

JOHNS HOPKINS ALL CHILDREN'S HOSPITAL

Acute Pancreatitis Clinical Pathway

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Owners: Dr. Michael Wilsey

This pathway is intended as a guide for physicians, physician assistants, nurse practitioners and other healthcare providers. It should be adapted to the care of specific patient based on the patient's individualized circumstances and the practitioner's professional judgment.

Johns Hopkins All Children's Hospital

Acute Pancreatitis Clinical Pathway

Rationale:

This clinical pathway was developed by a consensus group of JHACH physicians, advanced practice providers, nurses, and pharmacists to standardize the management of children hospitalized for **Acute Pancreatitis** Disease. It addresses the following clinical questions or problems:

1. When to evaluate for Acute Pancreatitis
2. When to consider admission for further evaluation and management
3. When to consult Gastroenterology clinical service or Pain team
4. When to consider enteral nutrition, endoscopic/surgical procedures, further imaging

Background

The incidence of Acute Pancreatitis (AP) has been increasing in recent decades and has a variety of etiologies in children estimated now at ~1/10,000 children per year¹. Etiologies for acute pancreatitis include anatomic, obstructive/biliary, infections, toxins, metabolic, systemic illness, inborn errors of metabolism, and genetic predisposition¹. Most of the literature is based on adult research studies and experience. In general, children usually have a mild clinical course; however, a subset of children can develop a more severe clinical course with local and systemic complications from AP². Although gastroenterologists can be consulted during a hospitalization for AP, children are more often first managed by pediatricians and pediatric hospitalists indicating a need for broader awareness of the most recent management recommendations. The most recent North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition Pancreatitis Committee recommendations were published in 2018 and serve as the basis for the recommendations detailed throughout this guideline.

The newest recommendations for AP including aggressive early fluid resuscitation/administration, close monitoring, adequate pain control, early enteral/parenteral nutrition, and clear indications for endoscopic and surgical procedures. Currently, there is still limited evidence for use of antibiotics and protease inhibitors in the setting of AP.

Diagnosis

Diagnosis of pediatric acute pancreatitis (AP) should be as per previously published INSPPIRE criteria¹. Diagnosis of AP in pediatric patients requires at least 2 of the following:

1. Abdominal pain compatible with AP
2. Serum amylase and/or lipase values 3 times upper limits of normal for age
3. Imaging findings consistent with AP²

Acute recurrent pancreatitis (ARP) and chronic pancreatitis (CP) are defined below for completeness but not covered entirely within this pathway:

Acute recurrent pancreatitis is defined by at least 2 acute attacks within a year, with interval resolution of pain or normalization of serum pancreatic enzyme levels, or by more than 3 lifetime episodes without evidence of CP.

Chronic pancreatitis requires the presence of exocrine or endocrine insufficiency and histologic and morphologic changes that are irreversible, including fibrosis, islet cell loss, inflammatory cell infiltrates, and intraductal calculi.

Lab tests:

Based on the most frequent etiologies and those for which therapeutic options exist, initial AP testing should include liver enzymes (ALT, AST, GGT, ALP, direct and total bilirubin), fasting triglyceride level, and calcium level.

The main biochemical markers are serum lipase and amylase. A diagnosis of acute pancreatitis is well supported by a serum amylase or lipase greater than 3 times the upper limit of normal for age. It should be noted that there are several non-pancreatitis conditions which could also elevate serum lipase or amylase including decompensated liver failure, renal failure, intestinal inflammation, trauma to the abdomen or head, and diabetic ketoacidosis. Please obtain Lipase on initial work-up alone, if inconclusive then can consider obtaining amylase. The following table demonstrates the age-based lab values for both amylase and lipase to be used as cut-off values when determining the upper limit of normal within the Johns Hopkins All Children’s Hospital lab system. These labs are applicable for both male and female patients.

