Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Radiology

Pediatric Pancreatic Mass Revealing a Pancreatoblastoma: A Case Report

M.A. Belhaj[1]*, N. Ait Said[1], S. Ouassil[1], C. Ahmanna[1], B. Zouita[1], D. Basraoui[1], H. Jalal[1]

[1]Department of Radiology, Arrazi Hospital, Mohammed VI University Hospital, Faculty of Medicine and Pharmacy, Cadi Ayyad University, Marrakech, Morocco

DOI: https://doi.org/10.36347/sjmcr.2026.v14i11.082 | **Received:** 22.09.2026 | **Accepted:** 17.11.2026 | **Published:** 21.11.2026

***Corresponding author:** M.A. Belhaj
Department of Radiology, Arrazi Hospital, Mohammed VI University Hospital, Faculty of Medicine and Pharmacy, Cadi Ayyad University, Marrakech, Morocco

Abstract **Case Report**

Pancreatic tumors in children are exceptional and are dominated by pancreatoblastoma. We report the case of a 6-year-old child admitted for deterioration of general condition evolving for one month. Abdominal ultrasound showed a heterogeneous hypervascular mass, associated with multinodular hepatomegaly and portal vein thrombosis. A complementary thoraco-abdomino-pelvic computed tomography (CT) scan confirmed the pancreatic origin of the mass and defined the locoregional and distant extension, revealing major locoregional invasion and hepatic metastases. The diagnosis of pancreatoblastoma was confirmed by histopathological examination and immunohistochemistry on a tumor biopsy. This case underlines the value of an integrated diagnostic approach, based on the complementarity of imaging and histological analysis, in order to rapidly guide therapeutic management.

Keywords: Pancreatoblastoma, pediatric pancreatic tumor, abdominal ultrasound, computed tomography, hepatic metastases.

Copyright © 2026 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution **4.0 International License (CC BY-NC 4.0)** which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

INTRODUCTION

Primary pancreatic neoplasms in children account for less than 1% of pediatric solid tumors [1]. Pancreatoblastoma is the most frequent malignant tumor of the pancreas during the first decade of life, with a peak incidence between 2 and 5 years of age [1,2].

The clinical presentation is often nonspecific, dominated by abdominal pain or deterioration of general condition, which may delay the diagnosis and lead to the discovery of large tumors at an advanced stage [2,3]. Abdominal ultrasound with color Doppler is the first-line initial examination, allowing characterization of the mass and assessment of its vascular relationships [3]. Thoraco-abdomino-pelvic computed tomography (CT) completes the work-up by defining the tumor origin, the anatomical relationships and the extension [4].

The radiological differential diagnosis notably includes solid pseudopapillary tumor of the pancreas, or Frantz tumor, whose features may overlap with those of pancreatoblastoma [2,5]. Histopathological examination, supplemented by immunohistochemistry, therefore remains indispensable for diagnostic confirmation.

In this article, we report a case of metastatic pancreatoblastoma in a 6-year-old child. We highlight the complementary contribution of Doppler ultrasound, CT and histopathological analysis in tumor characterization, assessment of extension and establishment of the definitive diagnosis.

CASE PRESENTATION

We report the case of a 6-year-old girl with no notable medical history, admitted for deterioration of general condition with asthenia, anorexia and weight loss evolving for one month, accompanied by a sensation of heaviness in the left hypochondrium. On clinical examination, the patient was hemodynamically stable, with jaundice. Abdominal examination found hepatomegaly and a palpable mass in the epigastrium and left hypochondrium.

The work-up began with an abdominal ultrasound, which revealed a large mass of approximately 9.1 × 7 cm, located in the upper left part of the abdomen (epigastrium, left hypochondrium and left flank). This mass displaced the neighboring organs (stomach and spleen) and showed vascular signals on

Doppler (Figure 1). The liver was enlarged, heterogeneous, with irregular contours, and riddled with multiple nodules vascularized on color Doppler (Figure 2). The portal trunk, of increased caliber, was obstructed by hyperechoic material extending to the bifurcation (Figure 3).

