Sensitivity of Biochemical and Imaging Findings for the Diagnosis of Acute Pancreatitis in Children

Sarah H. Orkin, MD[1], Andrew T. Trout, MD[2,3], Lin Fei, PhD[4], Tom K. Lin, MD[1,5], Jaimie D. Nathan, MD[6], Tyler Thompson, BS[1], David S. Vitale, MD[1,5], and Maisam Abu-El-Haija, MD[1,5]

Objective To determine the diagnostic sensitivity of serum biomarkers and imaging in the diagnosis of acute pancreatitis in children.

Study design This was a cross-sectional analysis of prospective registry data for children (age <21 years) whose first documented attack of acute pancreatitis occurred between March 2013 and October 2016 at a single-institution, tertiary care center. Main outcome was sensitivity of serum biomarkers and of imaging modalities, measured via descriptive statistics.

Results In total, 112 children met the criteria for acute pancreatitis; 57 (51%) were male with a median age of 13.4 years (IQR 9.3-15.8 years). Serum amylase and lipase levels were obtained in 85 (76%) and 112 (100%) patients, respectively. Imaging was performed in 98 (88%) patients, with abdominal ultrasound (US) performed in 84 (75%) and computed tomography and/or magnetic resonance imaging performed in 46 (41%) patients. Fifty-three (47%) patients met all 3 diagnostic criteria (clinical, biochemical, and imaging) for acute pancreatitis. Laboratory testing had a 5.4% false-negative rate for acute pancreatitis. Serum lipase alone and amylase alone were 95% (95% CI 89%-98%) and 39% (95% CI 28%-50%) sensitive for acute pancreatitis, respectively. Imaging (any modality) was 61% sensitive (95% CI 51%-71%) for acute pancreatitis with a 34% false-negative rate. US alone was 52% (95% CI 41%-63%) sensitive for acute pancreatitis and computed tomography/magnetic resonance imaging was 78% (95% CI 63%-89%) sensitive. Combinations of diagnostic criteria performed no better than laboratory testing alone.

Conclusions The majority of children coming to medical attention with their first documented occurrence of acute pancreatitis have characteristic symptoms. Serum lipase is highly sensitive for the diagnosis of acute pancreatitis, and serum amylase is moderately sensitive. Imaging, particularly US, is only moderately sensitive, and cross-sectional imaging provides greater sensitivity for diagnosing acute pancreatitis. *(J Pediatr 2019;213:143-8)*.

Acute pancreatitis is being recognized increasingly in children.[1,2] With an incidence of up to 13 per 100 000 per year and a median hospitalization time of 5-8.5 days, the economic burden associated with this diagnosis is significant.[1-4] On average, the median estimated cost per admission for an episode of pediatric acute pancreatitis is $22 663, with costs reaching $43 000 for those admitted to the intensive care unit.[5,6] The average total cost of admissions for pediatric acute pancreatitis approximate $200 million per year.[7] Despite both the growing recognition of this disease and the cost of care, there remain few data on optimal diagnostic approaches for pediatric acute pancreatitis. To date, data and clinical practice are largely extrapolated from studies in adults. This is problematic for multiple reasons, including the differing etiologies and risk factors for acute pancreatitis between adults and children, with gallstones and alcohol being the predominant causes in adults, and biliary, metabolic, systemic, hereditary, and anatomic abnormalities predominating in children.[7,8]

Presently, the diagnosis of acute pancreatitis in children is made when a patient meets 2 of 3 criteria: (1) biochemical (serum amylase or serum lipase ≥3 × the upper limit of normal), (2) imaging (findings of pancreatic edema, fat stranding, or peripancreatic fluid collection), and/or (3) clinical symptoms consistent with pancreatitis.[9] Each of these diagnostic elements are understudied in children and mainly derived from retrospective data, with a broad range of results reported in the literature. For example, the sensitivity of serum lipase has been reported to be 77%-100%, and amylase sensitivity is 52%-54% in pediatric studies.[3,4,10] In adult studies, the specificity has ranged from 85%-99% for serum lipase, and

CT	Computed tomography
MRI	Magnetic resonance imaging
US	Ultrasound

From the [1]Division of Pediatric Gastroenterology, Hepatology and Nutrition; [2]Department of Radiology, Cincinnati Children's Hospital Medical Center; [3]Department of Radiology, University of Cincinnati Medical Center; [4]Division of Biostatistics and Epidemiology, Cincinnati Children's Hospital Medical Center; [5]Department of Pediatrics, University of Cincinnati College of Medicine; and [6]Division of Pediatric General and Thoracic Surgery, Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio

M.A.-E.-H. is supported by National Institute of Diabetes and Digestive and Kidney Diseases (1K23DK118190-01). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. The authors declare no conflicts of interest.