LIPASE, Serum Ranges

Age	Normal Range (Male and Female)	3x Upper limit of normal
0 days- 18 years of life	4-39	>117
>19 years	8-78	>234

AMYLASE, Serum Ranges

Age	Normal Range (Male and Female)	3x Upper limit of normal
0-15 Days of life	3-10	>30
15days-3 months days of life	2-22	>66
3-12 months	3-50	>150
1-18 years	25-101	>303
>19 years	25-125	>375

For clinical disease monitoring, serum electrolytes such as BUN and creatinine and a CBC are important to monitor for signs of infection, bleeding, fluid/hydration status and renal function. A hepatic function panel is indicated if assessing for biliary or gallstone etiology. Baseline calcium and triglyceride levels can be obtained.

Consider testing for genetic causes/predispositions (such as cystic fibrosis transmembrane conductance regulator (CFTR), serine protease inhibitor Kazal-type 1 (SPINK1), and cationic trypsinogen (PRSS1), sweat chloride and more detailed imaging if 2nd episodes of AP with increased concern for underlying risk for recurrence based on presentation, medical history of family history¹.

Radiologic studies:

If clinical history and biochemical serum markers are consistent with diagnosis of AP, imaging in the early phase is usually not required. Imaging becomes important to evaluate for pancreatic necrosis, complications of pancreatitis including fluid collections, and etiology such as gallstone/biliary disease or anatomic abnormalities.

In cases that are ambiguous for a diagnosis of AP, such as in a delayed presentation when serum markers may be low, a contrast-enhanced CT may be required to confirm AP. IV contrast is key to distinguish necrotic areas. As early imaging may underestimate extent of disease and because complications evolve over time and findings may not be present in the early phase of the disease, CT should ideally be delayed at least 96 hours after onset of symptoms.

Ultrasound is used frequently when the suspicion for biliary pancreatitis is high because it is helpful for early determination of need for therapeutic intervention. If abdominal ultrasound shows biliary or pancreatic stone, it should be removed (with likely stenting) via ERCP. If gallstones are present, patient will likely need a cholecystectomy.

Magnetic resonance cholangiopancreatography is used for detecting distal common bile duct stones and diagnosing biliary causes of AP.

MRI of the abdomen/pelvis is typically not utilized initially but can be useful for late complications.

Initial imaging may be accomplished via transabdominal ultrasonography, with other imaging (CT, MRI) reserved for more complicated cases tailored to suspected etiology.

Determine Disease Severity

Mild

AP that is not associated with any organ failure, local or systemic complications, and usually resolves in the first week. This is the most common form.

Local complications would include development of (peri) or pancreatic complications including fluid collections or necrosis. Systemic complications would include exacerbation of previously diagnosed co-morbid disease (such as lung or kidney disease).

Moderately severe

AP with either the development of transient organ failure/dysfunction (lasting no more than >48 hours) or development of local or systemic complications.

Severe

AP with development of organ dysfunction that persists >48 hours. Persistent organ failure may be single or multiple and may develop beyond the first 48 hours of presentation.

General Considerations:

Mild	<ul style="list-style-type: none">● General (regular) diet and advance as tolerated (PO>NG)● Pain control with PO medications● Routine vital signs monitoring
Moderately Severe	<ul style="list-style-type: none">● Enteral Nutrition (oral, NG or NJ) should be attempted within 72 hours from presentation once deemed hemodynamically stable. If not tolerated, may use parenteral nutrition.● May require IV pain medications● Continue IVFs to maintain hydration● Monitor BUN and Cr
Severe	<ul style="list-style-type: none">● Enteral Nutrition (oral, NG or NJ) should be attempted within 72 hours from presentation once deemed hemodynamically stable. If not tolerated, may use parenteral nutrition.● Requires IVF, IV pain medications● Patient with signs of severe sepsis/septic shock, please refer to sepsis pathway and PICU management

Clinical Management

Fluid management

Children with AP should be initially resuscitated with crystalloids, either with lactated ringer's (LR) or normal saline (NS) in the acute setting¹. There may be some evidence that LR is favorable compared to NS but pediatric data are lacking. Of note, there was one recent large retrospective study showing LR was associated with shorter hospital stay and reduced cost compared with NS¹⁴. Based on assessment of hydration status/hemodynamic status, if evidence of hemodynamic compromise, a bolus of 10 to 20 mL/kg is recommended. Children with diagnosis of AP should be provided 1.5 to 2 times maintenance IV fluids such as D5W + 0.9%NS with monitoring of urine output over the next 24 to 48 hours. Please note, this is for patients with normal baseline electrolytes and does not include those with other complications that alter electrolyte status such as panhypopituitarism, metabolic disorders, psychogenic polydipsia, SIADH etc.

