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Current State of Imaging of Pediatric Pancreatitis: *AJR* Expert Panel Narrative Review

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Abstract

Pancreatitis is as common in children as it is in adults, though causes and accepted imaging strategies differ in children. In this narrative review we discuss the epidemiology of childhood pancreatitis and key imaging features for pediatric acute, acute recurrent, and chronic pancreatitis. We rely heavily on our collective experience in discussing advantages and disadvantages of different imaging modalities; practical tips for optimization of ultrasound, CT, and MRI with MRCP in children; and image interpretation pearls. Challenges and considerations unique to imaging pediatric pancreatitis are discussed, including timing of imaging, role of secretin-enhanced MRCP, utility of urgent MRI, severity prediction, autoimmune pancreatitis, and best methods for serial imaging. We suggest a methodical approach to pancreatic MRI interpretation

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in children and have included a sample structured report, and we provide consensus statements according to our experience imaging children with pancreatitis.

Keywords

CT; MRI; pancreas; pancreatitis; ultrasound

Acute pancreatitis (AP) and chronic pancreatitis (CP) are now believed to have similar incidence in children as in adults [1]. However, the causes of disease differ. Alcohol and smoking are significantly associated with adult disease but are uncommon in children. Genetics (e.g., *CFTR* and *PRSS1* mutations, among others) are a major driver in children, particularly of acute recurrent pancreatitis (ARP) and CP [2]. Unique to pediatrics is the importance of nonaccidental trauma, which must be considered in infants and young children who present with pancreatitis [3]. With direct trauma, the pancreas is compressed against the spine, potentially causing contusion, laceration, or transection [4] (Fig. 1). Obstructive causes (e.g., biliary stones) are significant in both children and adults.

Pancreatitis can be debilitating and have significant morbidity in childhood. Endocrine and/or exocrine insufficiency associated with CP can cause deficits in growth and development. Pancreatitis and its complications can cause significant life disruption and high health care utilization and costs [1, 5]. Thus, early and accurate diagnosis of pancreatitis is critical.

The International Study Group of Pediatric Pancreatitis: In Search for a Cure (INSPPIRE) [6] and other collaborative groups define diagnostic criteria for AP, ARP, and CP (Table 1). If a child presents with characteristic symptoms and elevated laboratory markers, imaging is not required to diagnose AP. However, imaging may be important in establishing a diagnosis in a child with vague symptoms or insufficiently elevated serum enzymes. Imaging also helps identify obstructive causes (e.g., stones), assess AP complications, and plan invasive interventions. For ARP, imaging helps confirm attacks of AP, identify and monitor complications, and evaluate for progression to CP. Imaging has a central role in CP diagnosis, assessment of CP-related complications, monitoring for disease progression, and planning invasive interventions and surgery [7].

This review discusses imaging of pediatric pancreatitis. Given a paucity of evidence in children, clinical practice is heavily influenced by consensus guidelines and individual experience.

Modalities for Imaging Pediatric Pancreatitis

Ultrasound

Consensus guidelines consider transabdominal ultrasound the initial imaging modality of choice in children with suspected AP [7]. This consensus reflects ultrasound's high sensitivity for gallstones and biliary obstruction, the modality's availability, and likely overemphasis on lack of ionizing radiation exposure. This consensus belies the moderate

sensitivity and specificity of ultrasound for AP, with recent studies suggesting 47–52% sensitivity [8, 9].

When performing ultrasound of the pancreas, careful technique, including varying transducer compression to displace bowel gas, specific patient breathing instructions, and altering patient positioning, can optimize pancreatic visualization [10, 11]. Fasting (4–6 hours) can diminish bowel gas and distend the gallbladder, oral fluid administration immediately before the examination can improve the acoustic window, and prone or decubitus imaging can facilitate visualization of the pancreatic body and tail using the left kidney or spleen as an acoustic window (Fig. 2).

CT

CT offers rapid imaging, making it the test of choice for unstable patients. CT is more sensitive than ultrasound for AP and provides a large FOV to evaluate the full extent of pancreatitis-associated collections [8]. CT is also the test of choice for children with air-filled distended bowel loops or large body habitus—both cases for which ultrasound is limited.

The primary disadvantage of CT is limited biliary assessment compared with ultrasound and MRI. CT also uses ionizing radiation, which likely contributes to avoidance of the modality despite the continued decrease in radiation dose with advancing CT scanner technology. In the current era, the benefits of clinically indicated CT for pancreatitis outweigh the small theoretic risks of radiation exposure.

For initial evaluation of AP or CP, abdominopelvic CT is recommended to ensure complete coverage of complications [7]. Limited CT can be considered for focused follow-up. Sedation should be reserved for patients who cannot follow instructions (e.g., very young or developmentally delayed) [12].

Iodine-based IV contrast media improve visualization of peripancreatic tissues and vasculature [13]. At the authors' institutions, imaging commences immediately after complete injection of the contrast media volume or at a 60-second delay. Unenhanced CT is rarely indicated in pediatric pancreatitis, particularly given the low frequency of calcifications and hemorrhage [14]. Although multi-phase CT has utility in assessment of pancreatic neoplasms and autoimmune pancreatitis (AIP) in adult patients, it is discouraged in children because of dose considerations [15]. Exceptions include the need for clear delineation of vasculature or suspected pancreatic mass. Dual-energy CT, although not specifically studied in pediatric pancreatitis, may provide additional information through virtual unenhanced imaging and iodine mapping.

