Aspergillosis is an uncommon opportunistic infection, usually caused by *Aspergillus fumigatus*. Invasive aspergillosis is one of the major infectious complications in immunocompromised patients with severe neutropenia, showing poor prognosis. Renal involvement by *Aspergillus* is usually presented as formation of abscesses associated with disseminated aspergillosis. Herein, we report a case of invasive renal aspergillosis with renal infarction in an acute lymphoblastic leukemia. To the best of our knowledge, this is the first report of renal infarction by invasive renal aspergillosis as an initial presentation in acute leukemia.

CASE REPORT

A 5-year-old girl was admitted to hospital with fever and abdominal pain for 2 days. The patient had been diagnosed with acute lymphoblastic leukemia 8 months ago and was on consolidation phase chemotherapy with cytarabine, L-Asparaginase, and intrathecal methotrexate. At the time of admission, the initial solid consolidation chemotherapy with cytarabine, L-Asparaginase, abdominal pain for 2 days. The patient had been diagnosed with perinephric fluid collection. The perfusion defect area of the kidney corresponded to vascular territory of right upper, middle, and lower anterior segmental arteries (Fig. 1A). The inferior vena cava, right renal vein, and visible right renal artery were well delineated without intraluminal thrombosis or luminal narrowing (Fig. 1B). On coronal images, mild dilatation of the right renal pelvis with internal hyperdense portion and delayed excretion were observed. Moreover, there was a long linear hyperdense lesion in the right psoas muscle. However, the fat plane between the right kidney and psoas muscle were intact and no definite enhancing lesion was observed in the right kidney. To find out possible cause of renal infarction, chest CT was performed, but there was no evidence of pulmonary aspergillosis.

Presumptive diagnosis of thrombotic event or infective etiology was made for the renal infarction. Subsequently, the patient was treated with empirical antibacterial and antifungal agents. After 5 days of empirical therapy, the fever subsided. However, the patient still complained of diffuse severe abdominal pain. Physical examination showed rigid, distended abdomen with right abdominal tenderness. Follow-up contrast-enhanced CT scan revealed absent perfusion in entire right renal parenchyma. Right renal fossa and perinephric space were filled with approximately 10 cm-sized hypodense mass with internal air bubbles and rim enhancement (Fig. 1C). The patient underwent nephrectomy and adrenalectomy under the impression of retroperitoneal abscess. Because of the extensive abscess formation, the duodenum, gallbladder, and jejunum were perforated and right posterior abdominal wall showed necrotic change. Histologic examination revealed diffuse renal necrosis with abundant branching fungal hyphae infiltrating the renal vessels and parenchyma, consistent with *Aspergillus* species (Fig. 1D). The final histologic diagnosis was invasive aspergillosis and identification of *A. fumigatus* from the specimen was based on cultural and morphologic criteria.

Amphotericin B was administered postoperatively for 4 months. During the therapy, the absolute neutrophil count level was maintained above 0.1 x 10^9/L and consolidation phase of chemotherapy was postponed. The serial galactomannan antigen level was not increased (< 0.5 index). Blood and urine cultures showed no growth of either bacteria or fungus. For the evaluation of the fever and mild abdominal pain, contrast-enhanced abdomen computed tomography (CT) was performed.

Contrast-enhanced abdominal CT scan revealed a sharply demarcated perfusion defect in the lower pole of the right kidney with perinephric fluid collection. The perfusion defect area of the kidney corresponded to vascular territory of right upper, middle, and lower anterior segmental arteries (Fig. 1A). The inferior vena cava, right renal vein, and visible right renal artery were well delineated without intraluminal thrombosis or luminal narrowing (Fig. 1B). On coronal images, mild dilatation of the right renal pelvis with internal hyperdense portion and delayed excretion were observed. Moreover, there was a long linear hyperdense lesion in the right psoas muscle. However, the fat plane between the right kidney and psoas muscle were intact and no definite enhancing lesion was observed in the right kidney. To find out possible cause of renal infarction, chest CT was performed, but there was no evidence of pulmonary aspergillosis.

