CASE REPORT – OPEN ACCESS

International Journal of Surgery Case Reports 5 (2014) 302–305

Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.casereports.com

Relapse of acute myeloid leukemia manifested by cholecystitis: A case report and review of the literature

Arash Azin[a], Jennifer M. Racz[b], M. Carolina Jimenez[d], Supreet Sunil[a], Anna Porwit[c], Timothy Jackson[d,e], Allan Okrainec[d,e], Fayez Quereshy[b,d,e,*]

[a] Faculty of Medicine, University of Toronto, Canada
[b] Department of Surgical Oncology, Princess Margaret Cancer Centre, University of Toronto, 610 University Avenue, Toronto, Ontario M5G 2M9, Canada
[c] Department of Laboratory Medicine and Pathobiology, University of Toronto, Canada
[d] Division of General Surgery, University Health Network, Canada
[e] Department of Surgery, University of Toronto, Canada

ARTICLE INFO

Article history:
Received 25 January 2014
Accepted 5 February 2014
Available online 19 March 2014

Keywords:
Cholecystitis
Acute myeloid leukemia
Gallbladder
Cholecystectomy

ABSTRACT

INTRODUCTION: AML is the most common form of leukemia in adults. In rare circumstances AML may present in the form of extra-medullary disease. Gallbladder infiltration with myeloblasts is rare and only a few cases exist in the literature describing this entity.
PRESENTATION OF CASE: We present a rare case of AML relapse in the form of extramedullary infiltration of the gallbladder in a 50-year-old male patient. The leukemic infiltration presented as symptomatic cholecystitis and sepsis. A laparoscopic cholecystectomy was performed and the gallbladder was pathologically examined. Histopathologic examination demonstrated multiple scattered, highly atypical single cells admixed with some plasma cells, small lymphocytes and macrophages consistent with leukemic infiltration. The abnormal cells demonstrated immunohistochemical staining for CD68, CD33 and CD117. The patient did well post-operatively but the relapse precluded him from bone marrow transplantation.
DISCUSSION: Although AML is relatively common, 3 cases per 100,000 population, extramedullary disease in the form of gallbladder infiltration is exceedingly rare. An extensive review of the literature revealed only four cases of myeloid infiltration of the gallbladder. To our knowledge this is the only case of relapsing disease in the form of gallbladder infiltration presenting as symptomatic cholecystitis in a pre-bone marrow transplantation patient.
CONCLUSION: This case highlights the importance of maintaining a high index of suspicion of atypical manifestations of AML when managing refractory sepsis. Extramedullary manifestations of AML in the form of gallbladder infiltration must be considered in the differential diagnosis of patients with a history of myeloid malignancies and for patients whom fail conventional non-operative management.
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1. Introduction

Acute myeloid leukemia is a hematological malignancy characterized by a variable clinical course. Patients diagnosed with acute leukemia may present with myeloid sarcoma, leukemic infiltration of the gastrointestinal tract or mediastinum, and in rare circumstances, infiltration of the biliary system with myeloid cells. We report a case of a patient who presented with acute cholecystitis as a manifestation of his acute myeloid leukemia through infiltration of the gallbladder wall with immature myeloid lineage cells.

2. Case

A 50-year-old male receiving care at our institution for acute myeloid leukemia (AML) in his second morphological free state, presented to the Emergency Department with worsening diarrhea, intermittent nausea and vomiting, fever, right upper quadrant pain, and decreased oral intake in the setting of a 2-month history of chronic cholecystitis. Initial laboratory investigations revealed pancytopenia (hemoglobin 98 g/L [120–160 g/L], platelets 17×10^9/L [$150–400 \times 10^9$/L], WBC 1.1×10^9/L [$4.0–11.0 \times 10^9$/L] with 0% blasts), and an elevated alkaline phosphatase (ALP) of 218 U/L [40–150 U/L] with a total bilirubin of 28 μmol/L [≤22 μmol/L].

