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Redefining Ultrasound's role in early diagnosis of hepatosplenic candidiasis during neutropenia: A pediatric case report in solid tumor

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ABSTRACT

We report a case of hepatosplenic candidiasis (HSC) in a neutropenic child with ovarian teratoma. Using high-frequency ultrasonography, we detected subtle hypoechoic pseudonodules in the spleen during profound neutropenia (ANC 30/μL), challenging the dogma that lesions only appear after immune recovery. These precursors evolved into classic "bull's-eye" lesions. *Candida tropicalis* was confirmed via splenectomy. This case highlights high-frequency ultrasound's role in early HSC diagnosis before neutrophil recovery.

1. Introduction

Invasive fungal infections (IFIs) have increased over recent decades in cancer patients. While invasive candidiasis is a well-known complication in hematologic malignancies, its incidence in pediatric solid tumors is significantly lower, generally below 5% [1,2]. The most frequently identified species are *C. albicans*, *C. parapsilosis*, *C. tropicalis*, and *C. glabrata*. Hepatosplenic candidiasis (HSC) represents the most prevalent form of chronic disseminated candidiasis (CDC), a rare manifestation of invasive candidiasis that typically occurs in patients with prolonged neutropenia, defined as an absolute neutrophil count (ANC) below 500/μL for more than 10 days [3]. The reported incidence of CDC ranges from 2.0% to 7.4% in patients diagnosed with acute leukemia; however, HSC is rarely described in individuals with lymphoma or other solid tumors [4]. According to the European Organization for Research and Treatment of Cancer and the Mycoses Study Group (EORTC/MSG) criteria, invasive candidiasis and HSC are classified as proven, probable, or possible. A “proven” diagnosis requires histopathological or microbiological identification of *Candida* from a sterile site or affected tissue [5]. A “probable” diagnosis requires host factors (e.g., prolonged neutropenia), clinical/radiological evidence, and indirect mycological evidence (such as positive BDG or PCR). Cases meeting only host and clinical criteria, without mycological evidence, fall into the “possible” category. Consequently, many reported cases of HSC represent merely possible or probable infections, as obtaining tissue for proven diagnosis is difficult. Furthermore, without histopathological confirmation, identical clinical and radiographic presentations can be caused by other etiologies, including bacterial/mycobacterial infections, drug toxicity, or metastatic progression of the underlying malignancy [6].

Typically, liver and spleen lesions become evident after bone marrow reconstitution, with recovery from chemotherapy-induced neutropenia. At this stage, small target-like abscesses (“bull's-eye” lesions) can be visualized on ultrasound, computed tomography (CT), or magnetic resonance imaging [7]. We report the rare diagnosis of HSC in a pediatric patient suffering from a solid tumor, in whom splenic and hepatic involvement was first documented on abdominal ultrasonography during a prolonged neutropenic phase.

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2. Case presentation

A 10-year-old girl diagnosed with immature bilateral ovarian teratoma (grade 3, stage 4 with hepatic metastases, ascites, and pleural effusion) was initially treated with 4 courses of cisplatin, etoposide, and bleomycin (PEB) followed by one course of ifosfamide, carboplatin, and etoposide (ICE), completing therapy. She relapsed nine months later with a paracervical/parametrial mass and widespread deep lymphadenopathy. Salvage chemotherapy was initiated with the ICE regimen. Following her second ICE course, she experienced an episode of febrile neutropenia requiring hospitalization. Restaging imaging demonstrated a significant dimensional reduction of the lesions, and a third course of ICE (dose-reduced by 25%) was administered. Twelve days after the end of this final chemotherapy cycle, the clinical picture severely deteriorated.

Definition of Time Course: Day 0 is defined as the onset of fever and hospital admission (12 days post-chemotherapy). Fig. 1 shows the progression of ultrasound and CT scan images and granulocyte counts over time.

On day 0, the patient, still granulocytopenic, presented with fever, diarrhea, and elevated inflammatory markers. Her general condition was poor, and she was unresponsive to broad-spectrum antibiotics. A chest CT scan revealed multiple bilateral parenchymal thickenings with a peripheral ground-glass halo (Fig. 2).