Portions of this study were presented at the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition Annual Meeting, October, 2018, Hollywood, Florida

https://doi.org/10.1016/j.jpeds.2019.06.028

~70% for amylase.[11-13] Ultrasound (US) has been shown to be 24%-86% sensitive for acute pancreatitis, and computed tomography (CT) has been shown to be 47%-75% sensitive, with few data regarding the diagnostic accuracy of magnetic resonance imaging (MRI) in pediatric acute pancreatitis.[3,4,7,10,14] The purpose of our study was to leverage a novel prospectively collected database of children presenting with a first attack of acute pancreatitis to assess the sensitivity of each of the diagnostic criteria for acute pancreatitis in children.

Methods

This was a cross-sectional analysis of patients enrolled between March 2013 and October 2016 in a prospectively collected, institutional review board–approved database of children <21 years of age presenting to Cincinnati Children's Hospital Medical Center (Cincinnati, Ohio) with their first documented episode of acute pancreatitis. Diagnosis of acute pancreatitis was based on the presence of 2 or more of the following criteria with testing acquired per the treating provider's discretion[9]: (1) pain consistent with a pancreatic origin or surrogate markers of vomiting, fussiness, and feeding intolerance, which may be more common signs/symptoms of acute pancreatitis in infants and toddlers[4]; (2) serum amylase or lipase level ≥3 times the upper limit of normal; (3) and imaging (US, CT, or MRI) with findings of acute pancreatitis, including pancreatic edema, pancreatic hypoenhancement, peripancreatic edema or fluid collection, and frank pancreatic or peripancreatic necrosis. Severe acute pancreatitis was defined as per the pediatric acute pancreatitis classification.[15]

A subset of the patients presented here have been included in previous publications, with different study aims and hypotheses.[16,17] Clinical, laboratory, and demographic data were extracted from the database at the time of presentation and included age, sex, and body mass index. Additional compiled data included risk factor(s) for acute pancreatitis such as concurrent illness, genetic risk factors, trauma, gallstones, and medications at the time of acute pancreatitis diagnosis. A patient's acute pancreatitis was considered idiopathic if no specific cause or risk factor was identified. All imaging obtained within +/− 1 week of acute pancreatitis diagnosis was retrospectively blindly reviewed for this study by a board-certified pediatric radiologist to confirm and document findings of acute pancreatitis. All available data were analyzed.

Statistical Analyses

Because the database included only patients with confirmed acute pancreatitis, the primary outcome of interest for the current study was sensitivity for acute pancreatitis diagnosis. Specificity could not be calculated due to the absence of negative cases. Sensitivity was calculated for individual diagnostic elements (eg, serum lipase and amylase levels and imaging by US and cross-sectional imaging [CT and MRI]), as well as for combinations of diagnostic elements.

The sensitivity analysis was carried out via a binomial distribution model, where exact CIs were computed using the Clopper–Pearson method. For categorical and continuous factors respectively, the Fisher exact and Kruskal–Wallis tests were used for inference testing. The association between severe acute pancreatitis as an outcome and serum lipase and amylase measurements was analyzed via a logistic regression model. All analyses were performed using SAS, version 9.4 (SAS Institute, Cary, North Carolina).

Results

During the study period, 112 patients were enrolled into the registry. The patient cohort included 57 (51%) male patients, with a median age of 13.4 years (IQR 9.3-15.8) (Table I); 84% of patients were white. Fourteen patients (12.5%) had severe acute pancreatitis.

The presence or absence of symptoms was recorded for all patients and serum lipase level was collected in all patients. Serum amylase was collected in 85 patients (76%). Imaging of any type (US, CT, MRI) was performed within 1 week of presentation in 98 patients (88%).

All but 1 patient had signs or symptoms of acute pancreatitis. The patient without characteristic signs or symptoms met the criteria for acute pancreatitis with an elevated serum lipase level (but normal serum amylase level) and acute pancreatitis findings identified by imaging performed as part of an evaluation for acute hepatitis.

Fifty-three patients (47%) met 3 of 3 diagnostic criteria for acute pancreatitis. The remaining 59 patients (53%) met 2 of 3 diagnostic criteria for acute pancreatitis. There were no significant differences in age, sex, or body mass index between patients who met 2 of 3 diagnostic criteria vs 3 of 3 criteria (Table II; available at www.jpeds.com).

Risk Factors for Acute Pancreatitis

The single most common identified risk factor for acute pancreatitis was medication (22%) followed by gallstones

Table I. Population characteristics including risk factors for acute pancreatitis (n = 112)

Characteristics	
Age at diagnosis, y, median (IQR)	13.4 (9.3-15.8)
Duration of follow-up, mo, median (IQR)	20.8 (10.0-32.8)
Sex, male, n (%)	57 (51)
Race, n (%)	
White	94 (84)
Black/African-American	9 (8)
Other	9 (8)
Body mass index percentile, median (IQR)	60.0 (24.1-92.1)
Risk factor for acute pancreatitis, n (%)*	
None (idiopathic)	34 (30)
Medication	25 (22)
Biliary or gallstone	21 (19)
Viral/systemic infection	19 (17)
Trauma	9 (8)
Genetic predisposition	8 (7)
Obstructive predisposition	3 (3)
Metabolic predisposition	2 (2)

Data presented as median (IQR) or n (%).
*Eight patients had multiple risk factors.