No robust data exist to guide use of enteric contrast media for CT of pediatric pancreatitis. In the authors' experience, enteric contrast medium (positive contrast media or water) is generally not needed for diagnosis.

MRI

MRI with MRCP is the optimal imaging modality for evaluation of ARP and CP given superior resolution of the pancreatic parenchyma and duct. Imaging after 4–6 hours of fasting reduces enteric content, minimizes peristalsis and artifacts, and helps distend the gallbladder and bile ducts. Negative oral contrast agents, such as ferumoxsil, ferric ammonium citrate, and various juices, have variable success in minimizing fluid signal in the stomach and duodenum but are infrequently used by the authors for logistical reasons [16].

Table 2 summarizes recommended sequences for evaluation of pediatric pancreatitis. In children, thick-slab (2–4 cm) MRCP provides superior visibility of the pancreatic duct compared with thin-slice 3D images [17]. MRCP quality directly relates to respiratory triggering or gating, and optimal imaging requires regular respirations. In the authors' experience, at least one backup duct-focused sequence such as a thin-slice respiratory-triggered T2-weighted SSFSE sequence should be performed in addition to dedicated MRCP sequence(s) to characterize duct anatomy (Table 2).

Both 1.5-T and 3-T MRI adequately image the pancreas and pancreaticobiliary ducts. 3-T MRI inherently provides more signal, theoretically improving visibility of small ducts. However, some artifacts, particularly dielectric effect, are typically worse at 3 T [18]. Radiofrequency coil selection is an important determinant of signal-to-noise ratio and impacts image quality. Depending on the child's size, phased-array cardiac or torso coils work well for pancreas imaging. Coverage should extend from diaphragm to lower poles of kidneys. Additional limited pelvic imaging should be considered to assess for fluid and collections.

Gadolinium-based IV contrast media are not required for pancreatic MRI. Gadolinium-based contrast media can help identify necrosis before liquefaction occurs, evaluate peripancreatic enhancement in AIP, and characterize pancreatic masses. It also helps assess pancreatitis complications including degree of organization of collections and vascular complications. Extracellular contrast media are generally used. Hepatocyte-specific contrast media (gadotetate disodium, Primovist or Eovist, Bayer Health-Care) do not opacify the pancreatic duct and are reserved for situations requiring bile duct assessment such as type III choledochal cyst or biliary stenosis in the pancreatic head (characteristic of fibrosing pancreatitis) [19].

Use of human secretin for MRI can improve visualization of the nondilated pancreatic duct and its junction with the common bile duct and increase sensitivity for pancreas divisum [20] (Fig. 3). This is achieved by stimulating pancreatic bicarbonate secretion. In the context of a dilated duct, however, the diagnostic contribution of secretin is uncertain [21]. Secretin is not necessary for all pediatric pancreas MRI examinations but should be used for first evaluation in all patients with ARP or CP or suspected anomalous pancreaticobiliary junction (APBJ). In addition to increasing conspicuity of a nondilated pancreatic duct, secretin can characterize pancreatic exocrine function, which is relevant to patient symptoms and the diagnosis of CP [22]. Dosing is the same as in adults: 0.2 µg/kg to a maximum of 16 µg. Human secretin has no contraindication other than secretin allergy.

The Normal Pancreas

Ultrasound

In neonates, the pancreas is homogeneously hyperechoic relative to liver on gray-scale ultrasound. Throughout the rest of childhood and adolescence, the pancreas appears isoechoic to slightly hyperechoic to liver [23]. The normal pancreas should be closely approximated to adjacent structures without thick intervening echogenic fat (Fig. 2). The normal pancreatic duct is usually imperceptible except when using a high-frequency (> 12 MHz) transducer.

CT

In the portal venous phase after IV contrast administration, the normal pancreas enhances homogeneously, similar to slightly less than normal liver and spleen. The size and shape of the pancreas vary by age and weight, with recent data defining normal pancreatic parenchymal thickness measurements in children [24]. The pancreatic duct is infrequently visible by CT except when abnormal.

MRI

The normal pancreas is uniformly high in signal on T1-weighted MRI because of high protein content [25] (Fig. 4). T1-weighted signal intensity should be greater than all other organs in the abdomen, particularly on fat-saturated imaging. After IV contrast administration, the normal pancreas shows homogeneous enhancement in the late arterial phase and is higher in signal than liver and adjacent bowel. On delayed contrast-enhanced phases, normal pancreatic parenchyma is isointense to the liver. On T2-weighted MRI, normal pancreatic parenchyma is isointense to the liver (Fig. 4). A normal pancreatic duct can be seen on T2-weighted images in children but is uncommonly visible along its entire length, and no side branches should be visible [26]. The pancreaticobiliary junction is variably visible and should never be visible above the level of the ampulla.

