

P-126. UNUSUAL CECAL PRESENTATION OF BLASTOID MANTLE CELL LYMPHOMA INVOLVING PERIPHERAL BLOOD AND CREATING A CONFUSING CONDITION OF ACUTE LEUKEMIA: A CASE REPORT

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INTRODUCTION

Blastoid mantle cell lymphoma B-MCL is an aggressive rare variant of MCL characterized by medium to large-sized lymphoid cells with high mitotic and proliferative index. It can arise de novo or as a transformation from classical MCL. It is challenging to diagnose based on morphology features alone as it mimics other neoplasms. Hence, immunophenotyping and molecular studies are of great importance. B-MCL as an isolated cecal mass is a diagnostic challenge given that it is a fairly rare presentation.

Herein, we describe an interesting case of B-MCL presenting as an isolated cecal mass in an elderly patient with history of chronic renal impairment and hypertension.

CASE PRESENTATION

We describe a case of a 76-year-old Algerian Arab man who presented to emergency department with acute-onset diffuse abdominal pain and melena. The pain was intermittently radiating to the suprapubic area. On examination, the abdomen was soft and diffusely tender on light palpation but without peritoneal signs. The patient appeared in mild distress due to pain. No recent history of nausea, vomiting, diarrhea or recent weight loss.

An extensive combining of laboratory, imaging and histopathology work-up was planned in order to explain his acute progressive pain.

RESULTS AND DISCUSSION

The diagnosis of MCL involves a comprehensive evaluation with laboratory tests, imaging studies, genetic and molecular markers, and tissue biopsies.

A complete CBC showed pancytopenia with hyperleukocytosis (Table1) made of abnormal lymphoid cell (Blasts flags)

A PBS showed a marked lymphocytosis with medium to large cells, many with prominent and multiple nucleoli. Some nuclei had prominent clumps. The chromatin pattern was quite delicate (Fig.1), leading to predict an acute leukemic process. However, the BM aspirate was normal.

Biochemistry: results were consistent with a kidney function decline. Elevated levels of LDH and uric acid are commonly associated with increased tumor burden and a poorer prognosis.

Flow cytometry analysis in PB specimen: a mature clonal (kappa bright restricted) CD5+ B-cell lymphoproliferative disorder was identified. The cells co-expressed CD20bright, CD38, FMC7, CD79b and HLA-DR. CD34, CD117 and CD23 were negative. An aberrant expression of CD10 was also detected.

Rectal examination revealed melanic stools without other specific abnormalities.

Imaging: A CT scan of the chest, abdomen and pelvis showed widespread small volume (<2 cm) lymphadenopathy in abdomen and pelvis. The spleen was also enlarged.

An upper endoscopy with gastric biopsies showed focal atypical lymphoid aggregates, consistent with minimal involvement of atypical lymphoid infiltrates and chronic gastritis.

Colonoscopy revealed nodular mucosa images in the ascending colon and the cecum with an ulcerated and partially obstructing large mass in the cecum (Fig2).

Histopathology of the cecal biopsy and immunostaining: revealed MCL with a blastoid morphology and a high proliferation index of 64%, a Cyclin D1 overexpression, resulting from the t(11;14) which is a hallmark confirming and distinguishing MCL from other lymphomas.

ISH analysis was positive for CCND1/IGH (t(11;14)) gene rearrangement, supporting the MCL diagnosis.

CONCLUSION

Mantle cell lymphoma of the cecum is a rare and aggressive malignancy that often presents with nonspecific GI symptoms including abdominal pain, altered bowel habits, and GI bleeding. Diagnosis relies on endoscopic evaluation, histopathological analysis, and molecular studies, with hallmark findings, such as cyclin D1 overexpression and t(11;14) translocation.

Despite advancements in immunotherapy and targeted therapies, such as BTK inhibitors and CAR-T cells, therapeutic results remain poor, particularly for aggressive blastoid variants.

After successfully removing the obstructive cecal mass, the patient received 4 cycles of palliative chemotherapy with a BR regimen and was transitioned to comfort care because of worsening clinical status. Unfortunately, he died due to septic shock from CDI and that illustrates how disease aggressiveness and treatment toxicity can combine to produce fatal infectious complications.