(19%). No specific risk factor for acute pancreatitis (ie, idiopathic pancreatitis) was identified in 30% of the patients. Eight patients (7.1%) had more than 1 identified risk factor for acute pancreatitis. Three patients had combined biliary and genetic risk, and 5 patients had combination of viral/systemic, medication, or metabolic risk factors.

Laboratory Markers

The frequency with which pancreatic enzyme levels were obtained and the frequency with which they were positive or negative is detailed in **Figure 1**. No patient had an elevated level of serum amylase without a corresponding elevated lipase. There were six patients in total who had normal biochemical values and instead met criteria for AP with both characteristic symptoms and positive imaging, resulting in a false-negative rate for serum lipase of 5.4% (95% CI 2.0%-11.3%) (**Table III**; available at www.jpeds.com). Serum lipase alone was 95% sensitive for acute pancreatitis (95% CI 89%-98%). Serum amylase alone was 39% sensitive for acute pancreatitis (95% CI 28%-50%), with a false-negative rate of 61% (95% CI 50%-72%).

For the patients who had both serum amylase and lipase measured at presentation, logistic regression (log-scale) showed both lipase and amylase were positively, but not statistically significantly, correlated to severe acute pancreatitis, with ORs of 1.28 (95% CI 0.13-12.66, $P = .83$) and 1.60 (95% CI 0.13-19.17, $P = .71$), respectively.

Imaging Findings

Eighty-four patients (75%) had an US performed, and CT or MRI was performed in 46 (41%) patients (**Figure 2**). The frequency of imaging findings identified by each imaging modality is detailed in **Table IV**. The most frequent finding across all 3 modalities was pancreatic edema (**Figure 3**; available at www.jpeds.com), followed by peripancreatic edema and acute peripancreatic fluid collection.

Imaging (of any type) within 1 week of presentation for subsequently confirmed acute pancreatitis was only 61% sensitive (95% CI 51%-71%) for acute pancreatitis (**Table III**). Thirty-eight (34%) patients had pain and positive laboratory findings with normal imaging, a 34% false-negative rate for imaging. US alone was 52% (95% CI 41%-63%) sensitive for acute pancreatitis and CT or MRI was 78% (95% CI 63%-89%) sensitive. CT or MRI identified acute pancreatitis in 7 of 12 patients (58%) for whom testing was performed following a negative US. CT/MRI and US were concordant in 25 patients (78% of the 32 patients for whom both studies were performed).

Combinations of Diagnostic Criteria

Diagnostic sensitivity of combinations of accepted diagnostic criteria are detailed in **Table III**. Of note, combinations of criteria performed no better than individual criteria alone.

