Abstract
Introduction
Nontraumatic perforations of the small bowel are uncommon complications of some chemotherapy regimens. When there are no metastatic implants on bowel, exact pathophysiologic mechanism is obscure.

Case Report
We report a case of a patient undergoing treatment for Acute Monocytic Myeloid Leukaemia with decitabine who presented a spontaneous jejunal perforation.

Discussion
This study presents the first reported case of decitabine-induced perforation. Early diagnosis and prompt surgical approach represent the pinnacle in the management of this uncommon complication.

Introduction
Bowel perforations are uncommon complications of some chemotherapy regimens. There are no statistics available about their incidence, but some agents, mainly bevacizumab and paclitaxel, are well known to be associated with this ominous side effect. The pathophysiologic pathways that lead to nontraumatic perforations are not completely understood, and may be generally related to metastatic implants on bowel which undergo transmural necrosis following chemotherapy. However, when there are no implants or disease on the affected bowel, the mechanism is more obscure, probably enrolling microvascular changes, poor local wound healing or aggravation of previous enteritis. They may lead to severe complications since they affect immunocompromised patients during their ongoing treatments. Prompt diagnosis and surgical treatment are often required to avoid further morbidity and mortality. To report a case of small bowel perforation treatment for Acute Monocytic Myeloid Leukaemia with decitabine and make a brief literature review.

Case Report
Male, 61 years old, undergoing chemotherapy with decitabine for the last 2 months due to Acute Monocytic Myeloid Leukaemia, was admitted at the Emergency sector complaining of acute abdominal pain mostly on left lower abdomen for 3 days. He had not used any corticosteroid or nonsteroidal agent recently, had no antecedents of tuberculosis or autoimmune diseases, had a recent negative HIV serology test, and had no recent abdominal trauma. At physical examination, patient was alert and oriented; he presented normal blood pressure, respiration, and temperature; tachycardia (pulse 112 bpm); at abdominal examination, he had intense pain during deep palpation of lower abdomen, with signs of peritoneal irritation. Current laboratory studies showed leucopenia (0.9 x 10^3 WBC/mm3 without left shift), low platelet count (49 x 10^3/mm3) and no further abnormalities. On CT scan it was observed pneumoperitoneum and free fluid in left lower abdomen and pelvis, with a thickened segment of small bowel and contrast leakage (Figure 1). A laparotomy was carried out and an isolated 3-cm perforation of jejunum on the antimesenteric side, 40 cm distal to Treitz ligament was observed, with no foreign body, gross infiltration or signs of obstruction. It was opted for a segmental resection due to extension of perforation (>30% of bowel circumference). Patient has an uneventful postoperative outcome and was discharged after 13 days. Histopathologic examination revealed a chronic inflammatory process without signs of malignancy. Another chemotherapy regimen (idanubicin/zytarbabin) was introduced 30 days after discharge without further gastrointestinal complications. This work conforms to the values laid down in the Declaration of Helsinki (1964). The protocol of this study has been approved by the relevant ethical committee related to our institution in which it was performed. All subjects gave full informed consent to participate in this study.

Discussion
Despite their somewhat rare occurrence, small bowel perforations during chemotherapy are well known and potentially fatal complications. Some agents, such as bevacizumab and paclitaxel, have their risk for this occurrence more extensively described. The perforations may occur due to secondary involvement of small bowel by the primary disease or due to direct effect of chemotherapy regimen on small bowel. When there is involvement of bowel by disease, necrosis of lesions caused by treatment lead to perforation. However when there is no metastatic bowel involvement, the exact mechanism is uncertain. It may be caused by microvascular changes that lead to ischemic injury; poor wound healing after small trauma; and secondary neutropenic enterocolitis. Decitabine is the most widely used inhibitor of DNA methylation, which triggers demethylation leading to consecutive reactivation of epigenetically silenced tumor

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Case report

Decitabine-induced small bowel perforation: Case Report

Case report

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All possible factors were identified, as histopathologic examination showed no signs of leukemic infiltration, no distal obstruction or foreign bodies were identified, and further investigation showed no evidences of primary inflammatory bowel disease. Furthermore, patient had not received corticosteroid or radiation therapy. Although the patient was neutropenic, perforation site was very distant to usual location of perforations secondary to neutropenic enterocolitis (distal small bowel and cecum). Thus, after extensive investigation, there was no other possible factor than chemotherapy to be enrolled in the etiology of perforation. Patient had an uneventful outcome probably due to early diagnosis and surgery carried out with minimum peritoneal contamination. Evolution to severe sepsis in such immunocompromised subjects might lead to ominous consequences. Hence patients undergoing chemotherapy which present acute abdominal pain demand a profound investigation at the emergency sector once this potential complication must be promptly treated due its potentially fatal outcome when diagnosis is delayed.

References