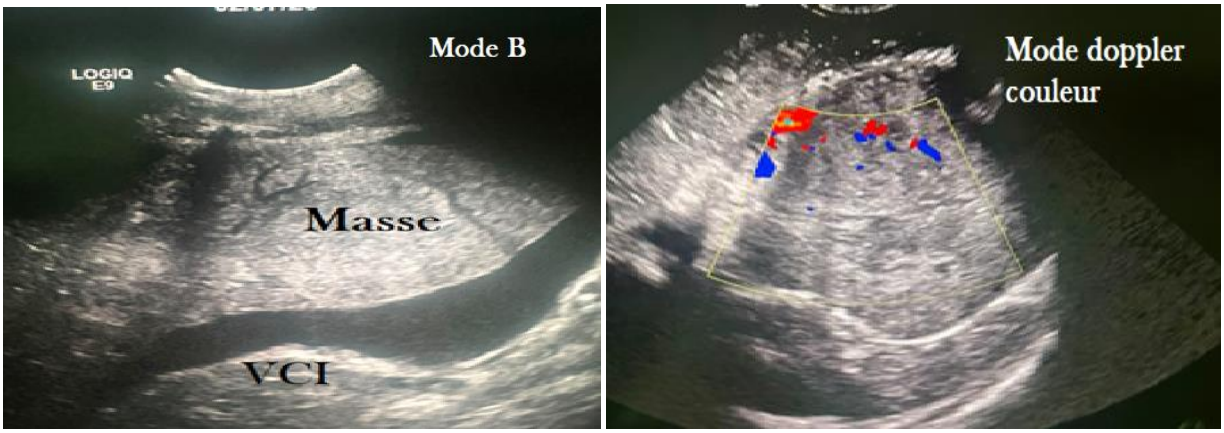


FIGURE 1: Epigastric mass extending to the left hypochondrium, vascularized on color Doppler, displacing neighboring structures, notably the inferior vena cava



FIGURE 2: Hepatic Doppler ultrasound showing multinodular hepatomegaly (nodules indicated by white arrows)



FIGURE 3: Obstructive hyperechoic thrombosis of the main portal trunk

To define the relationships of the mass and perform the extension work-up, a thoraco-abdomino-pelvic CT scan was performed without and after contrast medium injection. It showed a large retrogastric tumor process measuring 70 × 90 × 95 mm (AP × T × CC), forming a spur sign with the body and tail of the pancreas, which established its pancreatic origin (Figure 4). The mass was spontaneously heterodense, contained microcalcifications and showed heterogeneous enhancement delineating vast areas of necrosis (Figure 5).

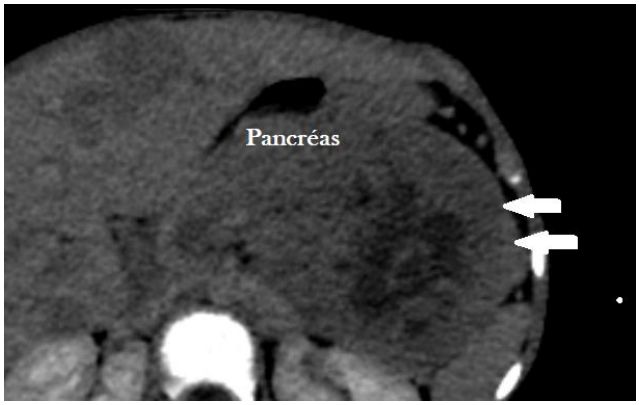


FIGURE 4: Pancreatic body–tail mass (indicated by white arrows)

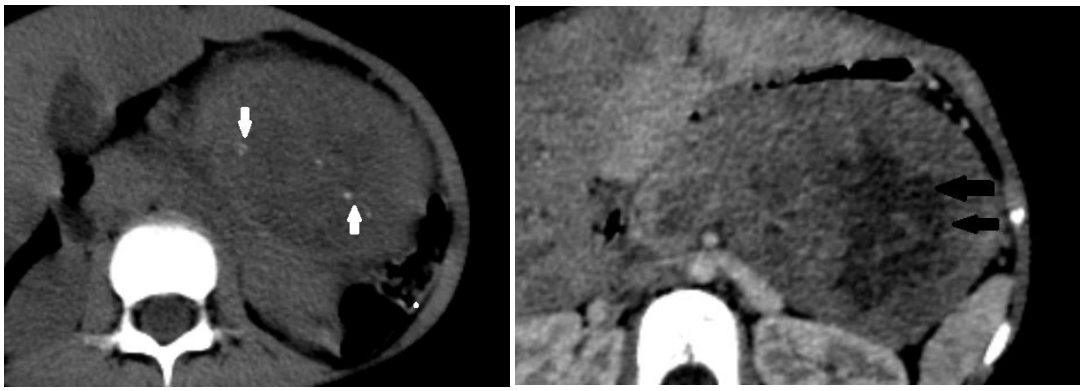


FIGURE 5: Heterogeneous pancreatic process containing calcifications (white arrows) and vast areas of necrosis (black arrows)