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developing these infections because of the degree and duration of neutropenia.1,4 The conditions that predispose to this infection are underlying malignancy including hematologic malignancy, comorbidities (graft-versus-host disease, diabetes, cytomegalovirus infection, renal or liver dysfunction, chronic obstructive pulmonary disease, postsurgery), medications (steroids, chemotherapy), and transplantation.1,2

In immunocompromised patients, Aspergillus infection can be invasive in nature and can disseminate throughout the body. The usual locations of involvement by Aspergillus include the lungs (most common primary site), central nervous system, sinuses, and skin.5 The lung is usual portal of entry and initial site of dissemination. However, entrance of invasive aspergillosis in our case was not identified, despite of extensive clinical and radiologic studies. Although the involvement of the urinary tract by Aspergillus is uncommon, Aspergillus infection of the kidney constitutes about 30% of total renal fungal infection and the majority results from disseminated infection.3,4 Aspergillosis of kidney may occur by 3 pathways: by ascending infection from the lower tract, from hematogenous renal involvement, or because of aspergillosis of the renal pelvis with bezoar formation.1,4 The most common pathway is from hematogenous spread of fungi to the kidney leading to formation of multifocal abscesses.1,5 Clinically, renal involvement in aspergillosis is commonly seen as fungal bezoars in the renal pelvis, producing hydronephrosis.6 Renal aspergillosis may cause renal failure, diffuse renal enlargement, unilateral or bilateral localized renal masses, or filling defect in the pyelocaliceal system with or without

**FIGURE 1.** A, Coronal contrast-enhanced computed tomography (CT) scan showing well-demarcated perfusion defect in the lower pole of the right kidney with perinephric fluid collection. On coronal image, mild dilatation of right renal pelvis (arrows) with slightly hyperdense internal lesion and delayed nephrogram are observed when compared with the left kidney. B, The inferior vena cava and right renal vein (arrow) are well delineated without intraluminal thrombosis. Further, visible right renal artery (arrowheads) is well delineated in axial contrast-enhanced CT scan. C, Follow-up coronal contrast-enhanced CT scan showing absent perfusion of the entire right kidney. Combined large hypodense mass with internal air bubbles and rim enhancement in perinephric space with extension to subcutaneous layer is observed. D, Microscopic examination of the kidney showing invasion of the renal vessels by branching septate hyphae of Aspergillus fumigatus.
obstruction. Nonspecific clinical features, such as fever and flank pain, are usual.

The CT findings of renal aspergillosis are nonspecific. Most of the cases demonstrated heterogeneous renal masses with strong enhancement on CT.4 Our case initially presented with a large area of hypoperfusion in renal parenchyma, suggesting renal infarction. There were no signs of fungal infection such as pyuria, elevated galactomannan level, and positive culture. However, galactomannan levels are not conclusive of invasive aspergillosis in the pediatric population and negative galactomannan assay may not reassure the physician against the use of amphotericin in pediatric patients with febrile neutropenia.8 Despite the empirical antibiotics and antifungal treatment, the patient’s severe abdominal pain only worsened. Thrombosis could be a possible complication during the course of acute leukemia, especially, treatment of acute lymphoblastic leukemia with L-asparaginase is well known to cause an impairment of anticoagulant and fibrinolytic mechanism, producing a prothrombotic state, leading to overt thrombosis in 2% to 10% of the patients.9 Moreover, thromboembolism is a well-recognized complication in malignancy. The pathogenesis of cancer-related prothrombotic state reflects the action of different mechanisms, including activation of blood coagulation by procoagulant substances, impairment of fibrinolytic pathway, and alterations of endothelium toward a thrombogenic state in patients with cancer.9 Rarely, inflammatory lesion involving the renal sinus and extension of renal inflammatory lesions into the renal sinus can cause vascular occlusion and multiple renal infarctions.10 With its inherent angioinvasive properties, the fungus can invade renal vasculature and produce renal infarcts.8 These hypotheses can explain our patient’s initial renal infarction caused by either the thrombotic mechanism or infective etiology.

According to the literature, fungus balls in the pelvis were seen in 12 of the 15 patients reviewed, and all these patients presented with fungal balls obstructing the upper urinary tract.6 Abdominal contrast-enhanced CT image of our patient revealed mild dilatation with internal slight hyperdense lesion in the right renal pelvis. On operative and pathologic findings, there was necrotic material in the right renal pelvis, which could have resulted in hydropnephrosis and delayed nephrogram of the right kidney. On retrospective review of the patient’s initial abdomen CT image, the linear hyperdense lesion in right psoas muscle and hydropnephrosis can be diagnostic clue in the first presentation of the renal infarction caused by infective etiology.

According to the data from the literature, there have been several cases of invasive aspergillosis presenting as infarction of brain and small bowel.11 There has been a case of renal mucormycosis in a patient with aplastic anemia who presented with renal infarction,12 but no previous case of invasive renal aspergillosis presenting initially with renal infarction has been reported.

In conclusion, invasive renal aspergillosis is a rare disease, which may unusually present as renal infarction, requiring high index of clinical suspicion. Awareness of this possibility and early attempts at diagnosis and treatment are important. This case emphasizes that renal aspergillosis should be included in the differential diagnosis of renal infarction in acute leukemia patients. Moreover, metastatic-embolic invasive aspergillosis may need to be considered in any focal lesion of the kidney occurring in a patient with leukemia.

REFERENCES