During this specific neutropenic episode (from day 0 to final microbiological diagnosis), more than 20 sets of blood cultures were

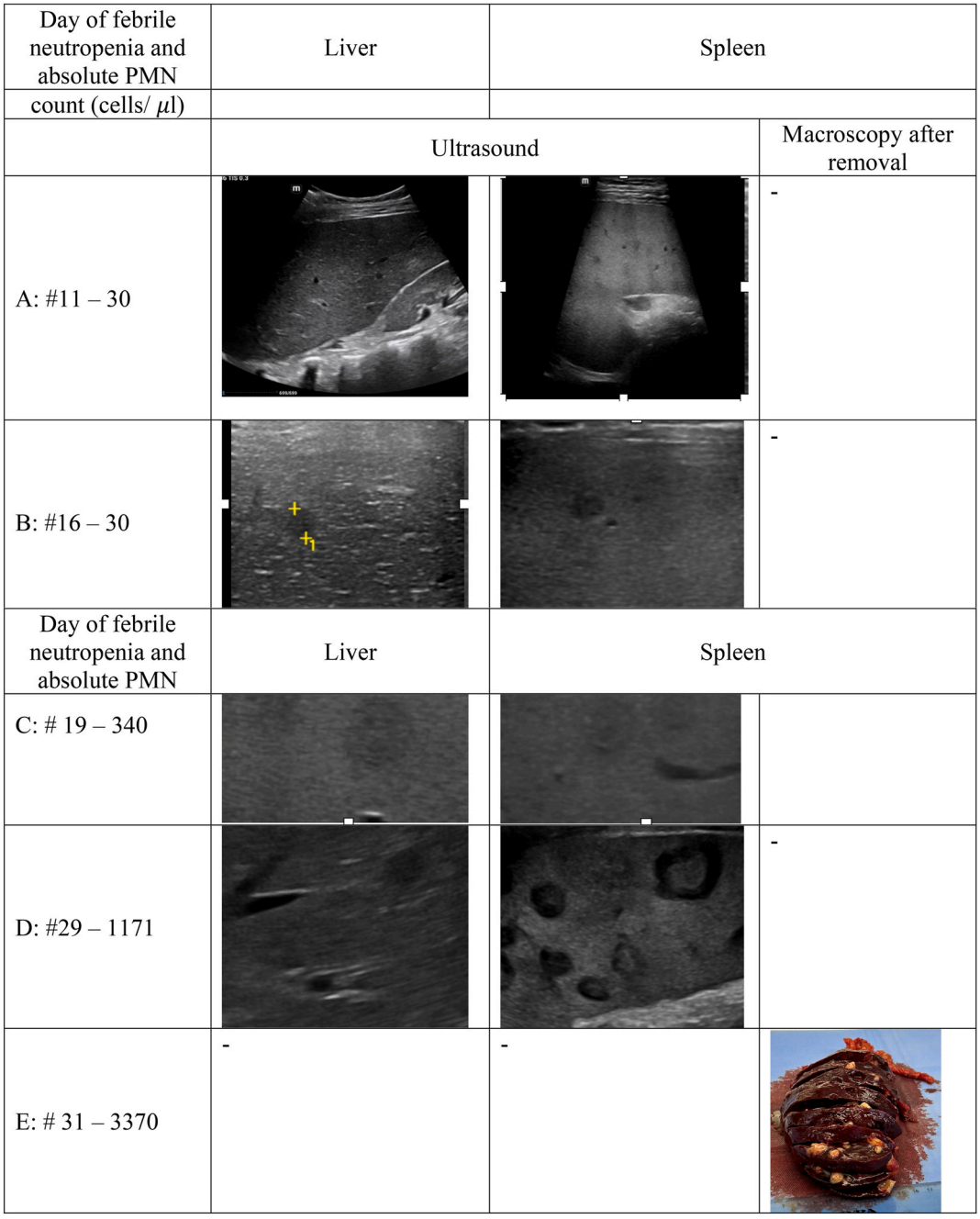

Fig. 1. Neutrophils absolute count, liver and spleen ultrasound and spleen macroscopy after removal. Macroscopic appearance of the spleen reveals numerous cavitated lesions distributed both on the outer capsular surface and throughout the parenchyma. These formations are filled with yellowish, purulent material and bordered by a similar-appearing cuticle.

