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Table of Contents

Varicella Fetal Myocarditis – A Rare Finding in the Current Era of Universal Immunization

Cyndee Jocson, MD; Priscila Lisboa, MD; Antonia Gaudig, MD; Manoj Gupta, MD

Abstract

Fetal myocarditis is an extremely rare manifestation of maternal varicella infection, with few cases reported in literature. We describe a case of fetal myocarditis following maternal varicella infection in the second trimester, confirmed with IgM positivity. In this case, the fetal echocardiogram showed findings of myocarditis including depressed ventricular function, endocardial fibroelastosis (EFE), ectopic beats, and pericardial effusion. Fetal myocarditis carries a poor prognosis with significant morbidity. Interestingly, there was a complete recovery in this fetus, and the postnatal echocardiogram showed normal left ventricular systolic function and no evidence of EFE. To date, there have been no reports of maternal varicella-associated fetal myocarditis with complete recovery in the fetus. This case highlights the rare cardiac manifestations of maternal varicella infection and an extremely rare recovery of cardiac function with successful outcome. Early detection, maternal antiviral therapy, and preventive strategies are key components in managing this condition and improving outcomes. More research is needed to develop effective fetal interventions for in-utero myocarditis.

Introduction

Congenital varicella syndrome is rare, with a reported risk of 0.4% to 2% in the first and second trimesters, respectively.[1]

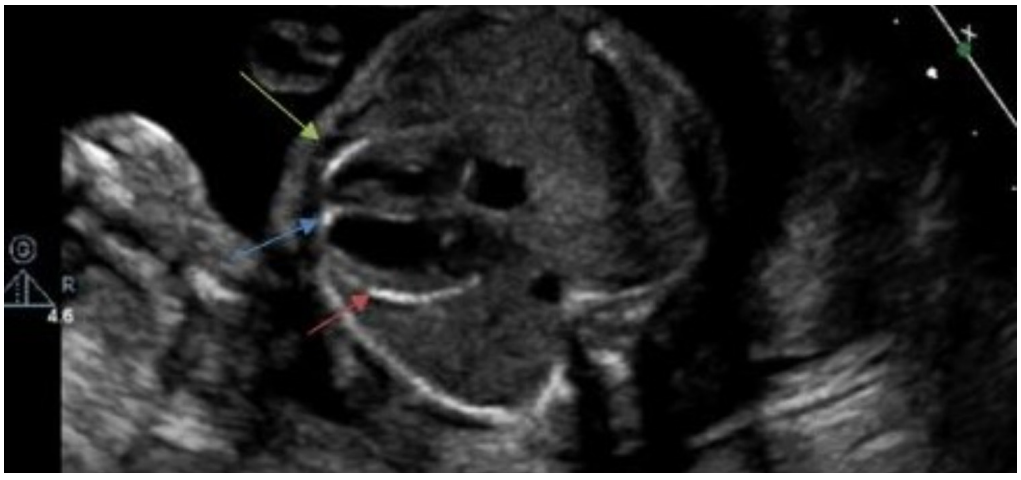


FIGURE 1 *Fetal apical 4 chamber view showing small apical pericardial effusion (green arrow), echo-bright left ventricular free wall (red arrow), echo-bright right ventricular free wall, and apical ventricular septum (blue arrow) suggestive of diffuse endocardial fibroelastosis. The echogenicity is also seen involving the left ventricular epicardial layer (red arrow).*