The purpose of fluid management, particularly in severe AP, is to provide support for the altered pancreatic microcirculation that can lead to disease progression and hypovolemia. Local inflammation leads to increased capillary permeability causing hypovolemia and increasing formation of microthrombi

Clinical Monitoring

In patients admitted to an inpatient ward, vitals should be obtained at least every 4 hours during the first 48 hours of admission and during periods of aggressive hydration to monitor oxygen saturation, pulse, blood pressure and respiratory rate. Frequency to be adjusted based on clinical status. Abnormalities of vital signs and altered clinical status should prompt immediate support and update to pediatric gastroenterologist.

Cardiovascular: Monitor for hypovolemia and tachycardia. Improvement in tachycardia may be used to confirm adequate fluid resuscitation in addition to monitoring urine output and skin turgor. Rare cases of cardiac tamponade and atrial fibrillation have been reported in AP and should be considered as part of the standard cardiac workup if patient develops unexplained hypotension, shortness of breath, and/or chest pain.

Pulmonary: Early complications of AP (within first 48 hours) may include acute respiratory distress syndrome, pneumonia, and pulmonary edema/effusions. Routine monitoring of oxygen saturation during aggressive hydration is typically implemented. May consider bed elevation to 30-degree angle to decrease likelihood of pulmonary sequestration. Consider further workup if patient develops unexplained shortness of breath, worsening cough, and/or difficulty breathing.

Renal: BUN, creatinine and urine output should be routinely monitored in the first 48 hours and to screen for Acute Kidney Injury which may occur via abdominal compartment syndrome or inflammatory-driven damage to proximal tubule. Acute kidney injury has been highly associated with increased risk of mortality in severe AP. There are no specific guidelines on frequency of monitoring these parameters. Any abnormalities should prompt nephrology consultation¹.

Pain Management

No data provide guidance for optimal pain management in pediatric AP. Therefore, providers should use analgesic that is most appropriate for patient history and pain status. There is no existing evidence supporting the contention that morphine causes adverse events on the sphincter of Oddi. IV morphine or other opioid should be used for acute pancreatitis pain not responding to acetaminophen or NSAIDs. Long-acting NSAIDs including indomethacin and diclofenac have been shown to be useful in the prevention of post-ERCP pancreatitis. Epidural anesthesia may also be considered to decrease pain in patients with AP. Acute pain specialist services should be consulted in cases of more severe pain to optimize pain management.

Enteral and Parenteral Nutrition

In the past, patients have generally had enteral nutrition withheld with the aim of suppressing pancreatic enzyme secretion and obtaining bowel rest⁴. However, more recent studies have demonstrated this approach may lead to an increased risk of infectious complications due to bacterial overgrowth and translocation from the gut leading to higher morbidity and mortality in patients with severe acute pancreatitis¹. Nutritional support should begin after any necessary fluid resuscitation and ensuring hemodynamic stability of the patient. Parenteral nutrition (PN) support should be considered if patient is anticipated to be without enteral nutrition (NPO) for greater than 5-7 days regardless of severity of disease. Unless there is a direct contraindication to use the gut, children with mild AP may benefit from early (within 48–72 hours of presentation) oral/enteral nutrition (EN) to decrease LOS and decrease risk of organ dysfunction³. Early nutrition should also be attempted in severe AP when possible.

Parenteral nutrition (PN) should be used when:

- oral/nasogastric/nasojejunal feeds are not tolerated or not possible for a prolonged period (longer than 5–7 days) such as in
- ileus,
- complex fistulae, abdominal compartment syndrome. Nutrition may be consulted on patients with difficulty tolerating PO, patients with significant weight loss, patients with suspected malnutrition, or patients who require nutrition support (EN or PN). Enteral nutrition should commence as soon as feasible, with the goal of moving from PN towards EN⁴.

