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Clinical Communications: Pediatric

DIAGNOSING ACUTE HEART FAILURE IN THE PEDIATRIC EMERGENCY DEPARTMENT USING POINT-OF-CARE ULTRASOUND

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□ **Abstract—Background: Acute heart failure (AHF) in children is associated with significant disease burden with high rates of morbidity, mortality, and resource utilization. These children often present to the emergency department with clinical features that mimic common childhood illnesses. Cardiac point-of-care ultrasound (POCUS) can be an effective tool for rapidly identifying abnormal cardiac function. Case Reports: This case series documents 10 children presenting with AHF between 2016 and 2019 and demonstrates how pediatric emergency physicians used cardiac POCUS to expedite their diagnosis, management, and disposition. All cardiac POCUS was performed before comprehensive echocardiograms were completed. One case is described in detail; the other cases are summarized in a Table. Why Should an Emergency Physician Be Aware of This?: Early recognition of AHF is critical to reduce pediatric morbidity and mortality. With proper training, cardiac POCUS can be an effective adjunct and should be considered for the early diagnosis and treatment of infants and children with AHF. © 2021 Elsevier Inc. All rights reserved.**

□ **Keywords—Pediatrics; Point-of-care ultrasound; Heart failure; Cardiac ultrasound**

Streaming video: Twenty-three brief real-time video clips that accompany this article are available in streaming video at www.journals.elsevierhealth.com/periodicals/jem. Click on Video Clips 1–23.

INTRODUCTION

Heart failure (HF) is a clinical syndrome that results from impaired ventricular filling or ejection of blood, leading to inadequate end-organ perfusion (1,2). Pediatric HF is relatively uncommon compared with adults but is associated with significant disease burden in terms of morbidity, mortality, and related health care costs (2). It is responsible for at least 11,000 hospitalizations per year (2–6). Children hospitalized with acute heart failure (AHF) have a 20-fold increase in mortality rates compared with those hospitalized without AHF, and often have several comorbidities (e.g., respiratory failure, renal failure, and dysrhythmia) (2,3). Diverse clinical features and the wide range of presentations often complicate or delay the diagnosis of AHF, which can have devastating consequences (7).

Cardiac point-of-care ultrasound (POCUS) can be used to determine the presence of cardiac activity, pericardial effusion, and cardiac tamponade, while also assessing cardiac function (8). Cardiac POCUS effectively evaluates heart function and correlates with cardiologist-performed echocardiograms (9–11). Few studies demonstrate the benefits of cardiac POCUS in pediatric patients with AHF. We present 10 cases of AHF in which cardiac POCUS expedited the diagnosis and guided treatment.

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Case 1 is presented in detail, and all 10 cases are summarized in Table 1 with associated videos (Videos 1–23).

In this case series, we identified patients ≤ 21 years old presenting with AHF between 2016 and 2019 from our POCUS database. Then we reviewed patient records and abstracted data for demographics, comorbidities, presenting complaints, triage vital signs, laboratory tests, radiology reports, and final attending cardiologist readings from electrocardiograms (ECGs) as well as echocardiograms, and the final diagnoses. POCUS examination included cardiac images in the parasternal long, parasternal short, apical four-chamber, and subxiphoid views, and inferior vena cava images in the longitudinal view. Images were acquired by POCUS-credentialed pediatric emergency medicine (PEM) physicians using the M-turbo™ (Sonosite; Bothell, WA), Edge II™ (Sonosite), and Venue Go™ (GE Healthcare SA; Athens, Greece) ultrasound machines. Findings and interpretations were detailed in Qpath™ (Telexy; Maple Ridge, British Columbia, Canada), with documentation completed by the POCUS-credentialed PEM physician at the time of the examination. All POCUS examinations were obtained before comprehensive echocardiograms were performed. The final diagnoses were acquired from discharge summaries and International Classification of Diseases, Tenth Revision discharge codes. This study was approved by the local institutional review board and was compliant with the Health Information Portability and Accountability Act. Written informed consent was waived.