Imaging Findings of Acute Pancreatitis

Imaging classification of AP is specifically defined in adults by the revised Atlanta classification as either interstitial edematous pancreatitis, the most common form representing pancreatic inflammation without necrosis, or necrotizing pancreatitis, which is far less common (5–10% of adult cases) and characterized by parenchymal and/or peripancreatic necrosis [27]. Necrotizing pancreatitis is subclassified as pancreatic or peripancreatic by location of necrosis. Mixed pancreatic and peripancreatic necrosis is the most common form of necrotizing pancreatitis in adults, occurring in approximately 75% of cases; however, no similar pediatric data exist [28]. On imaging, peripancreatic necrosis is characterized by heterogeneous debris or semisolid or solid tissue within a peripancreatic collection. Although the revised Atlanta classification was not developed for pediatrics, it provides a useful structure for standardized description of AP imaging findings, and we recommend its routine use in children.

Ultrasound

Variability in size and echogenicity of the acutely inflamed pancreas, combined with the pancreas commonly being incompletely visible by ultrasound, complicates diagnosis and contributes to only moderate sensitivity for AP in children. Part or all of the pancreas is reported to be obscured on ultrasound in up to 14% of cases [29]. In the authors' experience, the frequency of incomplete visualization is more common than this figure.

Ultrasound findings of AP include focal or diffuse pancreatic enlargement with increased (more commonly than decreased) parenchymal echogenicity (Fig. 5). More commonly, the pancreas may appear normal despite known disease [13]. Pancreatic duct dilatation has been cited as the most useful ultrasound finding of AP; however, in the authors' experience, it is uncommonly present and is unreliable [23, 30]. Reported thresholds for dilatation are greater than 1.5 mm for children 1–6 years old; greater than 1.9 mm for 7–12 years old, and greater than 2.2 mm for 13–18 years old [30]. Peripancreatic fluid and/or echogenic thickened fat may be present.

CT

CT findings of AP include pancreas enlargement, parenchymal heterogeneity, peripancreatic edema or stranding, and peripancreatic fluid accumulation (Fig. 5). Necrosis manifests as areas of parenchymal nonenhancement that become more conspicuous with time (Fig. 6). Although uncommon, high-attenuation material may reflect hemorrhage.

MRI

Although MRI is not currently considered a first-line imaging modality for AP in children, MRI maximizes sensitivity for interstitial edema and provides detailed assessment for structural and biliary causes. MRI findings of AP include pancreas enlargement (swelling), interstitial edema, and peripancreatic fluid, which are most conspicuous on fluid-sensitive (T2-weighted) sequences [13] (Fig. 5). On T1-weighted imaging, inflamed portions of the pancreas show loss of normal high signal resulting from interstitial edema. This can confound use of this finding as an indicator of CP. Contrast-enhanced parenchymal enhancement should be preserved but may be diminished in areas of interstitial edema, which can be misinterpreted to represent necrosis [28]. MRI findings of necrosis include absent parenchymal enhancement with or without peripancreatic necrosis. Intraparenchymal hemorrhage may manifest as high signal on T1-weighted imaging [13].

Acute Pancreatitis–Associated Fluid Collections

The revised Atlanta classification affirmed specific definitions for pancreatitis-associated fluid collections, differentiating these collections according to pancreatitis type, collection appearance and location, and disease time course [27, 28] (Table 3). Again, although not developed for children, accurate description and characterization of collections according to these criteria are important to guide management and is recommended.

On imaging, acute peripancreatic fluid collections are peripancreatic in location and conform to the adjacent spaces without a defined wall, often tracking along the anterior pararenal space [28] (Fig. 7). Collections exhibit homogeneous attenuation, signal, and

echogenicity, without semisolid or solid internal debris. Ultrasound and MRI are superior to CT in characterizing content of pancreatitis-associated fluid collections. Once the collection develops a well-defined enhancing wall, generally after 4 weeks, it is termed a pseudocyst (Fig. 7). The authors have rarely encountered true pseudocysts in practice. The literature is weak but suggests pseudocysts are less common in children than adults and are reported in up to 10% of interstitial edematous pancreatitis cases, with the most acute peripancreatic fluid collections resolving spontaneously [28, 31]. Collections within the pancreas, even if simple, should not be classified as acute peripancreatic fluid collections or pseudocysts because replacement of parenchyma indicates necrosis [27].

Acute necrotic collections are differentiated by presence of necrotic material and can be transspatial. Acute necrotic collections can appear loculated and may involve pancreatic or peripancreatic tissues (Fig. 6). On imaging, acute necrotic collections may contain fluid or semisolid or solid debris (typically irregular in shape, high attenuation on CT, and low signal on T2-weighted MRI, without enhancement), or fat globules [28]. Acute necrotic collections that develop a well-defined enhancing wall, generally after 4 weeks, represent walled-off necrosis (Fig. 6). Walled-off necrosis is often peripancreatic, but can be parenchymal and can communicate with peripancreatic spaces [28].

Necrotic collections are more likely to become infected [27, 28]. Infection of a fluid collection is a clinical diagnosis. On imaging, gas within a collection can suggest infection but this is neither sensitive nor specific [27].

Peripancreatic collections require intervention only if symptomatic. Collections are managed by surgery or endoscopic or percutaneous drainage [32]. Surgical approaches include creation of cystogastrostomy or Roux-en-Y cystojejunostomy. Endoscopic drainage typically involves placement of one or more plastic or metal stents via transmural or transpapillary approach. Large-bore lumen-apposing metal stents are increasingly used, allowing endoscopic necrosectomy and débridement [33] (Fig. 6).