Continuous enteral feeds are superior to cyclic or bolus enteral feeds. Nasogastric feeds can be used; post pyloric are not required. A peptide-based formula low in fat and high in medium chain triglycerides is ideal for enteral feeds. For patients not meeting caloric goals with EN alone, a combination of EN and PN appears preferable to PN alone. In cases of pancreatic laceration, fracture, or duct disruption, it is unclear whether oral/enteral feedings may be detrimental in the acute phase. Depending on the patient's clinical status, consult nutrition for specific recommendations regarding use of standard versus custom parenteral nutrition as clinical presentations can vary greatly. In general, IV fat emulsions are safe in patients with normal baseline triglycerides at time of admission.

Use of antibiotics, protease inhibitors and probiotics

Prophylactic antibiotics are not empirically recommended in severe AP. Antibiotic use is indicated only in cases of documented infected necrosis or in those with known necrotizing pancreatitis who do not improve throughout the hospitalization. Antibiotics to be considered include carbapenems (particularly meropenem), third generation cephalosporins, quinolones and metronidazole. Please consider choice based on patient past medical history, degree of infected necrosis or non-improving clinical status with infectious disease specialists. Anti-proteases cannot be recommended in the management of acute pancreatitis in children at this time⁶. Antioxidants should not be considered standard therapy in the management of pediatric AP⁷. Probiotics cannot be recommended in the management of pediatric AP at this time. Highest-quality published adult evidence suggests they may even increase mortality, but at a minimum do not demonstrate a benefit⁸.

Endoscopy

Esophago-gastroduodenoscopy is not considered a standard diagnostic tool in pediatric AP at this time⁷. Based on adult literature, EUS may be useful to determine the etiology of acute pancreatitis; however, its role for therapy is mostly limited for the treatment of complications of acute pancreatitis, such as pancreatic fluid collections or walled off pancreatic necrosis. Indication for its use should be determined on a case-by-case basis.

ERCP

The role of ERCP is limited in AP and depends on local expertise. ERCP is indicated in management of AP related to choledocholithiasis causing biliary pancreatitis, and for pancreatic duct pathologies such as ductal stones or ductal leaks^{9,10}.