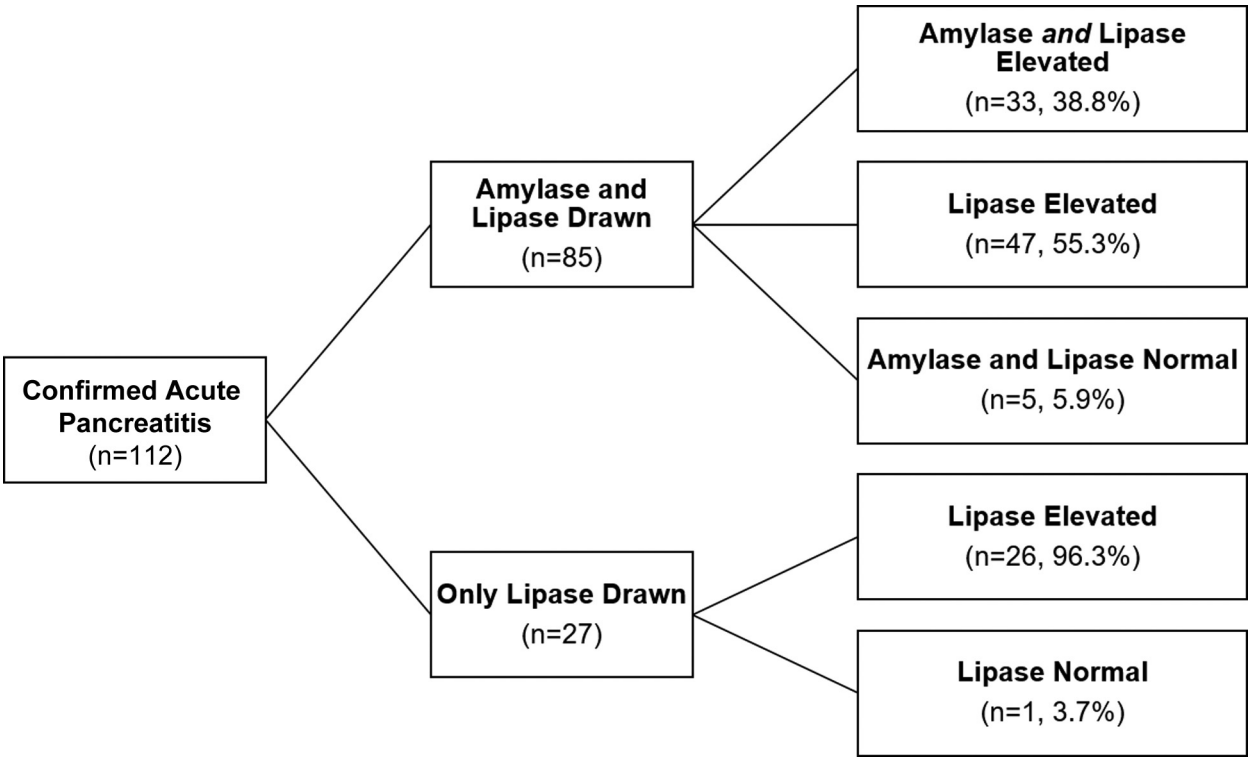

Figure 1. Frequency of lipase or amylase obtained in acute pancreatitis.