Severity Prediction

Predicting outcomes in severe pediatric AP is difficult because adult scoring systems such as Ranson, Glasgow, modified Glasgow, Bedside Index for Severity in Acute Pancreatitis, and Acute Physiology and Chronic Health Evaluation II are challenging to apply in children [34]. It is more useful to accurately report imaging findings and complications in children. Identifying local complications contributes to severity scoring per criteria defined by the North American Society for Pediatric Gastroenterology, Hepatology & Nutrition (NASPGHAN) Pancreas Committee [35].

The severity scoring system most explored in pediatrics is the CT severity index (CTSI) which incorporates the Balthazar score. In adults, CTSI is superior to clinical scoring systems in predicting outcome [13]. In children, Lautz et al. [36] reported CTSI to be better than clinical scoring systems for risk stratifying patients and guiding management. Nonetheless, no consensus exists on use of CT for routine staging of pediatric AP, and CTSI is not routinely used at the authors' institutions.

Autoimmune Pancreatitis

AIP is poorly understood in children, in part because of poor reference standards for diagnosis. Unlike in adults, IgG4 levels are rarely elevated in pediatric AIP [37].

AIP can present as focal, segmental, or global enlargement of the parenchyma or can have a masslike appearance. The characteristic sausage-like global pancreatic enlargement with a halo of surrounding enhancement is variably seen (Fig. 8). Dilation of the biliary duct or both the biliary and pancreatic ducts in the absence of mass suggests AIP. AIP may respond dramatically to steroid therapy.

Clinical history of other autoimmune diseases (e.g., inflammatory bowel disease) may suggest AIP; however, this relationship is complicated by higher incidence of drug-induced pancreatitis in children.

Imaging Findings of Acute Recurrent Pancreatitis

In children with ARP, imaging can document serial attacks of AP and provide information about the attack severity, complications, and progression to CP. Along with genetic testing, imaging plays an important role in workup, specifically to identify anatomic or autoimmune causes.

Imaging Findings of Chronic Pancreatitis

CP results from progressive inflammation leading to loss of acinar cells and pancreatic parenchymal fibrosis, with eventual exocrine and/or endocrine dysfunction. Imaging features of CP are well described in adults and extrapolated to pediatrics [38]. These findings include fibrotic parenchymal changes (atrophy, increased contour lobulation, MRI signal intensity alteration(s), decreased enhancement), calcifications, and duct changes (main and side branch dilation, main duct irregularity and strictures, periductal fibrosis, duct debris and/or calcifications) (Fig. 9). Imaging findings of CP specific to pediatrics are undefined. Notably, pancreatic calcifications, cysts, pseudocysts, and endocrine insufficiency are less frequent in children than adults [14]. Pancreatic parenchymal atrophy was previously deemed more common in children; however, a recent comparison showed similar frequencies [14].

Atrophy

Adult CP reporting guidelines recommend reporting pancreatic head, body, and tail thickness measurements, with threshold values to identify degrees of atrophy [39]. Importantly, these guidelines recommend exclusion of any dilated pancreatic duct from thickness measurements [39]. No similar threshold recommendations exist for children. Normative values exist for pancreas thickness in children on ultrasound and CT [23, 24].

Size quantification and detection of atrophy on the basis of linear measurements is complicated by normal growth and variable pancreatic shape [40]. Volumetric measurements can overcome this limitation but currently require manual postprocessing. Studies with small numbers of children describe a linear increase of pancreatic parenchymal volume and fat

content on CT with age [40]. A similar association has been shown between pancreatic volume and age, height, weight, and body surface area in healthy children on MRI [41].

Given the lack of consensus on the optimal means of assessing and defining atrophy, the authors currently provide a gestalt assessment of parenchymal bulk in clinical practice.

Duct Findings of Chronic Pancreatitis

Pancreas divisum and APBJ are risk factors for development of ARP and CP [42–46]. Pancreas divisum results in the bulk of the pancreas (body and tail) draining via the minor papilla (duct of Santorini) [44] (Fig. 10). Pancreas divisum has a frequency of less than 10% in the general population [47]. Most patients with pancreas divisum do not experience symptoms [44]. The sensitivity and specificity of diagnosing pancreas divisum with secretin-augmented MRCP in adults range from 73–100% and 97–100%, respectively [48].

APBJ, also called maljunction or long common channel, is defined as union of the pancreatic duct and common bile duct outside of the duodenal wall and above the sphincter of Oddi, allowing bile reflux into the pancreatic duct (predisposing to pancreatitis) [43]. Specific definitions for APBJ exist for adults according to common channel length (> 9 mm at MRCP) [49]. Suggested thresholds in children for ERCP are greater than 3 mm considered abnormal for children under 1 year old and greater than 5 mm considered abnormal for children until 15 years old [50]. No threshold values exist for MRCP diagnosis of APBJ in children. In the authors' experience, APBJ should be considered present anytime a common channel is visible by MRI [18] (Fig. 11).

