorphology features are present. The current review features a case report with gastric presentation of myeloid sarcoma in a patient with a diagnosis of acute myeloid leukemia with trisomy 8. In addition, a review of the literature of intestinal-type myeloid sarcomas shows that less than 15% of these cases have been reported in the stomach. The most common molecular aberrancy detected in intestinal myeloid sarcomas is the fusion protein CFBF-MYH11. A review of several large studies demonstrates that the presence of myeloid sarcoma does not constitute an independent prognostic factor. The therapeutic approach will be tailored to the specific genetic abnormalities present, and systemic chemotherapy with hematopoietic stem cell transplant is the most efficient strategy.

Keywords: Intestinal; Gastric; Myeloid sarcoma; Trisomy 8; Acute leukemia.

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Case Presentation. A 60-year-old male with a relevant past medical history of hepatitis C infection, methadone use, and heroin addiction was surgically managed for a toe infection. Persistent leukocytosis was noted despite antibiotic treatment, and a microscopic review of the peripheral blood smear demonstrated circulating blasts with left shift in granulocytes. Bone marrow biopsy evaluation demonstrated 70-80% myeloblasts, and the concurrent flow cytometric analysis show myeloblasts with the expression of CD34, CD117, CD33, CD11c, CD13, and negative for CD56. A diagnosis of acute myeloid leukemia was rendered. Karyotype analysis of the bone marrow aspirate demonstrated trisomy 8 (47, XY, +8[2]/46, XY[18]). Molecular testing demonstrated mutations in SRSF2, TET2, and KRAS genes (SRSF2 P95H, TET2 F1901Lfs*4, TET2 S585*, KRAS A146T).

During admission, the patient complained of persistent abdominal pain, with coffee-ground emesis and one episode of melena. Computed tomography showed thickening of the gastric wall near the gastroesophageal junction with multiple enlarged lymph nodes. An urgent esophagogastroduodenoscopy revealed a large, firm, ulcerated, and partially circumferential mass in the cardia extending into the fundus, with multiple ulcers within the gastric fundus and proximal duodenum. Histologic evaluation from biopsies of the cardia mass demonstrated dense submucosal aggregates, composed of large cells, with lobated nuclear contours and fine and dispersed chromatin with inconspicuous nucleoli (**Figure 1**). Immunohistochemical stains demonstrated that the infiltrates were positive for MPO, CD43, with weak CD68 expression, and negative for CD34, CD117,