* Corresponding author at: Division of General Surgery, Toronto Western Hospital, 399 Bathurst Street, Main Pavilion, Room 8-320, Toronto, Ontario M5T 2S8, Canada. Tel.: +1 416 603 5553.
E-mail addresses: Arash.Azin@mail.utoronto.ca (A. Azin), Jennifer.Racz@gmail.com (J.M. Racz), MCarolina.Jimenez@uhn.ca (M. Carolina Jimenez), Supreet.Sunil@mail.utoronto.ca (S. Sunil), Anna.Porwit@uhn.ca (A. Porwit), Timothy.Jackson@uhn.ca (T. Jackson), Allan.Okrainec@uhn.ca (A. Okrainec), Fayez.Quereshy@uhn.ca (F. Quereshy).

http://dx.doi.org/10.1016/j.ijscr.2014.02.003
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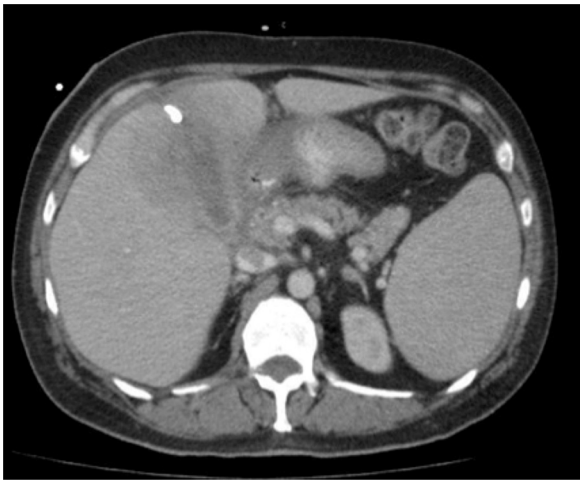

Fig. 1. Computed axial tomography (CAT) demonstrating thickening of the gallbladder with surrounding edema and inflammation.

He was initially diagnosed with AML in May 2012, which had transformed from chronic myelogenous leukemia (CML) with normal cytogenetics. As a result, he was started on induction therapy with standard-dose Cytarabine and Idarubicin and achieved a morphological free state by June 2012. Unfortunately, in November 2012, he was found to have a relapse of his AML that was confirmed by bone marrow biopsy (10–12% blasts), peripheral blood smear (5–6% blasts), flow cytometry and an elevated LDH of 638 U/L [125–220 U/L]. As a result, he underwent re-induction therapy with NOVE-HiDAC (Mitoxantrone, Etoposide and high-dose Cytarabine) in December 2012, and once again achieved a morphological free state without count recovery. He was subsequently accepted for allogenic bone-marrow transplant, with a tentative date of June 2013.

While awaiting a matched donor for bone-marrow transplantation, he was admitted for cholecystitis and febrile neutropenia, the former of which was managed conservatively with broad-spectrum intravenous antibiotics (Piperacillin-Tazobactam) given his thrombocytopenia and perioperative risk profile. Six days following discharge, he was readmitted with increasing nausea, vomiting, fever, and RUQ pain. In addition to a second course of antibiotics (Vancomycin, Colistin, Flagyl and Voriconazole), a percutaneous cholecystostomy tube was also placed for source control. Computerized axial tomography (CAT) of the abdomen was performed and demonstrated gallbladder distension and thickening consistent with cholecystitis. However, there was no evidence of peri-cholecystic fluid or associated inflammatory changes around the gallbladder. Dilation of intrahepatic ducts and common bile duct were not seen (Fig. 1). He was discharged home with improvements in his abdominal pain and oral intake; however, soon after discharge, he had a relapse of his cholecystitis-related symptoms and was once again managed conservatively and re-admitted to hospital. A percutaneous contrast study evaluating drain placement was performed and demonstrated persistent obstruction of the cystic duct. For these reasons, the cholecystostomy tube was left in situ.