Although set according to small samples, normative values exist for diameters of the common bile duct and main pancreatic duct at ultrasound and MRI for children [23, 24, 26, 30, 51]. Both ducts enlarge in diameter with age, with the normal pancreatic duct measuring between 0.6 and 1.9 mm in children and often not visible in very young children [26]. The authors rarely rely on measuring the duct, instead making a gestalt determination according to duct conspicuity and variation in duct caliber.

CP results in main pancreatic duct dilation compared with healthy individuals, but cutoff values do not currently exist to make a diagnosis of CP in children [30]. In our experience, a pancreatic duct that is easily visible along its entire length is generally abnormal.

T1-Weighted Signal Loss

T1-weighted signal loss can be a subtle finding of CP. This can be confounded in the context of superimposed in AP in which edema decreases parenchymal T1-weighted signal intensity. Normal liver and spleen can serve as internal controls for T1-weighted signal: if pancreatic signal is not greater than both organs, it is abnormal (Fig. 9). Literature on adults suggests semiquantitative identification of decreased T1-weighted signal via calculation of T1-weighted signal intensity ratios relative to spleen or skeletal muscle [52]. Specific signal intensity ratios have been shown to suggest exocrine insufficiency in adults but may not apply to children [52, 53].

T1-weighted relaxometry may ultimately provide a means of true quantitation of T1-weighted signal loss and has moderate sensitivity (77%) and specificity (83%) in adults with CP [54]. Pediatric normative relaxation times have recently been reported for a small cohort of healthy children at 1.5 T and 3 T [55].

Classification Systems

No standard imaging classification system currently exists for pediatric CP. In adults, the Cambridge classification based on ERCP pancreatic duct findings has been adapted for MRCP and is most commonly used [39]. The classification defines CP according to number of visualized abnormal side branches, cavities, filling defects, or duct obstruction [39]. This classification does not incorporate parenchymal changes of CP and is not validated in children [56, 57]. The M-ANNHEIM classification for adult CP relies partially on the Cambridge classification and other imaging findings on transabdominal ultrasound, CT, endoscopic ultrasound, or MRI, but also is not validated in children [58]. Recently, the Consortium for the Study of Chronic Pancreatitis, Diabetes, and Pancreatic Cancer proposed CT and MRCP reporting standards in adults with CP but these are not yet validated in adults and applicability to pediatrics is unknown [39]. Currently, none of the authors use a classification scheme in routine care, instead reporting findings and providing a gestalt interpretation of the presence of CP.

Functional Imaging

In addition to improving visualization of the nondilated pancreatic duct, secretin use can provide functional information. Adult data suggest that periductal fibrosis should be suspected if there is no duct caliber change after secretin administration. This finding is not well validated in children and, anecdotally, the authors have not found it to be particularly helpful. Adult data have shown that pancreatic exocrine function can be noninvasively assessed with ultrasound or MRI with secretin stimulation according to the volume of secreted fluid [59, 60]. Normative data exist for secreted fluid volume measured by MRI in children, but diagnostic cutoffs for detection of exocrine insufficiency are not defined [61, 62].

Special Imaging Considerations

Although consensus guidelines favor certain modalities, imaging strategies for pediatric pancreatitis should be tailored to each institution's capabilities. Considerations include modality availability, ability to perform CT with appropriate radiation dose, and ability to achieve adequate MRI quality.

Timing of Imaging

Studies in adults show that performing imaging within 48 hours of AP diagnosis does not alter management and poorly predicts severity of organ failure [27]. Similar data do not exist for children. In the acute setting, imaging may guide management particularly related to biliary stones and AP complications. Importantly, the presence of local complications such as peripancreatic fluid collections, necrotic fluid collections, pancreatic and peripancreatic necrosis, and development of pseudocysts or walled-off necrosis characterizes AP as

moderately severe according to the NASPGHAN Pancreas Committee [35]. However, assessment for anatomic pancreatic duct variants as a potential contributor to pancreatitis is optimal when AP has resolved because pancreatic and peripancreatic edema can cause duct compression, obscuring these variants (Fig. 10).

Urgent MRI

MRI with MRCP is uncommonly performed for AP in the acute setting, except when choledocholithiasis is suspected and ultrasound fails to identify an obstructing stone [63, 64]. One other potential indication for urgent MRI is assessment of duct transection, which may necessitate urgent ERCP with stenting. In the near future, rapid unenhanced MRI using limited sequences, similar to what is used for acute appendicitis, may play a role in AP diagnosis in the urgent setting, capitalizing on higher sensitivity than CT for edema and hemorrhage [65, 66]. AP diagnosis and identification of stones as a cause could be easily achieved with single-shot T2-weighted sequences with and without fat saturation.

Serial Imaging

Ultrasound, CT, or MRI can all be used to monitor complications and plan interventions. The optimal modality depends on the complication being followed, the location of the complication, and the clinical scenario. Overall, MRI is favored because of the large FOV and lack of ionizing radiation. Serial imaging also plays a role in monitoring for progression to CP.

Reporting Essentials

Table 4 highlights pearls and pitfalls of interpreting imaging for pancreatitis in children, according to the authors' clinical experience. Recognizing the imaging features of pancreatitis in a child is important, but communicating these findings in a methodical succinct manner that addresses referring clinicians' needs is equally important. In the authors' experience, this is best implemented through structured reporting. Appendix 1 outlines a suggested MRI reporting template.