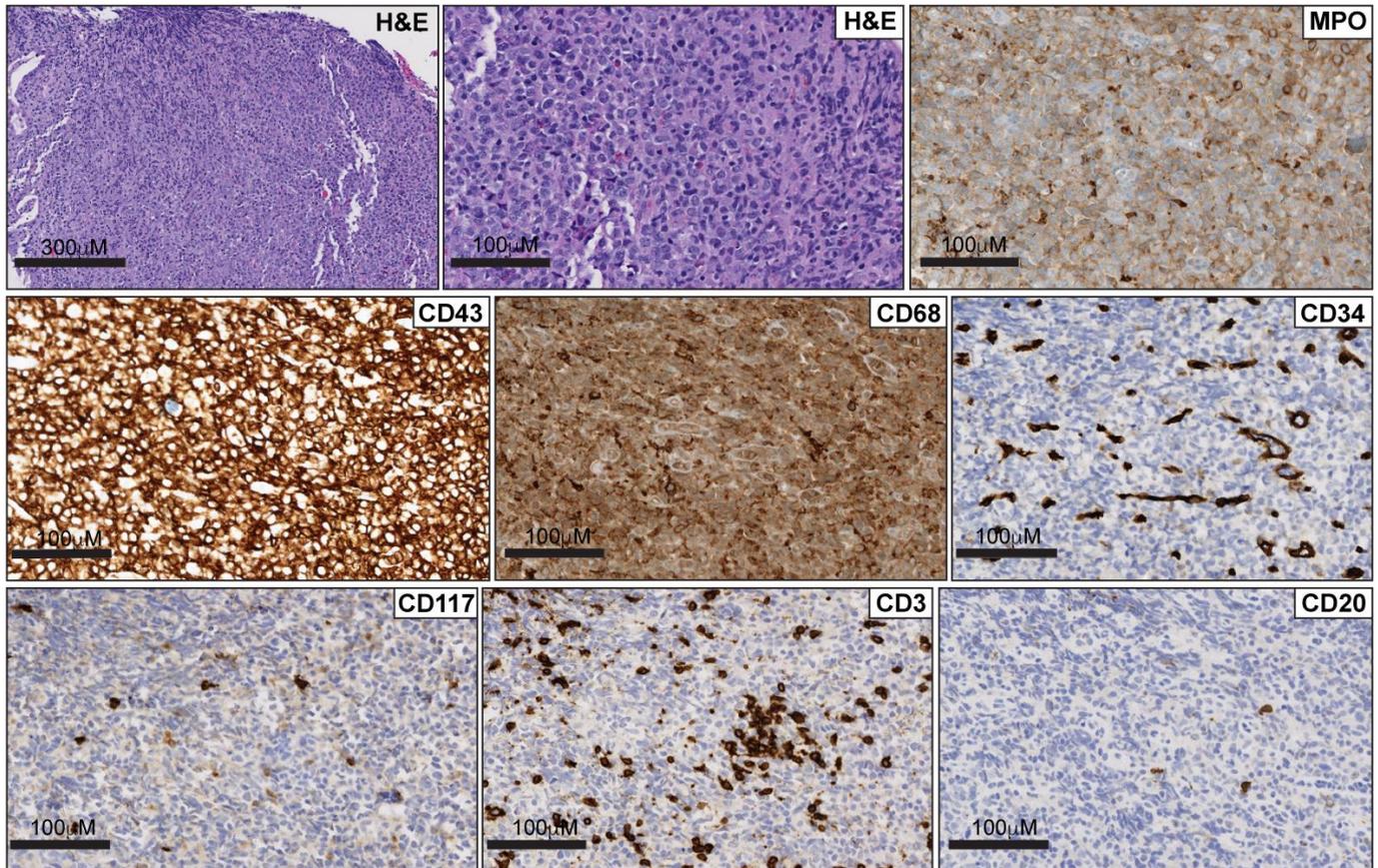


Figure 1. Representative pictures of gastric cardia mass. Each immunostain or tissue stain is indicated within each picture. **Abbreviations:** Hematoxylin and Eosin stain (H&E). Myeloperoxidase immunostain (MPO).

CD20, CD79a, CD5, CD3 and cyclin D-1 (**Figure 1**). A diagnosis of myeloid sarcomas was established. The following questions arise from this case: *How often is myeloid sarcoma identified in the stomach, and what are the specific histological features that help establish the diagnosis? Do intestinal myeloid sarcomas share common molecular features? What is the prognosis and management of myeloid sarcomas?*

Definition and Frequency of Myeloid Sarcoma.

Myeloid sarcoma (MS) is defined by the presence of neoplastic extramedullary infiltrates of myeloid precursors at a single or multiple sites, and the term leukemia cutis is reserved for when those infiltrates are present in the skin.¹ The incidence of myeloid sarcoma is variable, it has been reported in 7% to 30% of the cases of acute myeloid leukemia,² and this variability will largely depend on the criteria utilized to define extramedullary involvement. The majority of the studies that identified myeloid sarcoma by physical examination and/or imaging studies demonstrated a prevalence in the range of 20% to 30%, in contrast to studies with the only biopsy-proven diagnosis that identified a prevalence of 7-10%.¹⁻⁴ Position-electron tomography (PET) has also been utilized, with reported increased sensitivity compared to physical exam or other imaging techniques.^{5,6} A single prospective study that compared PET-imaging, clinical examination, and histologic

analysis, reported an incidence of 22% with a sensitivity of 77% and specificity of 97% for FDG-PET.⁵