CASE REPORTS

A previously healthy 5-year-old boy presented to the Emergency Department (ED) with a 4-day history of fever, anorexia, cough, sore throat, myalgia, nausea, vomiting, and generalized abdominal pain. He had been diagnosed at another facility with streptococcal pharyngitis and prescribed amoxicillin 3 days prior. His symptoms worsened, prompting an evaluation at our ED. Initial triage vital signs included a heart rate of 160 beats/min, respirations of 38 breaths/min, blood pressure of 116/64 mm Hg, oxygen saturation of 100% on room air, and temperature of 39.2°C (102.6°F). Ibuprofen was administered, and the patient was placed in the waiting room. Two hours later, he was roomed in the ED, and repeat vital signs revealed new hypotension (blood pressure 71/35 mm Hg) despite improving tachycardia (126 beats/min) and tachypnea (30 breaths/min), with a normal temperature (37.3°C) and oxygen saturation (98%). On examination, he was lethargic, pale, and dehydrated; he lacked hepatomegaly or a gallop. The initial impression was of decompensated shock. Laboratory tests were obtained, and he received parenteral antibiotics and a 20-mL/kg isotonic fluid bolus. Cardiac POCUS (Figure 1, Videos 1–3) was performed, concurrently with initial stabilization, and demonstrated mildly reduced left ventricular function (ejection fraction approximately 40–49%), prompting immediate cessation of the initial fluid bolus (12). An epinephrine infusion was ordered, and the cardiac intensive care unit (CICU) team was consulted within 40 min of initial ED evaluation. Chest radiography demonstrated a normal cardiac silhouette and pulmonary vascular markings. Initial laboratory results were significant for elevated lactate levels (3.1 mmol/L). Troponin (0.485 ng/mL) and brain natriuretic peptide (3820 pg/mL) were found to be elevated; an ECG showed sinus rhythm, possible biventricular hypertrophy, and nonspecific ST/T-wave abnormalities. Serial cardiac POCUS examinations demonstrated progressive worsening of ventricular function to moderately depressed ventricular function (ejection fraction approximately 30–39%), leading to admission to the CICU for cardiogenic shock (12).

A comprehensive echocardiogram in the CICU subsequently confirmed the cardiac POCUS examination findings of moderately to severely depressed left ventricular systolic function. He was weaned off the epinephrine infusion and discharged after 8 days with a diagnosis of myocarditis on standard oral HF therapies including enalapril and spironolactone. A 1-week follow-up echocardiogram demonstrated normalization of his left ventricular function.

DISCUSSION

Every 6-h delay in treatment of acute decompensated HF in adults has a 6.8% increased risk of death (13). Although extrapolating these data to pediatric patients is difficult, making a rapid and accurate diagnosis of AHF may allow for early initiation of life-sustaining or symptom-relieving therapies. Time to initiation of important therapies for AHF, such as vasoactive agents or diuretics, in children, has been shown to be significantly reduced when initiated in the ED, compared with the ICU or inpatient units (7). A missed diagnosis of new-onset HF in children may delay providing appropriate therapies by a median of 3 days (14). This case series demonstrates that cardiac POCUS can play a role in the early recognition of AHF, helping to expand the differential diagnosis and guide therapy, and has good correlation with echocardiograms (9–11). Most patients in our series received Cardiology-performed comprehensive echocardiography as an inpatient; POCUS superseded the need for performing a comprehensive echocardiogram in the ED in 80% of cases in our case series and led to expedited recognition of AHF and initiation of ED interventions.

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Table 1. Pediatric Heart Failure Cases

Case	Age	Gender	PMH	Chief Complaint/ Parental Concern	Initial Vital Signs*	Pre-POCUS Interventions†	POCUS Findings‡	Post-POCUS ED Interventions†	Electrocardiogram	Imaging	Labs	Final Diagnosis
1	5 y	Male	Previously healthy	Body aches, decreased appetite, fever, throat pain	HR 160 RR 38 BP 116/64 O_2 97% 102.6°F	NSB	Cardiac: diminished ventricular function, no PCE	IV fluids stopped, epinephrine, calcium gluconate, sodium bicarbonate	NSR, prominent mid-precordial voltage, possible BVH, nonspec ST and T wave abn	CXR: normal	BNP 3820 pg/mL Troponin 0.495 ng/mL	Myocarditis (suspected viral etiology)
2	8 days	Male	Previously healthy	Cyanosis, grunting	HR 152 RR 54 BP 101/53 O_2 70% (15L NRB) 95.5°F	EMS: D10 bolus ED: NRB, D10 bolus	Cardiac: impaired global ventricular function, dilated RV, flattened interventricular septum, no PCE, normal IVC	Epinephrine, sodium bicarbonate, prostaglandin, intubation	N/A	Post-intubation CXR: low lung volumes, bilateral central predominant airspace opacities, cardiomegaly	BNP N/A Troponin N/A	Biventricular non-compaction cardiomy-opathy
3	3 weeks	Male	Previously healthy	Tach, tachypnea	HR 155 RR 39 BP 104/75 O_2 89% 98.6°F	Vagal maneuvers, adenosine x4 aborted SVT	Cardiac: poor cardiac function, small PCE	Milrinone, adenosine 5th dose aborted 2nd SVT episode, calcium gluconate, sodium bicarbonate, epinephrine, magnesium, intubation during 3rd SVT episode, furosemide, CPR and defibrillation for ventricular fibrillation, VA ECMO initiated in ED	HR 269, SVT	CXR: moderately severe cardiomegaly, severe bilateral pulmonary edema, small pleural effusion	BNP N/A Troponin N/A	SVT induced cardiogenic shock and cardiogenic arrest
4	3 months	Male	Hyper-insulinemia, hypo-thyroidism, cardiomegaly with normal cardiac function	Grunting	HR 144 RR 41 BP 115/84 O_2 91% 97.7°F	Bubble CPAP, NSB	Cardiac: enlarged RV and RA, decreased RV function, preserved LV function, no PCE	Hydrocortisone, furosemide, intubation	N/A	CXR: new interstitial edema, persistent cardiomegaly	BNP 7970 pg/ml Troponin N/A	Pulmonary HTN with right-sided heart failure from diazoxide home medication, MSSA bacteremia