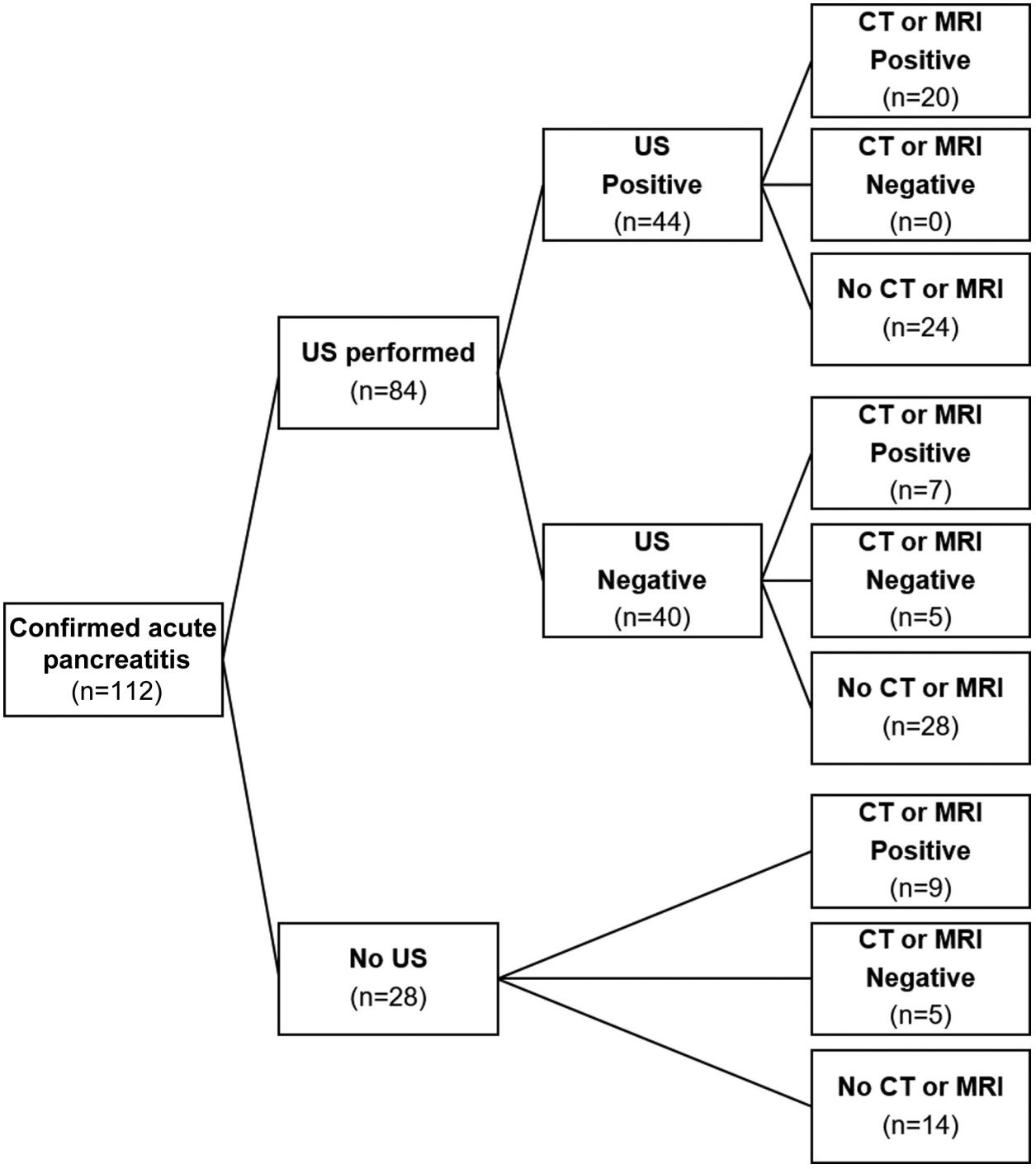

Figure 2. The frequency of CT, MRI or US performed in Acute Pancreatitis patients.

Discussion

In this cross-sectional study, based on a population enrolled in a prospective clinical database, we sought to define the sensitivity of each of the accepted diagnostic criteria (characteristic symptoms, laboratory values, and imaging) for the diagnosis of acute pancreatitis in pediatrics. Our results demonstrate that serum lipase is a more sensitive biomarker for acute pancreatitis in children than serum amylase, a finding that is consistent with the broader literature, but here confirmed from a prospectively collected registry.[3,12,18,19] Relative to serum amylase, serum lipase elevations are less time-dependent during the disease process of acute pancreatitis due to their delayed peak and longer duration of elevation, whereas serum amylase peaks and normalizes more rapidly.[8,12,20] Furthermore, serum amylase may be elevated on the basis of nonpancreatic pathology more frequently than lipase.[7,21,22] Although serum lipase is a highly sensitive

Table IV. Imaging findings of acute pancreatitis within 1 week of attack

Findings*	US	CT	MRI
Pancreatic edema	56.8% (42/74)	75.0% (27/36)	88.2% (15/17)
Peripancreatic edema	45.9% (34/74)	72.2% (26/36)	76.5% (13/17)
Acute peripancreatic fluid collection	22.2% (16/72)	52.8% (19/36)	47.1% (8/17)
Pancreatic necrosis	N/A	12.5% (4/32)	7.7% (1/13)
Peripancreatic necrosis	N/A	3.2% (1/31)	0%
Hemorrhage	N/A	3.3% (1/30)	0%
Pancreatic duct dilation	20.5% (15/73)	6.7% (2/30)	29.4% (5/17)

N/A, not available.
*Not all findings could be assessed on every examination, accounting for denominators less than the total.

biomarker for acute pancreatitis in children, elevated pancreatic enzymes may not be present in all cases of acute pancreatitis. From our patient cohort, 5.4% of patients did not meet diagnostic criteria by lipase elevation and/or amylase elevation but met criteria based on signs/symptoms and imaging findings supportive of acute pancreatitis.

Although our results indicate a relatively high sensitivity for cross-sectional imaging, there was likely selection bias of patients undergoing CT or MRI. Collectively, imaging had a 34% false-negative rate for acute pancreatitis. This moderate diagnostic performance is consistent with previous reports. Specifically, US has been shown to be 62%-67% sensitive in diagnosing acute pancreatitis in adults, whereas in pediatric patients, pancreatic findings suggestive of acute pancreatitis are present in roughly 30%-50% of cases.[3,4,14,18] CT findings of acute pancreatitis are present in pediatric patients approximately 47%-75% of the time.[4,7,14] Although US traditionally has been the initial imaging modality of choice due to wide availability, ease of use, and the absence of sedation needs or ionizing radiation exposure, our data as well as those of previous studies suggest that there are significant diagnostic limitations for this modality and call into question the use of US as the first-imaging modality of choice for suspected acute pancreatitis in children. Further prospective studies are needed to define the optimal imaging paradigm for the child with suspected acute pancreatitis.

Our study has several limitations. First, although enrollment in the registry from which this population is drawn was prospective, there was no standardization of the management of the enrolled patient population. Because of this, both amylase and lipase were not measured in all patients, imaging was not consistently obtained as part of a diagnostic algorithm, and the used imaging modality was not standardized. We have the leverage of standardizing management at our institution through an admission order set that is used hospital wide, and hence we see that imaging was highly available within admissions of acute pancreatitis. However, orders even from the order set remain provider dependent, and studies were not obtained in all patients systematically, which limits our ability to compare biochemical and imaging markers, albeit, significant differences were identified. Second, because this study is based on a population of patients enrolled on the basis of meeting established diagnostic criteria for acute pancreatitis, there is an inherent assumption in our study that these criteria captured all patients with acute pancreatitis during the time period of interest. It is feasible that occurrences of acute pancreatitis may have been missed if they did not meet diagnostic criteria. Third, because the study population was derived from a prospective registry of patients with confirmed acute pancreatitis, we are unable to report the specificity of the diagnostic elements. In addition, we only evaluated the first episode of acute pancreatitis and did not investigate the utility of laboratory testing or imaging in acute recurrent pancreatitis or chronic pancreatitis, as these populations may present differently. Finally, it is possible that our evaluation of imaging within 1 week of presentation missed detection of milder acute pancreatitis episodes, especially if obtained at either extreme of the 7-day period. This may have contributed to the 34% false-negative rate. We conclude that serum amylase has a limited utility in the diagnosis of pediatric acute pancreatitis and imaging paradigms for the diagnosis of pediatric acute pancreatitis need to be re-evaluated. ■