Regarding local and vascular extension, the mass had close relationships with the adjacent structures: it displaced the stomach anteriorly as well as the duodenal block and liver segments III and IV, and infiltrated the hepatic hilum (Figure 6). The portal vein was dilated, occupied by hypodense material consistent with portal thrombosis (Figure 7). Posteriorly, the mass was in close contact with the left kidney and its vascular pedicle, the spleen and the left colic flexure, with effacement of the separating fat planes (Figure 8).

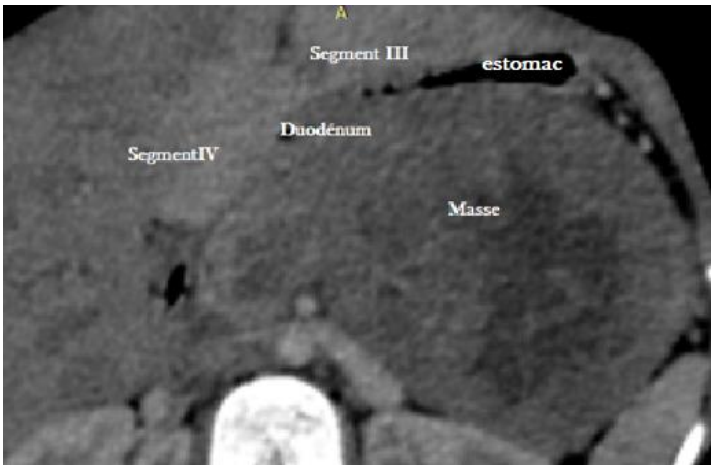


FIGURE 6: Pancreatic mass with mass effect on the adjacent digestive and hepatic structures and infiltration of the hepatic hilum