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Table 1. (*continued*)

5	16 months	Male	Previously healthy	Respiratory distress	HR 156 RR 44 BP 114/69 O_2 95% 99.1°F	HFNC, NSB	Cardiac: impaired global ventricular function, no PCE	IV fluids stopped, furosemide, comprehensive echo, milrinone	NSR, RVH, ST abn, T wave inversion lateral leads, borderline prolonged QT	CXR: bilateral pleural effusions and opacities suggestive of pulmonary edema, enlarged hila and cardiac silhouette	BNP 4977 pg/ml Troponin 0.255 ng/ml	Parovirus-induced myocarditis
6	3 y	Male	Diamond-Blackfan Syndrome on steroid taper	Fever, possible meningitis	HR 153 RR 38 BP 123/79 O_2 96% 98.5°F	HFNC, NSB	Cardiac: dilated LA and LV, poor function, no PCE Thoracic: bilateral trace pleural effusions, bilateral B-lines	Hydrocortisone, epinephrine, intubation	Sinus tach	CXR: perihilar pulmonary edema, normal cardiomediastinal structures	BNP 39 pg/ml Troponin 8.11 ng/ml	Fulminant enterovirus-induced myocarditis
7	8 y	Female	Previously healthy	Dizziness, syncope	HR 137 RR 34 BP 84/61 O_2 100% (6L SFM) 97°F	EMS: albuterol	Cardiac: poor function, no PCE, plethoric IVC Thoracic: bilateral B-lines	Comprehensive echo, milrinone	Sinus tach, LAD, possible RVH, nonspec ST and T wave abn	CXR: tiny fluid in R horizontal fissure otherwise clear lungs, normal heart size	BNP 1412 pg/ml Troponin 3.36 ng/ml	Myocarditis (suspected viral etiology)
8	14 y	Male	Previously healthy	Palpitations	HR 231 RR 20 BP 108/53 O_2 100% 98.1°F	Adenosine, verapamil	Cardiac: impaired global ventricular function, no PCE	Verapamil	Wide QRS tach, LAD, RBBB, superior axis	CXR: normal	BNP 17 pg/ml Troponin 0.816 ng/ml	Acute combined diastolic and systolic heart failure, wide complex tachycardia, previously undiagnosed Kawasaki's Disease w/ R and L coronary artery dilatation

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Table 1. (*continued*)

9	14 y	Male	Previously healthy	Abdominal pain, diarrhea, fever, weakness	HR 200 RR 28 BP 90/58 O_2 97% 99.5°F	NSB	Cardiac: reduced global ventricular function, no PCE	Epinephrine	Sinus tach, rightward axis, Q wave in aVL, STE	CXR: low lung volumes with bilateral parahilar streaky and subtle nodular opacities, normal cardio-mediastinal structures CT chest, PE protocol: no evidence of PE, multifocal groundglass and solid-appearing nodules seen in all lobes	BNP N/A Troponin 21.045 ng/ml	Rhinovirus-induced myocarditis
10	21 y	Male	S/p orthotopic heart transplant with normal biventricular function, lost to follow-up for 1 year	Abdominal pain, chest pain, shortness of breath	HR 101 RR 16 BP 104/67 O_2 94% 98.5°F	N/A	Cardiac: severely depressed ventricular function, no PCE	Pt emergently transferred to CICU prior to medication administration	Sinus tach, 1st degree AV block, RBBB, nonspec ST and T wave abn	CXR: stable enlargement of cardiac silhouette, bilateral lower lobe atelectasis	BNP 1608 pg/ml Troponin 0.018 ng/ml	Acute combined diastolic and systolic heart failure, biopsy negative transplant rejection