Submitted for publication Mar 27, 2019; last revision received Jun 7, 2019; accepted Jun 7, 2019.

Reprint requests: Maisam Abu-El-Haija, MD, Cincinnati Children's Hospital Medical Center, 3333 Burnet Ave, MLC 2010, Cincinnati, OH 45229. E-mail: Maisam.Haija@cchmc.org

References

1. Lopez MJ. The changing incidence of acute pancreatitis in children: a single-institution perspective. J Pediatr 2002;140:622-4.
2. Morinville VD, Barmada MM, Lowe ME. Increasing incidence of acute pancreatitis at an American pediatric tertiary care center: is greater awareness among physicians responsible? Pancreas 2010;39:5-8.
3. Park AJ, Latif SU, Ahmad MU, Bultron G, Orabi AI, Bhandari V, et al. A comparison of presentation and management trends in acute pancreatitis between infants/toddlers and older children. J Pediatr Gastroenterol Nutr 2010;51:167-70.
4. Kandula L, Lowe ME. Etiology and outcome of acute pancreatitis in infants and toddlers. J Pediatr 2008;152:106-10. 10.e1.
5. Hornung L, Szabo FK, Kalkwarf HJ, Abu-El-Haija M. Increased burden of pediatric acute pancreatitis on the health care system. Pancreas 2017;46:1111-4.
6. Pant C, Deshpande A, Olyaee M, Anderson MP, Bitar A, Steele MI, et al. Epidemiology of acute pancreatitis in hospitalized children in the United States from 2000-2009. PLoS One 2014;9:e95552.
7. Bai HX, Lowe ME, Husain SZ. What have we learned about acute pancreatitis in children? J Pediatr Gastroenterol Nutr 2011;52:262-70.
8. Abu-El-Haija M, Lin TK, Palermo J. Update to the management of pediatric acute pancreatitis: highlighting areas in need of research. J Pediatr Gastroenterol Nutr 2014;58:689-93.
9. Morinville VD, Husain SZ, Bai H, Barth B, Alhosh R, Durie PR, et al. Definitions of pediatric pancreatitis and survey of present clinical practices. J Pediatr Gastroenterol Nutr 2012;55:261-5.
10. Coffey MJ, Nightingale S, Ooi CY. Diagnosing acute pancreatitis in children: what is the diagnostic yield and concordance for serum pancreatic

enzymes and imaging within 96 h of presentation? Pancreatology 2014;14:251-6.
11. Agarwal NPC, Sivaprasad AV. Evaluating tests for acute pancreatitis. Am J Gastroenterol 1990;85:356-66.
12. Gomez D, Addison A, De Rosa A, Brooks A, Cameron IC. Retrospective study of patients with acute pancreatitis: is serum amylase still required? BMJ Open 2012;2.
13. Apple FBP, Preese L, Eastep S, Bilodeau L, Heiler G. Lipase and pancreatic amylase activities in tissues and in patients with hyperamylasemia. Am J Clin Pathol 1991;95:610-4.
14. Werlin SL, Kugathasan S, Frautschy BC. Pancreatitis in children. J Pediatr Gastroenterol Nutr 2003;37:591-5.
15. Abu-El-Haija M, Kumar S, Szabo F, Werlin S, Conwell D, Banks P, et al. Classification of acute pancreatitis in the pediatric population: clinical report from the NASPGHAN Pancreas Committee. J Pediatr Gastroenterol Nutr 2017;64:984-90.
16. Sweeny KF, Lin TK, Nathan JD, Denson LA, Husain SZ, Hornung L, et al. Rapid progression of acute pancreatitis to acute recurrent pancreatitis in children. J Pediatr Gastroenterol Nutr 2019;68:104-9.
17. Abu-El-Haija M, Lin TK, Khan S, Fei L, Thompson T, Nathan JD. Predictive biomarkers for acute gallstone pancreatitis in the pediatric population. Pancreatology 2019, In press.
18. Darge K, Anupindi S. Pancreatitis and the role of US, MRCP and ERCP. Pediatr Radiol 2009;39(suppl 2):S153-7.
19. Shi Y, Liu Y, Liu YQ, Gao F, Li JH, Li QJ, et al. Early diagnosis and severity assessment of acute pancreatitis (AP) using MR elastography (MRE) with spin-echo echo-planar imaging. J Magn Reson Imaging 2017;46:1311-9.
20. Tenner S, Baillie J, DeWitt J, Vege SS , American College of Gastroenterology. American College of Gastroenterology guideline: management of acute pancreatitis. Am J Gastroenterol 2013;108:1400-15. 1416.
21. Spector D, Perry Z, Shah S, Kim JJ, Tarnoff ME, Shikora SA. Roux-en-Y gastric bypass: hyperamylasemia is associated with small bowel obstruction. Surg Obes Relat Dis 2015;11:38-43.
22. Lankisch PG, Doobe C, Finger T, Lubbers H, Mahlke R, Brinkmann G, et al. Hyperamylasaemia and/or hyperlipasaemia: incidence and underlying causes in hospitalized patients with non-pancreatic diseases. Scand J Gastroenterol 2009;44:237-41.