Appendix

APPENDIX 1:

Reporting Template

Findings of Acute Pancreatitis: [absent / present]

Necrosis: [absent / present], location, % of pancreas (< 25%, 26–50%, > 50%, entire pancreas)

Fluid collection: [absent / present, size in three dimensions, location]

[acute / organized]

[simple / necrotic]

[communication with duct, yes / no]

Evidence of hemorrhage: [absent / present]

Evidence of infection: [absent / present]

Evidence of mass: [absent / present]

Findings of Chronic Pancreatitis: [absent / present]

Atrophy: [absent / present]

Contour irregularity: [absent / present]

T1-weighted signal: [normal / decreased]

Secretagogue response duct: [describe]

Secretagogue response exocrine function: [adequate / inadequate per Matos et al. [22]]

Pancreatic Duct:

Anatomy: [typical / variant, describe]

Pancreaticobiliary junction: [normal / long common channel]

Main duct dilation: [absent / present]

Main duct irregularity: [absent / present]

Main duct stricture: [absent / present, location]

Main duct filling defect(s): [absent / present]

Side branch dilation: [absent / < 3 mm / 3 mm]

Vasculature:

Hepatic arterial anatomy: [conventional / variant, describe]

Arterial pseudoaneurysm(s): [absent / present]

Splenic vein: [normal / narrowed / thrombosed]

Superior mesenteric vein: [normal / narrowed / thrombosed]

Portal vein: [normal / narrowed / thrombosed]

Venous collaterals: [absent / present, describe]

Biliary System:

Gallbladder: [absent / present]

Biliary stones/sludge: [absent / present, describe size and location]

Common bile duct: [normal / abnormal, describe]

Common bile duct diameter: [] mm

Other Findings: []**Impression:**

1. Imaging findings of acute pancreatitis: [absent / present, describe (including description of local complications and fluid collections)]
2. Imaging findings of chronic pancreatitis: [absent / present]
3. Complications of pancreatitis: [absent / present, describe]
4. Pancreatic duct anatomy: [conventional or variant, describe]
5. Biliary pathology: [absent / present, describe]

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Consensus Statements

- Imaging contributes to diagnosis of AP in clinically equivocal cases, contributes to identification of causes, and is necessary for identification of local complications.
- Ultrasound has high sensitivity for gallstones and should be used at the time of a first attack of pancreatitis to exclude gallstones or choledocholithiasis.
- Ultrasound has moderate sensitivity for AP. An ultrasound without findings of AP does not exclude AP.
- Contrast-enhanced CT is an acceptable first-line imaging modality for AP, particularly given the moderate sensitivity of ultrasound. Unenhanced CT is generally not indicated.
- The benefits of clinically indicated CT performed with optimal technique outweigh the small theoretic risks of radiation exposure.
- Nonaccidental trauma must be considered a cause of pancreatitis in infants and young children.
- The revised Atlanta criteria [27] should be routinely used to describe imaging findings of pediatric AP until a pediatric-specific system is developed.
- Care must be taken to correctly characterize organized fluid collections because classification has management implications. Misclassification of walled-off necrosis as pseudocyst is a common error.
- Given lack of consensus on imaging-based systems to score AP severity in children, accurate reporting of imaging findings and complications should be emphasized to guide management and clinical severity staging.
- MRI with MRCP is the optimal imaging modality for evaluation of ARP and CP and is the optimal modality to monitor complications and plan interventions.
- MRI to assess for anatomic variants of the pancreatic duct as a potential contributor to pancreatitis and to assess for CP features should be deferred until a quiescent period (absence of AP) to avoid obscuring findings by acute inflammation.
- Gadolinium-based IV contrast media are not required for pancreatic MRI but can help identify and characterize necrosis, evaluate peripancreatic enhancement in the context of AIP, and characterize pancreatic masses.
- Secretin should be used for the first MRI in patients with ARP, CP, or suspected APBJ.
- A pancreaticobiliary junction visible above the ampulla by MRI is abnormal and suggests APBJ.

- A normal pancreatic duct is rarely visible along its entire length. A fully visible duct is abnormal. Side branch ducts should never be visible in a normal pancreas.
- Specific imaging findings of CP are undefined for children. Characteristic findings in adults of pancreatic calcifications, cysts, and endocrine insufficiency are less frequent in children.

Fig. 1—

4-month-old boy with metabolic disorder with acute presentation for vomiting, irritability, and fever, determined to be victim of abuse.

A, Axial CT through upper abdomen with IV and oral contrast material shows pancreatic transection (*white arrow*) and acute fluid (*black arrow*).

B, Coronal reformatted CT of abdomen and pelvis shows pancreatic transection (*white arrow*) and fluid extending into pelvis (*black arrow*).

Fig. 2—
11-year-old boy with vomiting. Transverse ultrasound image using spleen (*asterisk*) as acoustic window clearly shows normal tail of pancreas (PANC TAIL). Pancreatic echogenicity is normal, and gently lobulated surface contour of pancreas is normal. Surrounding fat planes are thin and well defined.

Fig. 3—

15-year-old boy with history of acute pancreatitis.