Endoscopic Ultrasonography

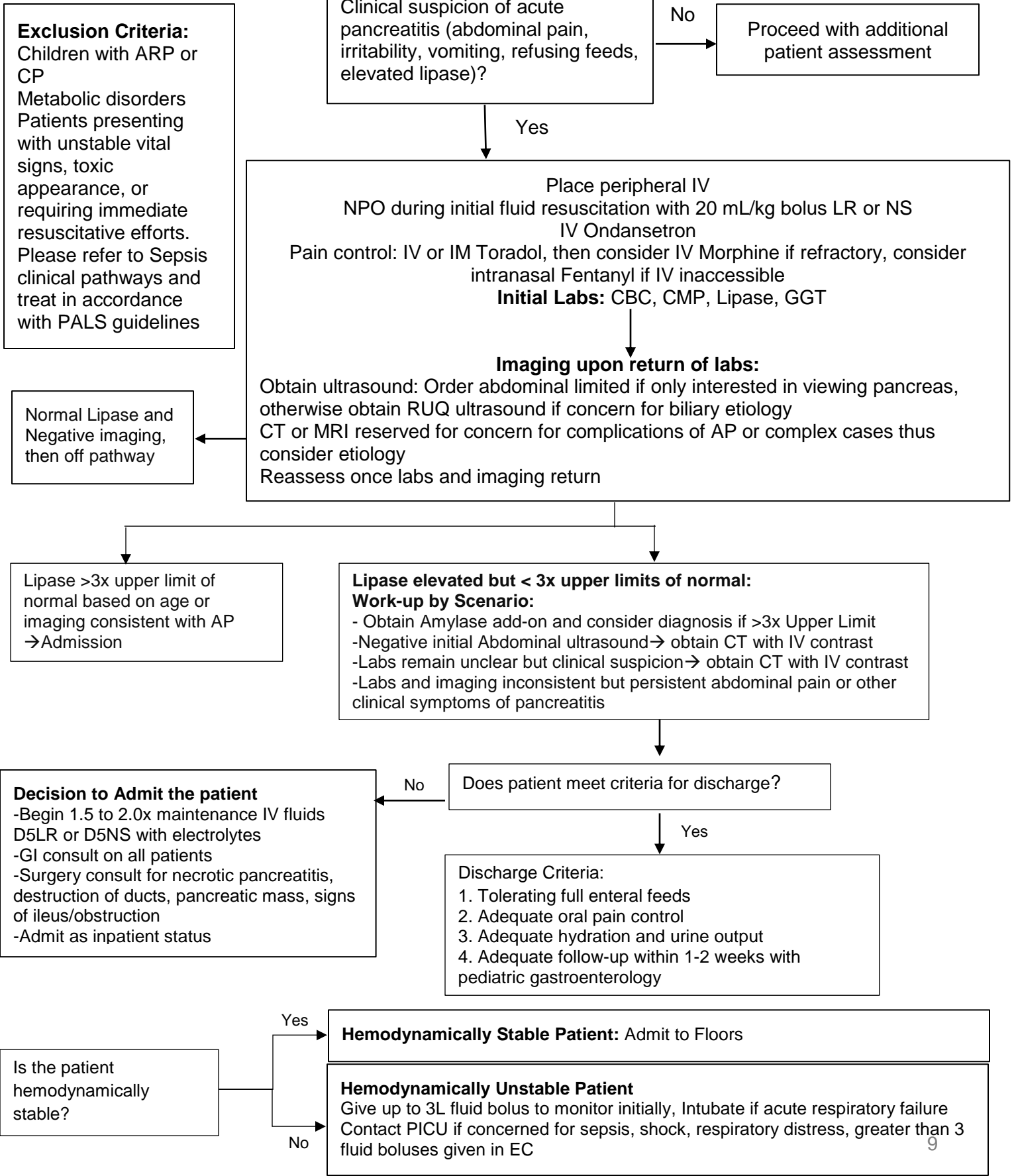
Endoscopic Ultrasonography is not considered a standard diagnostic tool in pediatric AP at this time⁷. Indication for its use should be determined on a case by-case basis.

Surgery

Indications for acute surgical intervention in AP indications include abdominal trauma where patient instability and/or search for associated injury to other organs is occurring. An early indication for surgery includes management of abdominal compartment syndrome.

Cholecystectomy safely can and should be performed before discharge in cases of mild uncomplicated acute biliary pancreatitis⁷. In the management of acute necrotic collections, interventions should be avoided and delayed, even for infected necrosis, as outcomes are superior with delayed (>4 weeks) approach⁹. When drainage or necrosectomy is necessary, non-surgical approaches including endoscopic (EUS, and ERCP-assisted) or percutaneous methods are preferred over open necrosectomy or open pseudocyst drainage¹¹.

Emergency Center Acute Pancreatitis Clinical Pathway



Emergency Center Management

Evaluation of patients presenting with abdominal pain, vomiting, or feeding refusal consistent with acute pancreatitis requires lab work and/or imaging to support the clinical diagnosis. Any patient diagnosed with acute pancreatitis should be admitted to the hospital for further evaluation and close monitoring. The initial management involves pain control and aggressive fluid management to ensure perfusion even in the case of stable vital signs. Lab work focuses on assessing the patient's hydration status and electrolytes, including a baseline creatinine. Amylase and lipase are serum biochemical markers which can be very important for confirming the diagnosis. Imaging is important if there are specific concerns for complications associated with acute pancreatitis (based on suspected etiology), or in the situation of unclear diagnosis based on lab results. Imaging can begin with an Abdominal ultrasound which may show pancreatic inflammation or biliary pancreatitis. CT with IV contrast may not be part of initial work-up unless there is concern for complications, patients with complex etiologies, or after discussion with GI or surgical teams pending clinical suspicion.