Approximately 7 weeks after his initial presentation, surgical consultation was sought in order to determine his eligibility to undergo an elective cholecystectomy for definitive source control, a prerequisite for bone-marrow transplantation. Although definitive surgical management was ultimately delayed secondary to subsequent hospital admissions for febrile neutropenia and cholecystitis, he eventually underwent laparoscopic cholecystectomy approximately 3 months following his initial presentation.

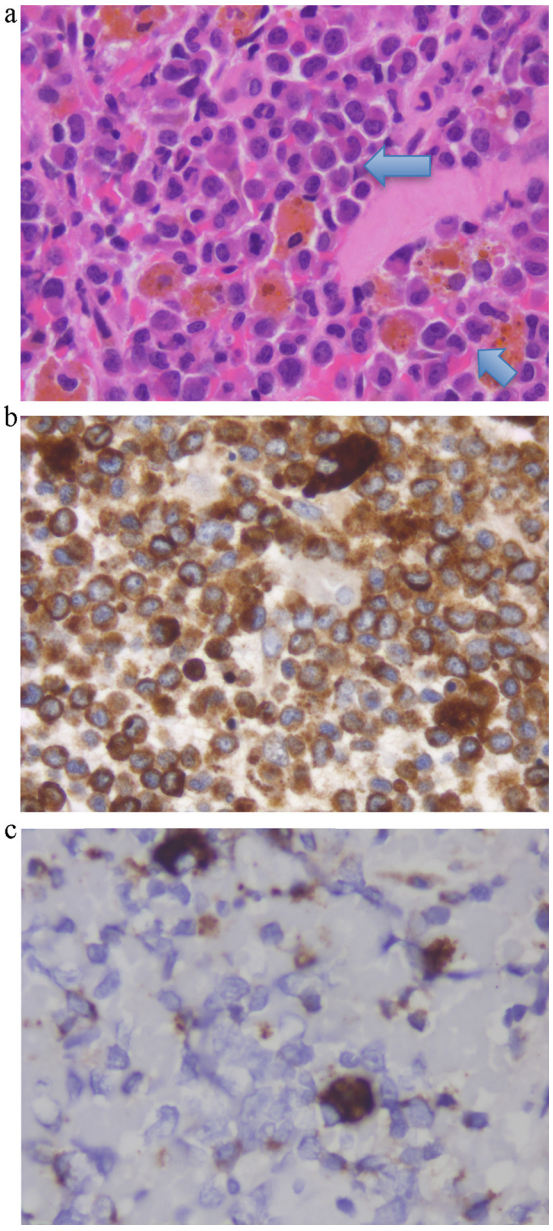

Fig. 2. (a) Hematoxylin–Eosin stained section from gallbladder wall showing diffuse infiltrates of mononuclear cells with irregular nuclei and some with prominent nucleoli. Scattered lymphocytes, plasma cells and macrophages are also present. (b) The majority of cells stain positive for CD68 KP-1 epitope, which is positive not only in macrophages but also in myeloid and monocytic precursors.[8] (c) Only rare cells stained positive for CD68-R PGM-1 epitope, which is more macrophage specific.[9]

Intra-operatively, there were extensive adhesions in the right upper quadrant. During the dissection, the gallbladder was entered with subsequent extrusion of purulent and necrotic debris. Overall, the patient tolerated the procedure well and there were no intra-operative complications. Macroscopically the resected gallbladder showed a tan colored fibrinous exudate and a completely necrotic back wall. Histopathologic examination of the specimen demonstrated multiple scattered, highly atypical single cells admixed with some plasma cells, small lymphocytes and macrophages consistent with leukemic infiltration (Fig. 2a). The abnormal cells demonstrated immunohistochemical staining for CD68 (KP1), CD33 and CD117, however, CD34 was not increased (Fig. 2b and c). These findings were consistent with the manifestation of his previously known myelomonocytic leukemia, and the second relapse of his disease. A peripheral blood smear and bone-marrow aspirate and

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