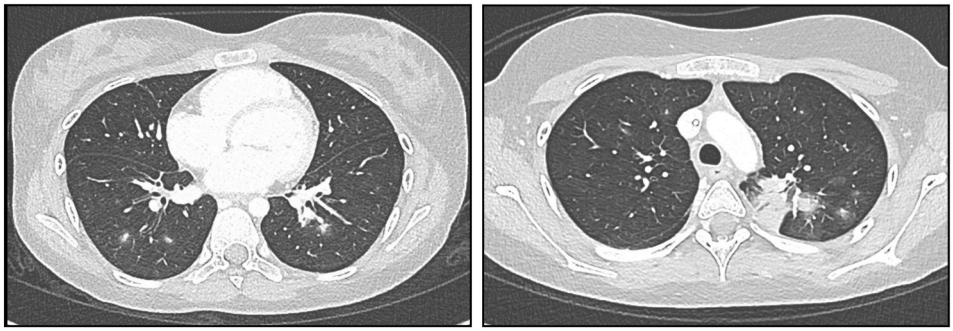

Fig. 2. Initial chest CT findings. Axial images performed at clinical deterioration demonstrate multiple millimetric parenchymal nodular thickenings with peripheral ground-glass opacities.

obtained, all of which remained persistently negative. Serum 1,3-beta-D-glucan (BDG) was not assessed (due to limited precision, insufficient specificity, and a high risk of false positives in pediatric oncology patients [8]). Serum galactomannan, bronchoalveolar lavage with fungal culture, galactomannan testing, and polymerase chain reaction (PCR) for *Pneumocystis jirovecii* were all negative.

On day +1, notwithstanding severe neutropenia (ANC 30/μL), the initial abdominal ultrasound performed using a GE Logiq E10 US device with a 6-15 MHz linear probe identified millimetric hypoechoic pseudonodular areas throughout the splenic parenchyma, with no hepatic abnormalities (Fig. 1A). These lesions had not been observed in previous examinations and were better visualized using the linear transducer.

On day +4, a second thoracic CT scan was performed due to clinical worsening. The field of view (FOV) included part of the upper abdomen and showed multiple millimetric hypodense splenic areas (6-8 mm). These lesions appeared better defined with an initial peripheral rim, despite persisting neutropenia. Similar lesions were now visible in the liver (Fig. 1B). These findings were considered suspicious for a fungal infection, and empirical liposomal amphotericin B was started.

From 5th day onwards, serial abdominal ultrasounds showed progressive hepatosplenomegaly (spleen increasing from 14.5 cm to 16 cm) and an increasing number of subtle hypoechoic pseudonodular splenic and hepatic lesions. Concurrently (from day +5), a new, vaguely hyperechoic area was identified in the right renal corticomedullary region during ultrasound monitoring.

On day +19 (approx.), with rising neutrophils (ANC 340/μL), hepatic and splenic lesions became more evident and gradually developed a target-like appearance (Fig. 1C). The patient experienced rapid deterioration with persistent fever, abdominal pain, melena, and refractoriness to platelet transfusion. On day +29, with moderate neutropenia (ANC 680/μL), an urgent abdominal CT scan was performed due to suspected gastrointestinal bleeding. The scan demonstrated an increased number of millimeter-sized hypodense areas throughout both hepatic lobes and the splenic parenchyma, with a focal triangular splenic lesion suggestive of infarction, and new renal corticomedullary hypodensities in both kidneys. Radiological lesions acquired a thick hypoechoic peripheral rim (Fig. 1D), becoming clearly suggestive of HSC as the ANC exceeded 1000/μL.

Treatment and Outcome: despite two weeks of empirical antifungal therapy and rising neutrophils (ANC 1710/μL), fever persisted. Follow-up CT showed resolving lung lesions but increasing hypodense lesions in the spleen (some becoming partially exophytic and confluent), liver, and kidneys. Ultrasound confirmed evolving splenic "bull's-eye" lesions and stable hepatosplenomegaly. A new, vaguely hypoechoic intracortical area with a hyperechoic halo was also confirmed in the right renal corticomedullary region.