Admission

- Admission criteria includes suspected or confirmed diagnosis of acute pancreatitis.
- Patients require close monitoring for early and late complication development as well as potential need for IV opioid therapy.
- Patient will be admitted NPO during initial fluid resuscitation.
- If the patient will be admitted on IV fluids with serial lab assessments, please place the patient in inpatient status.

Johns Hopkins All Children's Hospital
Inpatient Acute Pancreatitis Clinical Pathway

Patient admitted with Acute Pancreatitis:

Fluids: 1.5 to 2.0x maintenance IV fluids with D5LR or D5W+NS, monitoring urine output over next 24-48 hours.

Monitoring: Vitals q4h. Monitor Respiratory Status and urine output throughout admission. Obtain Triglycerides within initial 24 hours. Monitor CBC and CMP for first 48 hours, then if changes in clinical status, can repeat to monitor for signs of bleeding, infection, renal, liver and electrolyte status

Obtain Contrast enhanced CT abdomen with new onset worsening status

Consider PICU admission with signs of hemodynamic instability or Multiorgan system failure:

- Respiratory distress requiring oxygen support beyond floor guidelines
- Worsening Clinical status despite treatment
- Persistent hypotension despite fluid bolus

Analgesia: Acetaminophen, NSAIDs, oxycodone, IV morphine, IV Ketorolac for pain. Consult Pain team in cases of more severe or uncontrolled pain. Consider severity in the setting of Pain syndromes. Adjust Pain regimen and reassess in 12 hours. Transition to oral pain medications as tolerable

Nutrition: Start feeds as early as possible, ideally within 24-72 hours of presentation. Consider NG feeds if oral not tolerated. *No official guidelines have been published by NASPGHAN regarding when to start, thus determine in conjunction with patient tolerating Ondansetron and willingness to attempt po trial.

Antibiotics: Do NOT begin empirically, only in the situation of documented Infected Necrotizing Pancreatitis, or those with necrotizing pancreatitis who are not improving during hospitalization.

- Need to consider EN within 48-72 hours:
-Consider PN if unable to tolerate EN once >5 days and obtain Nutrition Consult
-Continue IVF, Anti-emetic prn
-Begin po challenge with liquid diet and progress as tolerable unless **contraindications**
-Continue IV or po Analgesia as tolerable

Reassess patient and consider if Discharge criteria met

Consider discharge when the following criteria are met:

1. Tolerating full enteral feeds
2. Adequate oral pain control
3. Adequate hydration and urine output
4. Adequate follow-up within 1-2 weeks with pediatric gastroenterology
5. Determine Classification of Pancreatitis (Mild, Moderately Severe, Severe) as defined

Inpatient Management

- Clinical management of admitted patients focuses on close observation for potential complications as well as appropriate pain management. Consider admission of at least 24 hours to ensure aggressive fluid management, appropriate monitor of symptoms and vital sign changes, and to assess for any disease progression.
- Early initiation of enteral nutrition and aggressive fluid resuscitation has been linked to shorter hospital stays, fewer ICU admissions, and decreased of severe AP than those made NPO¹. Although there is no specified timeframe of when to begin, typically of goal of within 48-72 hours of enteral feeding should be established. If the patient is unable to tolerate enteral feeding for greater than 5 days, then consider parenteral nutrition. Even in this situation however if the patient can tolerate some enteral with parenteral feeding, that is ideal over total parenteral nutrition. In the case of TPN, try to wean them off as early as able.
- Early complications include multiorgan dysfunction, shock, and acute peripancreatic fluid collections.
- Later complications include pseudocyst formation, pancreatic necrosis, and walled off pancreatic necrosis.
- Consider contrast enhanced CT if patient's clinical condition deteriorates or is persistently severe.

Discharge

Discharge criteria:

- Tolerating full enteral feeds
- PO pain meds
- Adequate hydration status
- Children with AP should receive close follow-up by a health care provider to identify early or late complications, or recurrence⁷(within 1-2 weeks)

Children need to be followed during their course of AP for local and systemic complications. Overall pediatric patients with acute pancreatitis have a good prognosis with extremely low rate of mortality, but up to 15-35% rate of recurrence is reported. Children should receive close follow-up with a health care provider to identify early or late complications or recurrence.