*All oxygen saturations obtained at room air unless otherwise specified.
†Antibiotics, antiemetics, antivirals, pain medications, and rapid sequence intubation medications given when clinically indicated.
‡Each case has associated point-of-care ultrasound videos.
PMH = past medical history; POCUS = point-of-care ultrasound; ED = emergency department; HR = heart rate; RR = respiratory rate; BP = blood pressure; O_2 = oxygen saturation;°F = Fahrenheit; NSB = normal saline bolus; PCE = pericardial effusion; IV = intravenous; NSR = normal sinus rhythm; BVH = biventricular hypertrophy; Nonspec = nonspecific; abn = abnormalities; CXR = chest radiograph; BNP = B-type natriuretic peptide; NRB = non-rebreather mask; EMS = emergency medical services; RV = right ventricle; IVC; inferior vena cava; Tach = tachycardia; N/A = not applicable; SVT = supraventricular tachycardia; VA = veno-arterial; ECMO = extracorporeal membrane oxygenation; CPR = cardiopulmonary resuscitation; CPAP = continuous positive airway pressure; RV = right ventricle; LV = left ventricle; HTN = hypertension; MSSA = methicillin-susceptible *Staphylococcus aureus*; LA = left atrium; HFNC = high-flow nasal cannula; echo = echocardiogram; RVH = right ventricle hypertrophy; SFM = simple face mask; LAD = left axis deviation; RBBB = right bundle branch block; STE = ST segment elevation; CT = computed tomography; PE = pulmonary embolism; S/p = status post; CICU = cardiac intensive care unit; AV = atrioventricular.

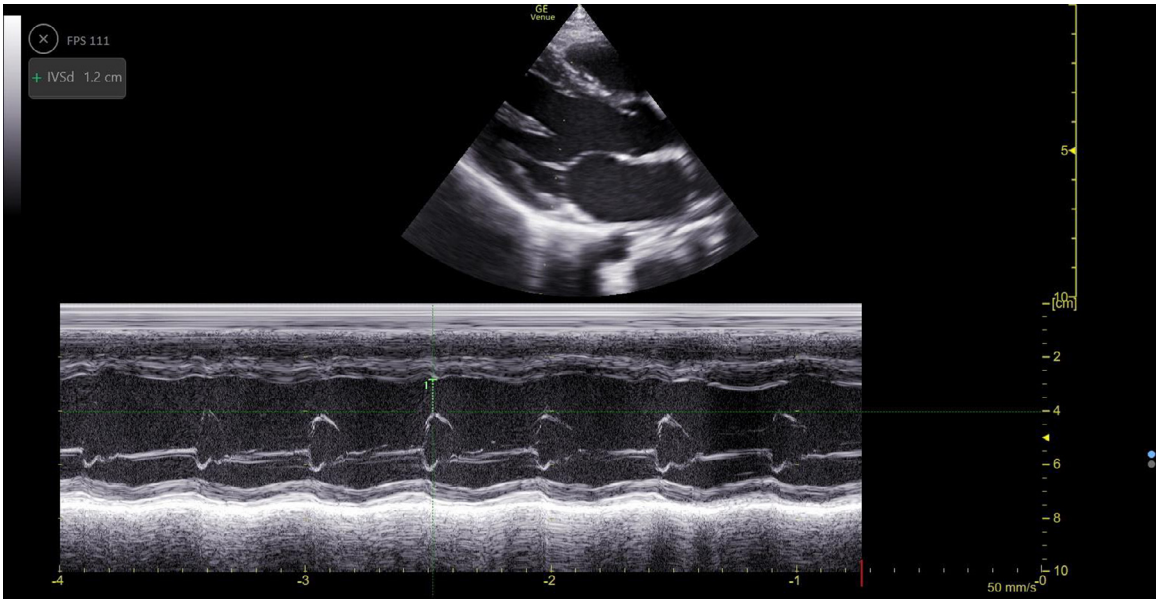

Figure 1. Cardiac point-of-care ultrasound examination: Using the E-Point Septal Separation method, this image displays the M-Mode of the cardiac parasternal long axis view with mitral valve at maximum excursion far away from the interventricular septum demonstrating depressed cardiac function in a 5-year-old male (24–26).