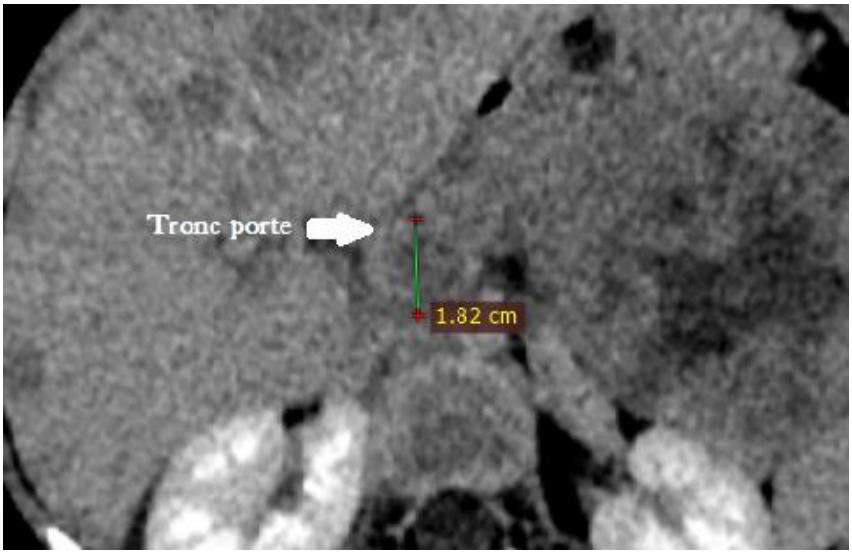


FIGURE 7: Portal vein thrombosis

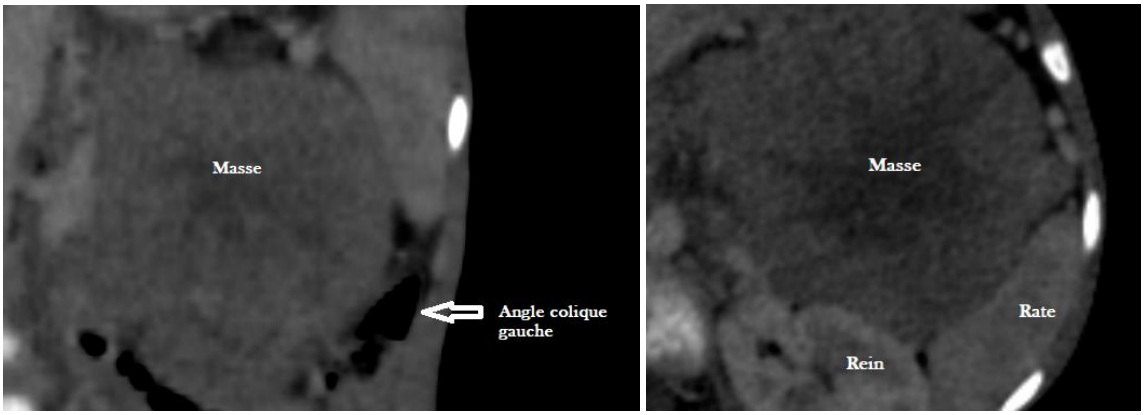


FIGURE 8: Posterior tumor extension in direct contact with the renosplenic block (A) and the left colic flexure (B)

The liver was enlarged (liver span 15 cm) and riddled with multiple bilobar metastatic masses and nodules, isodense, with slight peripheral enhancement (Figure 9). Marked peri-splenic and perigastric collateral venous circulation reflected segmental portal hypertension (Figure 10).

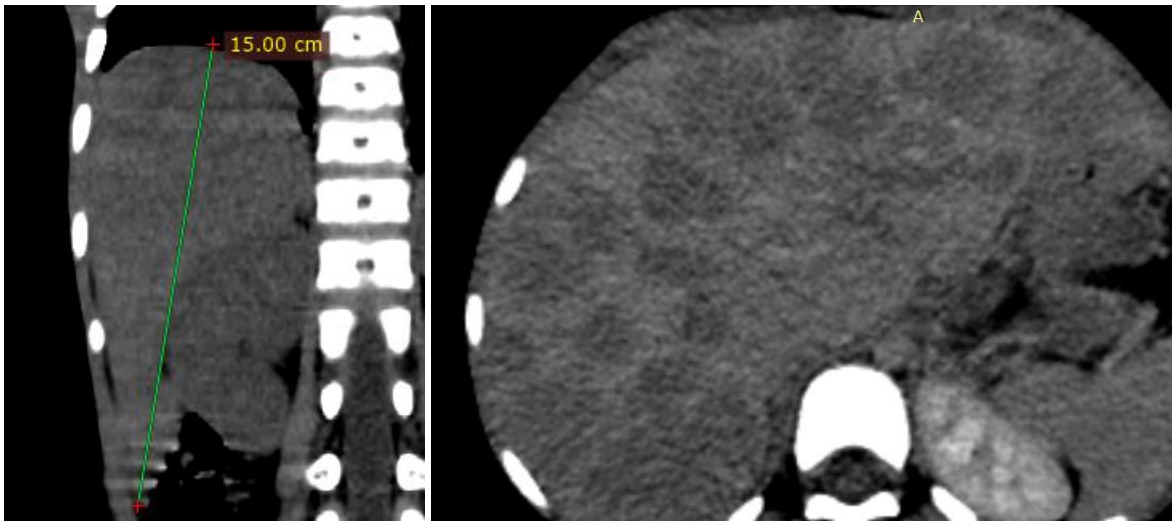


FIGURE 9: Hepatomegaly riddled with multiple bilobar metastatic masses and nodules showing slight peripheral enhancement