50 Years Ago in *The Journal of Pediatrics*

Fifty-Two Forms of Childhood Cancer, United States Mortality Experience, 1960-1966

Miller R. J Pediatr 1969;75:685-9

Fifty years ago, Miller reviewed childhood cancer mortality with the important observation that adolescents demonstrate distinct epidemiology. With increasing age, increasing frequency of deaths because of bone cancer, Hodgkin disease, and glioblastoma and decreasing frequency of deaths because of embryonal tumors such as neuroblastoma, nephroblastoma, and retinoblastoma was observed.

Despite tremendous progress, cancer remains the leading disease-related cause of death among children in the US. Improvements in survival of adolescents have failed to keep pace with improvements in younger patients.[1] Adolescents have been recognized as distinctive from their younger and older counterparts with cancer from both a biologic and psychosocial standpoint. In addition to disparities by age, substantial survival disparities by race, ethnicity, and socioeconomic status exist.[2]

Miller detected the first hints of these disparities in noting that white patients with leukemia had a peak in mortality at 4 years of age that was not detected in minority patients. Recent studies have further elucidated that the distribution of black children with acute lymphoblastic leukemia (ALL) is skewed toward adolescence. Among those 1-9 years of age, mortality among black children with ALL is higher compared with white children. These differences translate to a 49% higher mortality risk among black patients with ALL.[3] In acute myeloid leukemia, black and Hispanic children have lower survival, which seems less linked to age disparities.[4]

Although we have come far in understanding the biological roots of the differences in cancer by age, we have made less progress in understanding the etiology of other disparities. Ongoing attention to not only cancer biology, but other mechanisms of disparities, including the roles of access to care and social determinants of health, in pediatric cancer disparities may further explain persistent age and racial/ethnic disparities.

Lena Winestone, MD, MSHP
Michael D. Cabana, MD, MPH
University of California, San Francisco
San Francisco, California

References

1. Bleyer A, Barr R. Cancer in young adults 20 to 39 years of age: overview. Semin Oncol 2009;36:194-206.
2. Bhatia S. Disparities in cancer outcomes: lessons learned from children with cancer. Pediatr Blood Cancer 2011;56:994-1002.
3. Kadan-Lottick NS, Ness KK, Bhatia S, Gurney JG. Survival variability by race and ethnicity in childhood acute lymphoblastic leukemia. JAMA 2003;290:2008-14.
4. Patel MI, Ma Y, Mitchell BS, Rhoads KF. Understanding disparities in leukemia: a national study. Cancer Causes Control 2012;23:1831-7.