No single history, symptom, or physical examination variable can determine the likelihood of AHF in adults (15). Our case series evaluated some of the historical features and physical findings of AHF. Patient ages ranged from 8 days to 21 years, with a predominance of males (90%) and previously healthy patients (70%). No patient had a history of abnormal cardiac function prior to presentation, including one patient with pulmonary atresia status post orthotopic heart transplant with normal biventricular systolic function on his last comprehensive echocardiogram. Chief complaints varied, and many symptoms were not referable to a cardiac etiology. However, all patients had at least one abnormal vital sign upon presentation (16,17). Tachycardia was the most common abnormality (70%), followed by hypoxemia (60%), defined as saturations $\leq$ 95% (based on consensus by authors). Although vital signs aid triage of patients based on the severity of the illness, they are nonspecific findings that lack sensitivity or specificity.

Electrocardiograms

ECG may show only nonspecific or subtle changes, but provides important data relevant to a diagnosis of AHF such as decreased voltages (myocarditis or pericardial effusion), Q waves in leads I and AVL (anomalous left coronary artery), and rhythm disturbances. In our case series, ECG was performed in the ED in 8 patients. One patient with fulminant viral myocarditis had sinus tachycardia (Case 6) on ECG, without other abnormalities. If this case was excluded for isolated sinus tachycardia, then 7 of the 8 patients who received an ECG in the ED had abnormal ECGs, so an abnormal ECG may prompt the need for a cardiac POCUS examination.

Chest Radiographs

Nine of our patients received a chest radiograph. Macicek et al. reported that 98% of patients with AHF in their study had cardiomegaly revealed on chest radiographs (7). In our case series, 5 patients had signs of cardiomegaly, but only 3 were new-onset, and 2 demonstrated stable cardiomegaly. One possible explanation is that the patients in this case series were diagnosed earlier—using POCUS in the ED—than cases in the Macicek et al. study, and cardiomegaly had not yet developed on chest radiographs (7). Cases 1, 2, and 10, in particular, highlight this potential advantage of expediency in diagnosing AHF with POCUS. PEM physicians performed POCUS examinations in these three cases prior to obtaining the results of any other diagnostic testing, including an ECG, a chest radiograph, or laboratory tests, supporting evidence that POCUS is a useful examination tool for confirming the presence of AHF (15).

Laboratory Tests

Plasma B-type natriuretic peptide (BNP) is a hormone secreted by the heart in response to increased cardiac wall stress from volume or pressure overload. Studies in children have shown that BNP is a reliable test to diagnose HF (18–20). Three patients in our case series did not have

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a BNP drawn at the time of presentation but had increased values during their inpatient hospital course. Of seven BNP tests performed, five were abnormal in patients with confirmed AHF. The patients with normal BNPs had fulminant viral myocarditis and a previously undiagnosed Kawasaki's disease.

Troponin I is a serum marker released during myocardial injury and occasionally used to identify myocarditis. However, it is nonspecific and may occur in noncardiac pathologies such as sepsis or cardiac causes (e.g., tachydysrhythmias, cardiomyopathies, and myocarditis) (21–23). Therefore, an elevated troponin level would be helpful only in certain scenarios. Seven patients had a troponin level obtained, of which six were elevated. Five of these six patients who had increased troponin values had myocarditis, and one had undiagnosed Kawasaki's disease with bilateral coronary dilatation. Elevated troponin levels alone may not help diagnose AHF, but may help highlight the underlying etiology for AHF.

In adults, the cause of AHF is predominantly ischemic heart disease, whereas the etiology in the pediatric population is much more heterogeneous. Despite all the historical features, physical findings, and diagnostic testing at our disposal, POCUS may hold the most promise in early recognition of this critical diagnosis of AHF.

This case series has several limitations. It is a nonrandomized, convenience sample of 10 patients who received a POCUS examination based on the availability of a POCUS-credentialed PEM physician. We identified these patients through a retrospective review of our online image database, potentially introducing bias. Also, the patients were from a single institution at a quaternary care children's hospital, potentially limiting generalizability. Finally, this case series included only patients with no known heart dysfunction, so whether the findings can be generalized to known heart failure patients is unclear.

Several factors complicate the challenge of diagnosing AHF in pediatric patients: vague patient complaints mimicking common pediatric illnesses, nonspecific physical examination findings, poor sensitivity of chest radiographs, subtle findings on ECGs, delay in obtaining comprehensive echocardiography in the ED, and delay in subspecialist evaluation. Further studies are needed to determine the broader impact of POCUS in the pediatric ED.

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

Early recognition of AHF is critical to reduce pediatric morbidity and mortality. With proper training, cardiac POCUS can be an effective adjunct and should be considered for the early diagnosis and treatment of infants and children with AHF.

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SUPPLEMENTARY MATERIALS

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jemermed.2021.03.015.

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