The more frequent localizations of myeloid sarcoma will also depend on the methodology utilized for its detection. Detection by physical examination identifies, as the most frequent sites, the gingiva, spleen, and lymph nodes.² When imaging or biopsy studies are utilized during the diagnosis, the most frequently involved sites include the lymph nodes, testis, bone, and soft tissue.^{1,7} Some studies do not consider gingival, lymph node, liver, or splenic involvement as myeloid sarcoma, as the hypertrophy identified during physical examination may constitute migration/extravasation of blast precursors and not a truly ‘tumor’ lesion.^{5,8,9}

Myeloid sarcoma can occur de-novo without simultaneous bone marrow involvement, and in these instances, the diagnosis may be challenging, as it can be misdiagnosed as lymphoma.^{10,11} Moreover, a diagnosis of myeloid sarcoma can precede the occurrence of acute myeloid leukemia, or the occurrence of myeloid sarcoma can be the sole manifestation of relapse. No specific risk factors for myeloid sarcoma are identified; however, surface expression of CD56,¹² and CD11b,² and monocytic differentiation⁴ are reported to be associated with an increased risk for the presence of extramedullary involvement by leukemia.

Intestinal Myeloid Sarcoma. Involvement of the

gastrointestinal tract by myeloid sarcoma is not a common occurrence, and the most involved site is the small intestine. A PubMed search using a combination of the terms 'intestinal' 'gastric' 'myeloid sarcoma' and 'granulocytic sarcoma' identified 58 individual clinical case reports of myeloid sarcoma localized in the intestinal tract. From the list, only 7 cases were reported to be localized in the gastric compartment (**Table 1**). The clinical presentation for gastric myeloid sarcoma was characterized by non-specific symptoms, including persistent vague abdominal pain and vomiting. Upper gastrointestinal endoscopy usually revealed thickening of the stomach mucosa with hyperemic lesions and occasional nodularity or tumor-forming lesions. Imaging studies, including computer tomography (CT) scans, often revealed enlarged lymph nodes around the stomach. In the current literature review series, 60% of the cases (n = 50) did not feature synchronous bone marrow involvement by acute leukemia, making the diagnosis more challenging. Importantly, five reported cases featured relapse of acute myeloid leukemia in the form of gastrointestinal myeloid sarcoma, and three of those cases were post stem cell transplantation.

Table 1. Identified reported cases of myeloid sarcoma within the intestinal wall.

Site	Case	BM	LN
	number	involvement	involvement
Stomach	10/61 (16%)	4/7 (57%)	3/7 (43%)
Small Intestine	39/61 (63%)	10/38 (26%)	9/39 (23%)
Colon	4/61 (7%)	3/3 (100%)	2/4 (50%)
Appendix	4/61 (7%)	3/3 (100%)	NR

Abbreviations: Bone Marrow (BM), Lymph node (LN). Cases in the stomach, references: (43-51). Cases in the small intestine, references: (16,17,23,24,29-32,40,52-76). Cases in the colon, references: (26,77-79). Cases in the appendix, references: (33,39,65,80).

Histologic Evaluation and Differential Diagnosis of Intestinal Myeloid Sarcomas. Myeloid sarcomas are histologically characterized by dense submucosal infiltrates, predominantly composed of large cells with cleaved or slightly folded nuclear contours, variable amounts of cytoplasm, and finely dispersed chromatin.¹¹ However, similar features can be identified in lymphoid and non-lymphoid neoplasms in the intestinal tract; so, the differential diagnosis includes mature lymphoproliferative neoplasms like diffuse large B-cell lymphoma, the blastoid variant of mantle cell lymphoma, and mature T-cell neoplasms with secondary GI tract involvement or primary to the GI tract. Non-hematolymphoid neoplasms that may show similar morphological features includes poorly differentiated carcinoma and melanoma. The possibility of benign extramedullary hematopoiesis (myeloid metaplasia) should also be included in the differential diagnosis; however, those are characterized by multilineage

hematopoietic elements at progressive stages of maturation.