Given the risk of splenic rupture from marked splenomegaly and extensive lesions, a splenectomy was performed on day +45 (Fig. 1E). Histology showed multiple cavitated lesions filled with yellowish-purulent material and necrotizing abscesses. Microscopy revealed necrotic-leukocytic foci, bordered by histiocytes, lymphocytes, and plasma cells (Fig. 3).

Histological examination revealed numerous and extensive aggregates of medium-to-large-sized spores, pseudohyphae, and fungal hyphae. Culture yielded a strain of *Candida tropicalis*, confirming the diagnosis of HSC according to EORTC/MSG criteria. Post-splenectomy, hepatic lesions evolved similarly to prior splenic patterns. Intravenous anidulafungin and fluconazole were initiated. Methylprednisolone (0.5-1 mg/kg/day for 3 weeks, then tapered) was added. The patient showed rapid clinical improvement, and antifungal therapy continued for over six months.

3. Discussion

CDC is a rare complication of immunosuppression, presenting with persistent fever or non-specific symptoms. A recent review identified *C. tropicalis* as the most prevalent species in HSC cases [9]. While *C. albicans* accounted for over half of invasive *Candida* infections a decade ago, its proportion has declined. This epidemiological shift towards non-*albicans* species, particularly *C. tropicalis*, is multifactorial. Although phylogenetically related to *C. albicans*, the emergence of *C. tropicalis* reflects a combination of its specific virulence factors (such as enhanced mucosal adhesion and biofilm formation), geographic trends, and the intensification of oncological therapies that promote fungal translocation [10,11]. Furthermore, widespread azole prophylaxis continues to play a contributing role by exerting selective pressure, as *C. tropicalis* exhibits an increasing capacity to persist and adapt in hospital environments [12,13].

HSC poses a significant diagnostic challenge due to the frequent absence of fungemia (up to 70% of cases [6]), and imaging findings that are described as dependent on ANC [14,15].

Regarding laboratory diagnostics, while 1,3-beta-D-glucan (BDG) is a recognized biomarker in adult cohorts, its utility for diagnosing

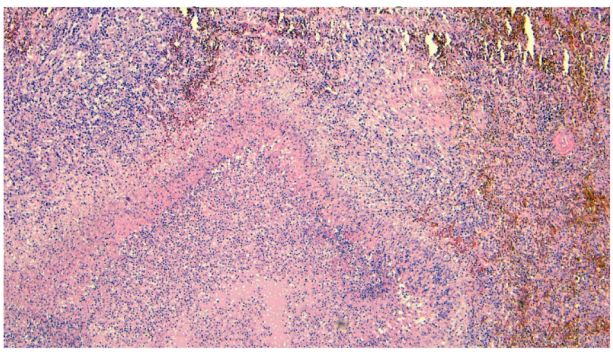

Fig. 3. microscopic pathological examination exhibits necrotic-leukocytic foci, which are circumferentially bordered by a "palisade" of histiocytes interspersed with lymphocytes.

invasive candidiasis in pediatric oncology patients remains highly limited. Literature demonstrates that BDG in children exhibits variable sensitivity and specificity, resulting in poor overall accuracy and a low positive predictive value [16]. International pediatric guidelines do not recommend BDG to guide clinical decisions in this population [8]. The optimal diagnostic cut-off for children is currently unknown, and baseline BDG levels in healthy pediatric patients can be intrinsically higher than in adults, leading to frequent false positives, further exacerbated by common pediatric oncology variables such as mucosal damage and broad-spectrum antibiotic use [17,18]. Consequently, BDG is not recommended as a standalone diagnostic instrument in pediatric oncology, making the reliance on advanced, early imaging even more critical.