A, Coronal thick-slab (6-cm) fat-saturated T2-weighted MRI before secretin administration shows normal-caliber biliary tree and fluid in stomach (*asterisk*) and proximal jejunum. Pancreatic duct is not visible.

B, Identical coronal thick-slab image after secretin administration clearly shows pancreatic duct (*arrow*) extending toward major papilla. Response to secretin administration is qualitatively normal with filling of duodenum beyond genu inferius (*arrowhead*) and with clear accumulation of fluid in left upper quadrant jejunal loops.

Fig. 4—.

17-year-old girl with right upper quadrant pain.

A, Axial T2-weighted fat-saturated MRI shows normal pancreas (*arrow*) with signal intensity nearly identical to normal liver.

B, Axial T1-weighted fat-saturated 3D gradient-recalled echo MRI shows normal pancreas (*arrow*) with highest signal of all visible solid organs.

C and **D**, Coronal fat-saturated T2-weighted SSFSE sequence before (**C**) and 15 minutes after (**D**) secretin administration shows filling of duodenum (*arrow*) and distention of proximal jejunal loops with fluid, reflecting adequate response. Stomach (*asterisk*) also distends with fluid.

Fig. 5—

13-year-old girl with interstitial edematous acute pancreatitis.

A, Transverse transabdominal ultrasound performed at diagnosis shows enlarged or thickened pancreas (*arrow*) with thickening and increased echogenicity of peripancreatic fat (*arrowheads*).

B, Axial contrast-enhanced CT performed on same day shows pancreas to be diffusely swollen and hypoenhancing compared with liver and spleen. Peripancreatic fat planes are poorly defined (*arrowheads*) due to peripancreatic edema.

C, Axial fat-saturated T2-weighted MRI performed next day shows pancreas to be diffusely swollen with diffusely increased T2-weighted signal reflective of interstitial edema. There is peripancreatic edema and acute fluid (*arrowheads*) without fluid collection.

Fig. 6—

17-year-old boy with necrotizing pancreatitis after cardiac transplant.

A, Axial contrast-enhanced CT shows absent enhancement of large portion of pancreas (*arrow*), reflecting necrotizing pancreatitis with contiguous high-attenuation fluid in adjacent tissues reflecting acute necrotic collection.

B, Axial contrast-enhanced CT obtained 4 weeks later shows organization of collection and development of well-defined wall compatible with walled-off necrosis. Solid material is present within collection (*arrowhead*), which partially surrounds some residual pancreas (*arrow*).

C, Axial contrast-enhanced CT obtained after endoscopic intervention shows lumen opposing metal stent (*arrow*) extending from stomach into collapsed collection.

Fig. 7—.

14-year-old boy with abdominal pain after bicycle accident.

A, Axial contrast-enhanced CT of abdomen shows transection of pancreatic body (*arrow*) with adjacent parenchymal edema and small volume of acute peripancreatic fluid.

B, Axial fat-saturated T2-weighted MRI obtained 1 week later shows focal accumulation of fluid in lesser sac without defined wall (*black arrow*), compatible with acute peripancreatic fluid collection. Adjacent pancreas remains edematous (*white arrow*).

C, Axial contrast-enhanced CT of abdomen obtained 1 month after initial injury shows enlargement of simple fluid collection in lesser sac (*arrow*). There is now defined wall compatible with pseudocyst.

Fig. 8—
10-year-old girl with abdominal pain and enlarged pancreas by ultrasound, ultimately given diagnosis of autoimmune pancreatitis. Axial fat-saturated T2-weighted MRI shows diffuse, sausage-like enlargement of pancreas (*arrow*) with diffusely increased parenchymal signal and surrounding T2-weighted hyperintense halo. There is lack of substantial peripancreatic edema or fluid despite extent of parenchymal abnormality.

Fig. 9—

13-year-old girl with parenchymal and duct findings of chronic pancreatitis.

A, Axial T2-weighted SSFSE MRI shows diffuse pancreatic parenchymal atrophy with only thin band of tissue on both sides of dilated pancreatic duct (*arrow*).

B, Axial T1-weighted fat-saturated 3D gradient-recalled echo MRI shows diffusely decreased T1-weighted signal in visible parenchyma (*arrow*), which appears less intense than normal liver.

C, Coronal maximum-intensity-projection image from T2-weighted MRCP shows dilation and irregularity of main pancreatic duct and dilation of side branches (*arrowheads*). Discrete filling defect reflecting stone is present in dilated main duct (*arrow*).

Fig. 10—

13-year-old girl with acute recurrent pancreatitis.

A, Axial T2-weighted single-shot MRI during acute attack of pancreatitis shows swelling of pancreatic head (*arrow*) without visible duct.

B, Coronal maximum-intensity-projection (MIP) image from 3D MRCP shows dilation of pancreatic duct in body and tail without visible duct in pancreatic head.

C, Follow-up axial T2-weighted single-shot MRI 6 weeks later shows resolution of inflammation in pancreatic head (*arrow*) with duct now visible and passing anterior to common bile duct, reflecting pancreas divisum.

D, Coronal MIP image confirms pancreas divisum with pancreatic duct draining to minor papilla (*arrow*).