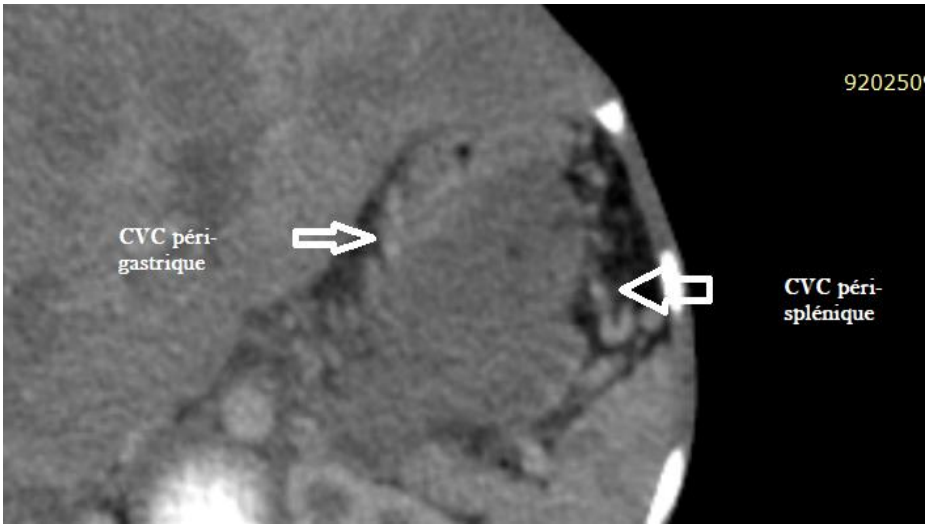


FIGURE 10: Marked perigastric and peri-splenic collateral venous bypass pathways

An ultrasound-guided percutaneous tumor biopsy was performed to establish the histological diagnosis. Histopathological examination revealed a malignant tumor proliferation with a lobulated architecture, characterized by squamoid corpuscles suggestive of pancreatoblastoma. Immunohistochemical study showed positivity for pancytokeratin and beta-catenin, as well as focal expression of synaptophysin and chromogranin A. Together, these results confirmed the diagnosis of pancreatoblastoma.

DISCUSSION

Pancreatoblastoma is an extremely rare malignant embryonal epithelial tumor. Although it is one of the main primary malignant tumors of the pancreas in young children, its low incidence and often nonspecific clinical presentation may delay the diagnosis. It generally presents as a large abdominal mass, sometimes associated with abdominal pain, deterioration of general condition or compressive signs. In our case, the presentation as a compressive abdominal mass initially suggesting gastric disease illustrates the diagnostic difficulties related to this tumor [1,2].

Radiologically, abdominal ultrasound is the first-line examination in children, because it is non-irradiating, widely available and able to explore the abdominal organs and their anatomical relationships. Pancreatoblastoma usually appears as a large, well-defined or irregularly contoured mass of heterogeneous echotexture, combining solid components and cystic or necrotic areas. Calcifications may also be visualized as hyperechoic foci with posterior acoustic shadowing. Doppler study clarifies the relationships of the mass with the vascular structures and looks for possible circulatory abnormalities, while hepatic exploration contributes to the detection of secondary lesions. In our case, ultrasound allowed characterization of the mass, assessment of its vascular impact and identification of hepatic lesions suspicious for metastases [3].

Multidetector abdominal CT is complementary, defining the tumor origin, characterizing the different components of the mass and mapping its locoregional and distant extension. Pancreatoblastoma classically presents as a large heterogeneous mass, with solid components that enhance after contrast injection, areas of necrosis and, sometimes, calcifications. Analysis of anatomical relationships is essential to assess extension to adjacent organs and possible vascular involvement. In this context, the pancreatic spur sign, corresponding to the persistence of normal pancreatic tissue stretched around the mass, can help identify its pancreatic origin, notably when it is initially mistaken for a gastric tumor [4,5]. In our case, CT revealed the calcifications and the vast areas of necrosis.

The main radiological differential diagnosis in children is solid pseudopapillary tumor of the pancreas, also called Frantz tumor (Table 1). Both entities may present as a large heterogeneous mass with solid components, hemorrhagic or necrotic changes and calcifications. However, certain CT features help distinguish them. Pancreatoblastoma may show infiltrative contours, locoregional extension, vascular involvement and synchronous hepatic metastases. Conversely, Frantz tumor is generally well circumscribed, encapsulated and pushing, with a fibrous capsule often visible and hemorrhagic or necrotic changes; vascular invasion or metastases at diagnosis are less usual. In our case, extension to adjacent structures, portal thrombosis, calcifications and secondary hepatic lesions were elements in favor of pancreatoblastoma, although they could not by themselves establish the definitive diagnosis [2,5,6].