Myeloid sarcoma localized within the intestinal tract is not a frequent presentation and lacks specific clinical symptoms; therefore, the diagnosis may remain elusive unless clinical suspicion and a careful histological analysis exist. A misdiagnosis of myeloid sarcoma is not uncommon,^{11,13} one report indicated that 58% (n = 26) of cases were originally diagnosed as lymphoma,¹⁴ and two independent reports demonstrated that 44% (n = 61) and 46% (n = 158) were initially mislabeled as lymphoma.^{11,15} Therefore, immunohistochemical stains are required to establish a diagnosis, and a panel that includes myeloid-specific markers (MPO, CD117), should be included with any suspicious infiltrate. The current literature review demonstrates that the majority of the cases within the intestinal tract featured positive expression of either CD34 (91%, n = 36) or CD117 (93%, n = 29), and only a single report was identified with negative expression of both of these markers (**Table 1**).¹⁶ Importantly, expression of either CD43 (94%, n = 17) and MPO (97%, n = 32) is frequently detected in the tumor cells (**Table 1**). Aberrant expression of associated lymphoid markers has been reported in cases of myeloid sarcoma, including CD3 and CD79a. The current review for the intestinal presentation of myeloid sarcoma shows that CD3 was positive in a single case,¹⁷ and expression of CD5, CD20, and CD79a was not detected in any of the cases included. The current case report also demonstrated divergent CD34 and CD117 between the bone marrow biopsy and the stomach biopsies, a phenomenon previously described in leukemia cutis.¹⁸

Recurrent Cytogenetic Abnormalities in Intestinal Myeloid Sarcomas.

The previous series indicated that trisomy 8 is commonly detected in cases of myeloid sarcoma¹⁹ and leukemia cutis,²⁰ and the presence of chromosome 8 abnormalities are associated with worse responses to initial therapies and worse overall clinical outcomes.^{21,22} Unlike the current case, the literature review did not identify any previous report of abdominal myeloid sarcoma with trisomy 8. Previous reports demonstrated a higher prevalence for abdominal localization of myeloid sarcomas that are positive for the CFBF-MYH11 fusion.²³ The largest series to date included 13 cases of myeloid sarcoma with an abdominal presentation, and 11 (85%) of those cases were positive for CFBF-MYH11 fusion protein.²³ The same report included an additional number of 22 cases that were identified from reported cases in the literature, and 20 (92%) of those cases were positive for CFBF-MYH11 fusion protein.²³ The current review of the literature identified 11 additional cases of intestinal myeloid sarcoma^{17,24-33} that evaluated for genetic abnormalities by either cytogenetic or molecular testing, and the most common chromosomal rearrangement was inv(16) in

Table 2. Identified reported cases with available molecular studies (not included in Dalland et al. reference # 12). References for the studies included: (13-23).

Positive stains (positive / total tested)	References
CD34 (4/5), CD117 (5/5), CD43 (3/3), CD68 (2/2), MPO(4/4)	43-51
CD34 (27/29), CD117 (20/22), CD43 (12/13), CD68 (11/12), MPO(26/27)	16,17,23,24,29- 32,40, 52-76
CD34 (1/1), CD117 (1/1), CD43 (nt), CD68 (1/1), MPO(nt)	26,77-79
CD34 (1/1), CD117 (1/1), CD43 (1/1), CD68 (1/1), MPO(1/1)	33,39,65,80

27% (3/11) of the cases (**Table 2**).