CDC pathogenesis involves *Candida* translocating chemotherapy-damaged intestinal mucosa. Once in hepatosplenic sinusoids [19], the host immune response strongly influences imaging patterns. With neutrophil recovery, an exaggerated inflammatory response resembling Immune Reconstitution Inflammatory Syndrome (IRIS) may occur [20], leading to paradoxical worsening and formation of granulomatous "target", "bull's-eye" or "wheel-within-wheel" lesions described in on imaging studies [14,21]. Localized immune activation can sequester fungi [22,23], explaining persistently negative cultures and incomplete or slow response to antifungal therapy alone; consequently, therapeutic strategies that address this excessive host inflammatory response, such as corticosteroids, may be necessary [6].

Historically, CT and MRI scans were considered more sensitive than ultrasound for CDC detection, though they were all thought ineffective before neutrophil recovery [24,25]. However, recent and rapid advances in ultrasound techniques have significantly expanded its diagnostic capabilities in pediatric patients, demonstrating a transformative role in early detection [26]. Modern ultrasound technologies, including microbubbles for contrast enhancement, elastography, ultrafast Doppler, and high-frequency imaging offering resolutions down to 30 μm, can now provide highly sensitive bedside evaluations for organ injury, viable tumors, and vascular pathologies [26]. These innovations are particularly crucial in pediatric oncology, as they enable early diagnosis while avoiding radiation exposure and lengthy procedures like MRI [26]. Above all, the introduction of next-generation ultrasound systems equipped with high-frequency probes provides exceptionally high anatomical resolution. This allows for the early detection of small or subtle splenic and hepatic lesions that often remain undetectable with conventional probes—particularly convex transducers, which are typically used for abdominal imaging but offer lower detail for superficial structures [27–29].

In our patient, splenic lesions were first detected during severe neutropenia (ANC 30/μL) as hypoechoic pseudonodules, evolving into "bull's-eye" lesions as neutrophils rose. As the granulocyte count increased, these lesions progressively grew in both number and size, and their pattern changed (Fig. 1). This highlights the capability of modern ultrasounds to detect CDC lesions at stages previously considered invisible in the absence of neutrophil recovery. Radiological progression mirrored neutrophil trends and involved multiple organs, consistent with previous reports [9]. HSC lesions may decrease or temporarily disappear during neutropenia [24], likely due to reduced inflammatory containment and lesion indistinctness, then reappear with neutrophil recovery [14,15]. Diagnosis in this case was ultimately confirmed only through splenectomy, more than six weeks after symptom onset, with *C. tropicalis* never isolated from blood. This diagnostic odyssey underscores the challenges in identifying HSC, particularly in cases with elusive fungemia, and emphasizes the critical need for advanced diagnostic approaches. Prompt HSC recognition is essential for initiating combined antifungal and anti-inflammatory therapy. Guidelines recommend prolonged treatment until complete radiologic resolution [30], and splenectomy is a feasible option in refractory cases [31].

Clinicians should maintain high suspicion for HSC in febrile neutropenic pediatric oncology patients with negative cultures and antibiotic-refractory fever. This case illustrates that modern ultrasound is an invaluable, radiation-free tool for early fungal lesion detection—even during profound neutropenia—and may significantly improve outcomes. Further studies are warranted to define ultrasound's precise role alongside CT/MRI in CDC diagnostic pathways.

CRediT authorship contribution statement

Francesca Cappozzo: Conceptualization, Methodology, Data curation, Writing – original draft. **Teresa Battaglia:** Conceptualization, Methodology, Data curation, Writing – original draft. **Marcello Mariani:** Conceptualization, Project administration, Supervision, Writing – review & editing. **Marta Nebiolo, Virginia Livellara, Gabriele Gaggero, Elena Arkangelskaya, Maria Beatrice Damasio, Lorenzo Anfigeno, Camilla De Luca, Stefano Avanzini, Federico Palo, Massimo Conte:** Investigation, Resources, Data curation, Validation, Writing – review & editing. **Elio Castagnola:** Supervision, Validation, Writing – review & editing.

Consent

Please declare that you have obtained written and signed consent to publish the case report from the patient or legal guardian(s).

Please state that consent has been obtained from the patient or legal guardian(s)

Written informed consent was obtained from the patient or legal guardian(s) for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Please state any sources of funding for your research

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Please state any competing interests

No conflict of interest to declare.

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