APRIL 2026

North American Edition Vol. 24 - Issue 4

TABLE OF CONTENTS

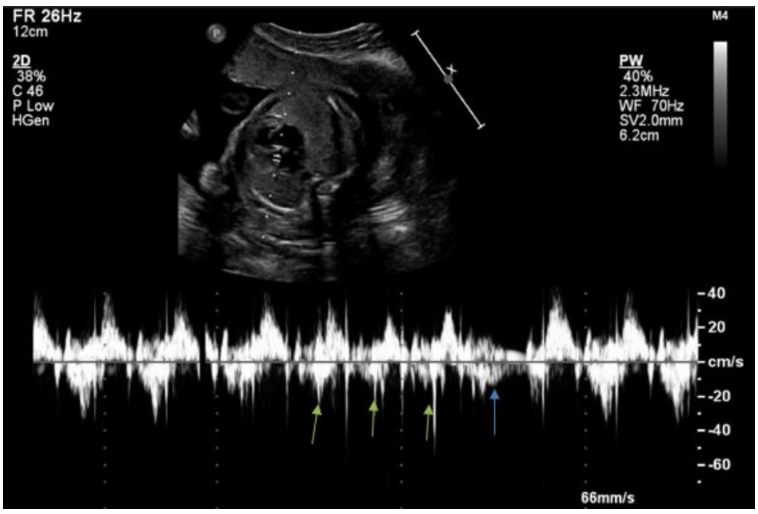


FIGURE 2 *Mitral inflow/outflow Doppler showing a three-beat run of atrial tachycardia (AT) (green arrow) followed by a blocked premature atrial conduction (blue arrow).*

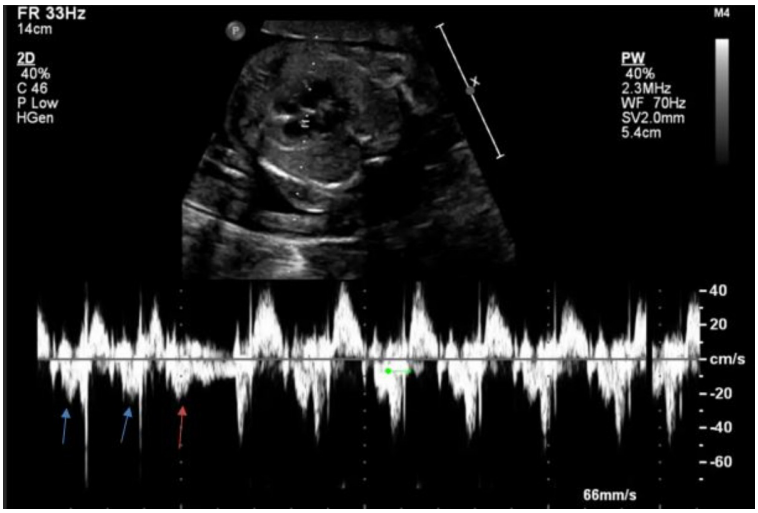


FIGURE 3 *Mitral/aortic inflow/outflow Doppler from four chamber showing atrial couplet (blue arrow) along with a blocked premature atrial contraction (red arrow).*

Fetal myocarditis is a serious condition characterized by inflammation of the fetal heart muscle. When associated with the varicella zoster virus (VZV) infection—more commonly known as chickenpox—the condition reflects the virus's capacity to cross the placenta and affect fetal organs, particularly in the setting of maternal primary infection during pregnancy. Fetal myocarditis is typically caused by infectious agents like coxsackie virus B, parvovirus B19, human herpes virus, Cytomegalovirus (CMV), VZV, as well as non-infectious causes including hereditary, autoimmune, and metabolic disorders. In most cases, it leads to an adverse outcome in the fetus.[1] We report a case of fetal myocarditis caused by varicella infection, confirmed with IgM positivity. In this case, the fetus showed echocardiographic findings of myocarditis with depressed ventricular function, endocardial fibroelastosis (EFE), fetal arrhythmia, and pericardial effusion, followed by gradual and complete recovery. Fetal myocarditis typically carries a poor prognosis with significant morbidity, but interestingly, there was a complete recovery in this fetus, and the postnatal echocardiogram showed normal left ventricular (LV) systolic function and no evidence of EFE. Fetal myocarditis is a rare manifestation of maternal varicella infection, with few cases reported in literature. The case is reported in view of the rarity of varicella-associated fetal myocarditis and even rarer complete spontaneous recovery during fetal life.

Case Presentation

A 29-year-old gravida 3, para 2 female was referred for fetal echocardiogram given concern for cardiac hypertrophy on routine anatomy scan at 21 weeks gestational age (GA). History revealed mild upper respiratory infection (URI) symptoms in the preceding week with low-grade fever, cough, and congestion. Fetal echocardiogram showed mildly depressed biventricular systolic function, globular-looking right ventricle (RV), mild RV hypertrophy, multiple foci of EFE in both right and left ventricular walls, and a small pericardial effusion (**Figure 1**). On mitral valve inflow/outflow Doppler interrogation, there were episodes of non-sustained atrial tachycardia, and both blocked and conducted atrial premature beats (**Figures 2 and 3**). Repeat echocardiogram at 23 weeks GA revealed similar EFE, moderate tricuspid regurgitation, qualitatively depressed RV and LV systolic function, and a small anterior pericardial effusion. Inflow/outflow Doppler interrogation showed frequent non-sustained runs of atrial tachycardia. Fetal echocardiogram was repeated every two weeks, and showed gradual improvement in biventricular systolic function, along with resolution of the ectopic beats. The pericardial effusion persisted for a few months, but at 31 weeks GA, there was complete resolution of the effusion with qualitatively normal biventricular systolic function.