Prognosis of Myeloid Sarcoma. Poor outcomes with negative trends in the overall survival and event-free survival sarcoma have been associated with a diagnosis of myeloid sarcoma.^{7,22} However, when adjusted to age and cytogenetic risk, myeloid sarcoma is not identified as an independent prognostic factor for overall survival,^{2,4,34} and the overall 5-year survival rate for patients with concomitant myeloid sarcoma is no different from those without it.¹ A retrospective study that evaluated the clinical outcomes of 84 patients with acute myeloid leukemia (AML) patients positive for t(8;21)(AML1/ETO) included eight patients with extramedullary involvement with significant worst responses to initial induction chemotherapy. However, half of the patients with extramedullary involvement featured CNS leukemic infiltration,²¹ and additional studies did not identify myeloid sarcoma as an adverse prognostic factor in this subset of cases.⁴

Management of Myeloid Sarcoma. The treatment of myeloid sarcoma will depend on the form of presentation and whether this occurs at the initial diagnosis or at relapse.¹ In the current era of targeted therapies, the consensus is to tailor the therapeutic approach based on the tumor's genetic profile, and therefore molecular and cytogenetic testing are required to formulate the treatment strategies.³⁵

As a rare condition, the lack of randomized controlled trials limits the treatment strategies for isolated myeloid sarcoma without bone marrow involvement. Retrospective series have demonstrated that delayed or localized therapy alone will almost always progress to AML.^{1,35} Remission-type induction chemotherapy regimens have also been used in isolated myeloid sarcoma, demonstrating a decrease in the rate of progression and an increase in overall survival.^{35,36} The role of hematopoietic stem cell transplantation (HSCT) following induction therapy is supported by retrospective studies, which indicate that allogeneic HSCT is associated with a 5-year overall survival rate of 48% and leukemia-free survival of 36%.³⁷ Localized radiation therapy (RT) has also been considered as

consolidation treatment for isolated MS.³⁸

Systemic treatment is the first-line option for myeloid sarcoma with concurrent marrow involvement at diagnosis. First-line treatments for gastrointestinal myeloid sarcoma usually include conventional induction chemotherapy with a combination of cytarabine and anthracycline agents.^{39,40} Unfortunately, there have been no randomized trials comparing the different types of chemotherapy regimens and assessing the optimal AML remission-induction chemotherapy regimens in the setting of myeloid sarcoma with marrow involvement. However, autologous and allogeneic HSCT are usually considered as treatment intensification based on the superior outcomes with the use of HSCT in several retrospective studies.^{37,38}

Isolated myeloid sarcoma at relapse is rare and usually precedes marrow relapse. For relapse after chemotherapy alone, strategies such as relapse-type chemotherapy regimens and localized radiation therapy (RT) are recommended.¹ Isolated myeloid sarcoma after HSCT can present as the first manifestation of relapse but has rarely been described, and currently, there is no established standardized management. The therapeutic strategies include donor lymphocyte infusion, tapering of immunosuppression, or enrollment in clinical trials.⁴¹ Concomitant MS and marrow relapse are usually treated with reinduction chemotherapy taking into account the possibility of HSCT or RT.³⁸ In the setting of marrow and myeloid sarcoma relapse after HSCT, the survival is poor and investigational agents or palliative care measures must be considered.¹

Finally, noncontrolled anecdotal reports have described the use of highly targeted therapies as a treatment strategy for myeloid sarcoma. For example, the humanized anti-CD33 monoclonal antibody has been associated with good responses.^{35,36} In addition, tyrosine kinase inhibitors have also shown promising results in a patient with myeloid sarcoma associated with FIP1L1-PDGFR α fusion gene and eosinophilia.⁴²

Conclusions. Myeloid sarcoma can occur in up to 30% of newly diagnosed acute myeloid leukemia. According to the site presentation, it is usually identified by physical examination and imaging studies, and a confirmatory biopsy will be required in a proportion of the cases. Histology analysis shows overlapping morphology with mature lymphoproliferative neoplasms, and immunohistochemical analysis is required to establish the diagnosis. The most common site within the intestinal tract is the small intestine, and the expression of CD34, MPO, and CD117 are the most sensitive and specific markers. The aberrant fusion protein CBF β -MYH11 is the most predominant cytogenetic abnormality detected in intestinal myeloid sarcoma. A diagnosis of myeloid sarcoma does not appear to render a worse prognosis in comparison with patients that do not

feature extramedullary disease. The clinical management of myeloid sarcoma will largely depend on the timing of

the presentation and the underlying genetic landscape of the tumor.

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