Discharge Recommendations

1. Suggest patient begins a low-fat diet for minimum 1 month after diagnosis
2. Discuss potential etiology for AP and make steps to mitigate inciting event

Documentation Reminders

1. It is important to document if the cause is idiopathic, biliary, infectious, drug induced, etc. If drug induced, please document the causative medication. See Figure below to review causes of pediatric acute pancreatitis
2. It is important to document if there is necrosis present.
3. It is important to document if the pancreatitis is acute, acute on chronic, chronic, or even resolving acute pancreatitis with developing chronic pancreatitis, if that is suspected.
4. It is important to document any manifestations of the acute pancreatitis, such as acute malnutrition. If the patient has experienced associated weight loss, consider nutrition consult and/or review of RD notes to confirm presence of acute or chronic malnutrition, and severity.

Documentation Details: Causes of Acute Pancreatitis

The cause for acute pancreatitis is crucial to document for a patient with Acute pancreatitis as this may be their primary diagnosis and reason for admission, or a development that occurs secondary to numerous etiologies while a patient is being treated.

Patient with primary Acute Pancreatitis admitted to the hospital have a more favorable outcome when comparing morbidity and mortality compared to secondary acute pancreatitis¹⁸.

Additionally, those with secondary AP present in more severe clinical conditions. Mortality associated with AP is more often related to multiorgan system failure than directly from AP.

Causes of Acute Pancreatitis (Saeed, 2020)

Congenital Anomalies	Choledochal cyst, pancreas divisum, pancreaticobiliary malunion
Biliary	cholecystitis, gall stones, tumor
Systemic	Sepsis, immune mediated conditions (SLE, JIA, HSP, IBD), Kawasaki disease, polyarteritis nodosa, organ transplantation, chronic total parenteral nutrition use
Medication	Acetaminophen Azathioprine Cannabis Cocaine Codeine Cytarabine, Dapsone Enalapril Fosphenytoin Furosemide, Indomethacin, Metronidazole, mercaptopurine, Pegylated asparaginase Phenytoin, Prednisone, Salicylic acid, Tetracyclines Thiopurines Trimethoprim/sulfamethoxazole Valproate
Trauma	Abdominal trauma, post-ERCP, perforated gastric or duodenal ulcer

Infection	Measles, mumps, coxsackie virus, echovirus, influenza, Epstein-Barr virus, mycoplasma, Salmonella, hepatitis A, and Escherichia coli
Metabolic	Hyperlipidemia, hypertriglyceridemia, cystic fibrosis, diabetes mellitus, hypercalcemia, Reye's syndrome, renal disease, propionic acidemia, nutritional deficiency, and hereditary pancreatitis
Miscellaneous	Familial, Idiopathic

Complications of Acute Pancreatitis and Management:

Local	Systemic
Localized inflammation	Shock
Localized edema	Sepsis
Ileus	Hypermetabolic state
Pancreatic Necrosis	Hypocalcemia
Pancreatic Abscess	Hyperglycemia
Fat necrosis pancreatic hemorrhage	Vascular leak syndrome
Pancreatic pseudocyst	Multiorgan system failure
Pancreatic duct rupture	Disseminated intravascular coagulation
Pancreatic duct stricture	Pleural effusions
Thrombosis of adjacent blood vessels	Acute renal failure
Extension to nearby organs	Splenic artery pseudoaneurysm
	Acute Respiratory failure

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Outcome Measures

- Admissions
- Length of stay

Clinical Pathway Team
Acute Pancreatitis Clinical Pathway
Johns Hopkins All Children's Hospital

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Disclaimer

Clinical Pathways are intended to assist physicians, physician assistants, nurse practitioners and other health care providers in clinical decision-making by describing a range of generally acceptable approaches for the diagnosis, management, or prevention of specific diseases or conditions. The ultimate judgment regarding care of a particular patient must be made by the physician in light of the individual circumstances presented by the patient.

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