This case underscores the need for early recognition, prompt intervention, and a multidisciplinary approach to improve outcomes in patients with such unusual presentation of MCL of the GI tract.

Table1. Laboratory testing results

PARAMETERS	VALUE	NORMAL RANGE
CBC RESULTS		
Hb	121	13 – 17 g/dL
WBC	245	4 – 10 G/L
Neutrophils	1.4	1.5 – 7 G/L
Platelets	54	150 – 400 G/L
BIOCHEMISTRY		
Creatinine	246	60 – 120 µmol/L
eGFR (CKD-EPI)	23	≥ 90 mL/min/1.73m²
BUN	58	3 – 20 mmol/L
ALP	650	26 – 88 IU/L
Serum LDH	718	90 – 180 IU/L
Uric acid	2.5	0.24 – 0.51 mmol/L

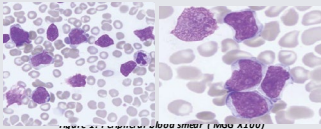


Figure 1: Blastoid lymphoid cells smear (100x100)



Figure 2: Endoscopic view of ulcerated partially obstructing large mass in the cecum (5).

Cell marker studies identified a CD5+ lymphoproliferative disorder in a patient with a high leucocyte count, lymphadenopathy and splenomegaly.

The differential diagnosis included CLL, MCL and MZL. The immunophenotype was not indicative of CLL (CLL score 1/5). MZL was unlikely in view of the morphology and marked lymphocytosis.

Given the previous cytological, immunophenotyping-immunostaining and imaging results, the most likely diagnosis was MCL in its blastoid variant as this was confirmed by the cyclin D1 positivity and the presence of t(11;14)(q13;q32) which translocates the CCND1 gene (11q13) so that it is adjacent to the IGH gene (14q32).

MCL is a rare disease accounting for <10% of all NHLs. It is usually a disease of the elderly, especially males, and presents with advanced stages disease. Blastoid MCL involvement are common. Peripheral blood involvement is common but the lymphocytosis is usually mild[1].

Marked lymphocytosis, as in this case, is unusual but well described initially as leukemic cells; the morphology and immunophenotype may mimic B-cell prolymphocytic leukemia.

However, cases with circulating atypical lymphoid cells but no marrow involvement suggest leukemic spillover from nodular extranodal disease with secondary marrow suppression, rather than true leukemic transformation.

So, the GI imaging studies especially colonoscopy with biopsy was of great tool to confirm the diagnosis of B-MCL. MCL in the GI tract usually manifests as multiple lymphomatous polyps, making this case a rare event.

Flow cytometry showed brightly CD45 positive cells but CD34 negative, thus, confirming that the atypical cells were mature lymphoid cells rather than blasts. Further, immunophenotyping of these atypical mature lymphoid cells showed positive B cell markers (CD5, CD19, CD20, CD22, CD79a) with surface kappa light chain restriction. In addition, the atypical cells expressed aberrant CD10 [1].

The present case constitutes a rather infrequently reported clinical presentation of B-MCL with extra nodal GI involvement.

Its leukemic presentation is an exceedingly uncommon event. In addition, differentiating blastoid MCL from lymphoblastic lymphoma and centroblastic large cell lymphoma is extremely difficult (especially when BM involvement occurs) solely on the basis of morphology, necessitating the use of additional analytical techniques such as immunophenotyping and molecular studies [2].

Imaging studies play a critical role in the assessment of MCL, including staging. The ESMO guideline is recommended BM biopsy and CT scans of the neck, chest, abdomen, and pelvis, as part of the initial workup for MCL [4].

A PET-CT scan is highly recommended for the rare early stages (I-II) before starting localized radiotherapy. Additionally, the Ki-67 index, which measures cell proliferation, is assessed to gauge the aggressiveness of the lymphoma. High Ki-67 levels are indicative of a more aggressive disease course. Genetic studies are performed to identify mutations in genes such as TP53 and CCND1, which are often associated with MCL and can influence prognosis and treatment [5].

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