Fig. 11—

5-year-old girl with choledochal cyst and biliary obstruction resulting from stone in long common channel. Maximum-intensity-projection image from 3D MRCP shows anomalous pancreaticobiliary junction with pancreatic duct joining dilated common bile duct well above papilla, resulting in long common channel (*arrow*). Large ovoid filling defect in common channel reflects obstructing stone (*asterisk*). Common bile duct and central hepatic ducts are dilated, reflecting choledochal cyst.

TABLE 1:

Definitions of Pancreatitis Based on INSPPIRE Criteria [6]

Type of Pancreatitis	Definition
Acute pancreatitis (AP)	Meets two of three criteria: <ul style="list-style-type: none"> • Abdominal pain consistent with AP • Serum lipase or amylase at least three times the upper limit of normal • Characteristic imaging findings of AP
Acute recurrent pancreatitis (ARP)	Two or more distinct attacks of AP. Attacks must be separated by either a 1-month pain-free interval or by resolution of pain and normalization of serum amylase and lipase
Chronic pancreatitis (CP)	Imaging findings of CP and one of the following: <ul style="list-style-type: none"> • Abdominal pain consistent with pancreatitis • Exocrine pancreatic insufficiency • Endocrine pancreatic insufficiency

Note—INSPPIRE = International Study Group of Pediatric Pancreatitis: In Search for a Cure.

TABLE 2:

MRI Sequences for Assessment of Pediatric Pancreatitis

Sequence	Purpose	Notes
MRCP: heavily T2-weighted 3D FSE	Overall assessment of pancreaticobiliary tree, anatomy, anomalies, stones, or obstruction	Requires respiratory triggering or navigation; near isotropic
MRCP: heavily T2-weighted thick-slab SSFSE	Overall assessment of pancreaticobiliary tree, anatomy, anomalies, stones, or obstruction	Adjust thickness between 2 and 4 cm based on patient size
Coronal and axial T2-weighted SSFSE	Backup to MRCP; for visualizing duct anomalies, connections, common channel and small biliary stones	Thin slice (3–4 mm), no gap; moderate TE (140–200 ms) to accentuate duct fluid; no fat saturation to maximize signal
Axial balanced SSFP	Backup to MRCP; motion robust; complementary for duct visualization	High signal in vessels (WBCs) can be problematic
Axial T2-weighted FSE or SSFSE fat-saturated	Overall assessment of pancreas, fluid collections, and other organs	Accentuates parenchymal edema and peripancreatic fluid
Axial 3D T1-weighted GRE (VIBE, THRIVE, LAVA) fat-saturated	Assessment of pancreatic parenchyma; pigment biliary stones appear bright on this sequence [67]; volume data can be used for assessment of pancreatic volume	
Optional sequences		
DWI	Inflamed pancreas shows diffusion restriction both in acute pancreatitis [68] and autoimmune pancreatitis [69]; may contribute to assessing complexity of fluid collection	
T2-weighted 3D FSE or SSFSE performed before and after secretion administration	Assessment of pancreatic duct anatomy; assessment of pancreatic duct dynamic change; assessment of secretory function	
Axial contrast-enhanced 3D T1-weighted GRE (VIBE, THRIVE, LAVA) fat-saturated	Assessment of pancreatic parenchymal enhancement; assessment of peripancreatic vessels	

Note—FSE = fast spin-echo, GRE = gradient-recalled echo.

TABLE 3:
Revised Atlanta Criteria–Defined Fluid Collections and Their Imaging Features [27]

Pancreatitis Type, Fluid Collection Type	Imaging Features
Interstitial edematous pancreatitis	
Acute peripancreatic fluid collection (APFC)	No defined wall; peripancreatic in location; conforms to adjacent spaces; homogeneous; no solid or semisolid material
Pseudocyst	Same as APFC, but with defined wall, typically more than 4 wk from onset
Necrotizing pancreatitis	
Acute necrotic collection (ANC)	No defined wall; pancreatic and/or peripancreatic in location; may be transspatial; contains fluid, nonliquefied debris, fat globules, or loculations
Walled-off necrosis	Same as ANC, but with defined wall, typically more than 4 weeks from onset

TABLE 4:**Interpretation Pearls and Pitfalls**

Factor	Considerations
Imaging modality	Findings of interstitial edematous pancreatitis by ultrasound can be very subtle; ultrasound may be particularly insensitive for chemotherapy-induced pancreatitis
Abnormal pancreas	A pancreatic duct, visible in its entirety in a child, is generally abnormal; any visible common biliary and pancreatic duct channel should be considered abnormal; a normal liver serves as an internal reference for pancreas parenchymal signal by MRI; on T1-weighted MRI, the pancreas should be the brightest organ in the upper abdomen
Acute pancreatitis	Fat-saturated T2-weighted imaging highlights subtle parenchymal and peripancreatic edema; interstitial edematous pancreatitis can result in heterogeneous enhancement on CT or MRI that can be difficult to distinguish from limited or early necrosis; evolution on follow-up imaging can confirm a diagnosis
Fluid collections	Fluid collections within the pancreas, even if simple, are necrotic collections; 4 wk is a useful estimate of the timeline for organization of collections but the maturity of the investing wall, not the timeline, defines the degree of organization
Abuse	In young (< 3 y of age) or developmentally delayed children, abuse must be considered as a possible cause of acute pancreatitis