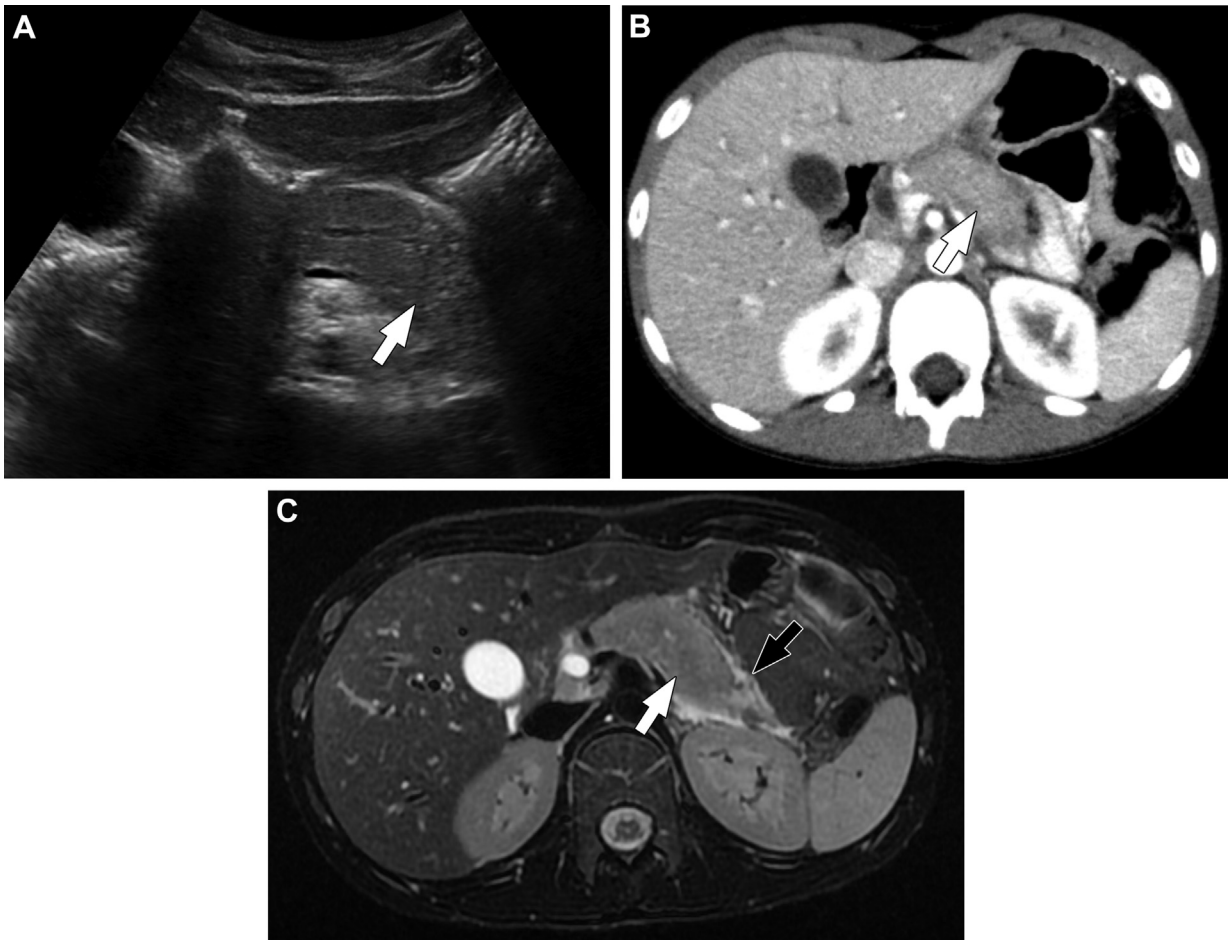

Figure 3. **A**, Transverse US image shows an edematous/enlarged pancreas (*arrow*) with pancreatic duct dilation and mild peripancreatic edema; **B**, Axial CT image in the same patient shows an edematous/enlarged pancreas (*arrow*) with peripancreatic edema (*white arrow*). **C**, Axial T2-weighted fat saturated MR image in the same patient shows an edematous pancreas (appearing T2-weighted hyperintense; *white arrow*) with peripancreatic edema (*black arrow*).

Table II. Comparison of patient demographics depending on presenting symptoms

Criteria met for diagnosis of acute pancreatitis	n	Median age, y (IQR)	Sex	BMI*
Laboratory testing and imaging (regardless of clinical)	54	14.2 (9.87-16.2)	34 (63%) male 20 (37%) female	Underweight: 7 (13%) Normal: 29 (54%) Overweight: 4 (7%) Obese: 14 (26%)
Laboratory testing and clinical† (no or negative imaging)	52	12.6 (9.09-15.4)	21 (40%) male 31 (60%) female	Underweight: 5 (10%) Normal: 27 (52%) Overweight: 6 (12%) Obese: 5 (10%)
Imaging and clinical (ie, no testing or negative laboratory tests)	6	12.6 (11.5-13.1)	2 (33%) male 4 (67%) female	Underweight: 0 (0%) Normal: 4 (67%) Overweight: 2 (33%) Obese: 0 (0%)
P value		.294	.053	.292

BMI, body mass index.
*No BMI calculated for patients <2 years or >20 years of age.
†BMI data not available for 52 patients, as height not obtained at time of attack.

Table III. Sensitivity of individual diagnostic criteria and combinations of diagnostic criteria for acute pancreatitis

Criteria	Sensitivity ratio	Sensitivity	95% CI (Clopper–Pearson)
Single criterion			
Clinical	111/112	0.99	(0.95-1.00)
Laboratory	106/111	0.95	(0.90-0.99)
Imaging	60/98	0.61	(0.51-0.71)
Combination of criteria			
Any 2 of 3 (clinical, laboratory, imaging)	112/112	1.00	(0.97-1.00)
Laboratory and clinical	105/111	0.95	(0.89-0.98)
Clinical and imaging	59/98	0.60	(0.50-0.70)
Laboratory and imaging	54/97	0.56	(0.45-0.66)