Table 1: Comparison of the main pancreatic tumors of childhood

Characteristic	Pancreatoblastoma	Solid pseudopapillary tumor (Frantz)	Neuroendocrine tumor
Typical age	Young child	Adolescent, predominantly female	Variable
Morphological appearance	Large, heterogeneous, sometimes lobulated mass	Well-circumscribed, often encapsulated mass	Often well limited
Internal components	Solid, necrotic, sometimes calcified	Solid, cystic, hemorrhagic and necrotic	Generally solid
CT enhancement	Heterogeneous	Variable, capsule sometimes visible	Often intense if well differentiated
Calcifications	Possible, sometimes central	Possible, sometimes peripheral	Possible, less characteristic
Vascular extension	Possible	Uncommon	Variable depending on grade
Hepatic metastases	Possible, sometimes synchronous	Rare at diagnosis	Possible depending on type and grade
Biological marker	AFP often elevated	No usual specific serum marker	Hormonal markers depending on secretion
Diagnostic confirmation	Histology with characteristic squamoid corpuscles	Pseudopapillary architecture and immunohistochemistry	Histology and neuroendocrine markers

Biology can also contribute to diagnostic orientation. Elevation of serum alpha-fetoprotein (AFP) is frequently reported in pancreatoblastoma and is a useful marker for initial diagnosis and treatment follow-up. However, because its sensitivity is not absolute, a normal level does not exclude this tumor. Its interpretation must also take into account the age of the child and the clinical context [1,6].

Histopathological analysis is the decisive step of diagnostic confirmation. Pancreatoblastoma generally shows a lobulated architecture, composed of epithelial cells with predominantly acinar differentiation, associated with variable degrees of ductal, endocrine or mesenchymal differentiation. The presence of squamoid corpuscles, characteristic of this tumor, is a major histological element that distinguishes it from other pancreatic neoplasms, notably solid pseudopapillary tumor and neuroendocrine tumors. Immunohistochemical study complements the morphological analysis and helps refine the diagnosis [1,6].

In our case, the locoregional extension of the mass, characterized by effacement of the separating fat planes with the adjacent organs, notably the duodenum, the left kidney and the hepatic hilum, associated with portal thrombosis and synchronous hepatic metastases, indicates advanced disease. These features make upfront complete surgical resection difficult or even impossible, and justify discussion at a multidisciplinary team meeting. For locally advanced or metastatic forms, the therapeutic strategy generally relies on neoadjuvant chemotherapy based on cisplatin and doxorubicin, according to the PLADO protocol. The objective is to obtain tumor shrinkage and control of metastatic disease, so as to allow, when it becomes possible, secondary complete surgical resection. Complete surgery indeed remains an essential element of curative treatment [1,4,6].

The prognosis of pancreatoblastoma depends mainly on the possibility of achieving complete resection, the presence of metastases at diagnosis, locoregional extension and response to chemotherapy. Despite its malignant nature, this tumor can be associated with prolonged survival when treatment achieves durable tumor control and complete resection. In contrast, metastatic or unresectable forms pose greater therapeutic difficulties and carry an increased risk of persistent or recurrent tumor. AFP can also contribute to follow-up, its decrease under treatment possibly reflecting a therapeutic response. In our case, the presence of synchronous hepatic metastases and vascular extension are factors of severity, underlining the importance of early multidisciplinary management and regular reassessment of resectability [1,6].

CONCLUSION

Pancreatoblastoma is a rare but highly aggressive pediatric tumor. This clinical case illustrates the indispensable synergy between Doppler ultrasound (first-line examination), CT (extension and resectability assessment) and histopathological analysis (histological certainty). In the presence of a large solid abdominal mass in a child, prompt consideration of this diagnosis should lead to early biopsy in order to start neoadjuvant chemotherapy, an essential step before considering secondary curative surgical resection.

ADDITIONAL INFORMATION

Conflict of interest: The authors declare that they have no conflicts of interest.

REFERENCES

1. Chung EM, *et al.,* From the archives of the AFIP: pancreatic tumors of childhood: radiologic-pathologic correlation. *Radiographics*. 2006; 26:1211-38.
2. Perez EA, *et al.,* Solid pseudopapillary tumor of the pancreas in children: a single-institution experience and review of the literature. *J Pediatr Surg*. 2009; 44:1314-9.
3. Montagne JP, *et al.,* Pancreatoblastoma: the CT and US findings in four cases. *Pediatr Radiol*. 1990; 20:187-9.
4. Restrepo R, *et al.,* Primary pancreatic tumors in children: radiologic-pathologic correlation. *Eur J Radiol*. 2011; 80:494-501.
5. Buetow PC, *et al.,* Solid and papillary epithelial tumor of the pancreas: imaging-pathologic correlation in 56 cases. *Radiology*. 1996; 199:707-11.
6. Bien E, *et al.,* Pancreatoblastoma: a report from the European Cooperative Study Group for Pediatric Rare Tumors (EXPERT). *Eur J Cancer*. 2011; 47:2347-52.