The maternal past medical history was unremarkable, and she was not sure if she had ever been diagnosed with chickenpox as a child. She reported not having received the varicella vaccine. Although her VZV vaccination status was not available, there were no reports of VZV reactivation during pregnancy, even though pregnancy is sometimes considered an immunocompromised state. VZV titer was not drawn before the diagnosis of fetal myocarditis as there were no clinical stigmata of primary varicella infection, and there was no community outbreak in her neighborhood. She developed clinical respiratory illness along with nonspecific skin rash and skin redness a week before her scheduled anatomy scan. In the absence of clinical VZV infection, she was not given antiviral therapy or immunoglobulin therapy. VZV IgM was elevated at 1.13 (normal value <0.90), and VZV IgG was positive, indicating a recent varicella infection. She reported a flu-like illness during the second trimester but denied any rash. Workup for other infectious etiology was negative. Non-invasive prenatal testing was low risk. The glucose challenge test was normal. Rheumatologic work-up was negative for any autoimmune diseases. The rest of the maternal pregnancy course was uneventful. After a detailed discussion with the fetal cardiologist, infectious disease specialist, obstetrical and maternal-fetal medicine teams, the decision was made not to treat with varicella zoster immunoglobulin (VZIG) as the mother's symptoms had resolved, and the fetus was not in a hydropic state. The rest of the pregnancy was uneventful. A male infant

was born at 38 weeks GA via spontaneous vaginal delivery. Apgar scores were 5 and 9 at one and five minutes, respectively. He required continuous positive airway pressure for about one minute and was transferred to the neonatal intensive care unit for observation. He had no skin lesions or signs of neonatal varicella. His cardiac examination was normal, and electrocardiogram (ECG) showed sinus rhythm, nonspecific ST/T wave flattening, and no ectopic beats (**Figure 4A**). Echocardiogram showed mildly decreased LV systolic function with ejection fraction of 49% and no EFE. RV systolic function was normal. There was a small patent ductus arteriosus and a patent foramen ovale. The infant remained on room air and tolerated feeds throughout the hospital stay. Repeat echocardiogram on day 3 of life showed an ejection fraction of 61%, and he was discharged home in a hemodynamically stable condition. No medical therapy was started before discharge or at follow-up visits. Cardiomyopathy workup was not initiated in view of normalization of cardiac function. He was last seen by cardiology at three years of age and continues to do well. His last echocardiogram showed an ejection fraction of 60%. His ECG at three years showed no ST/T wave abnormalities (**Figure 4B**).

FIGURE 4A *ECG at age 3-years-old*
ECG showing normal ST/T waves.

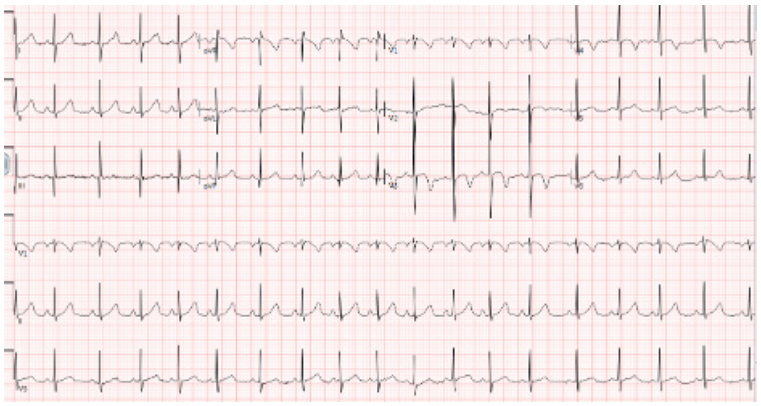
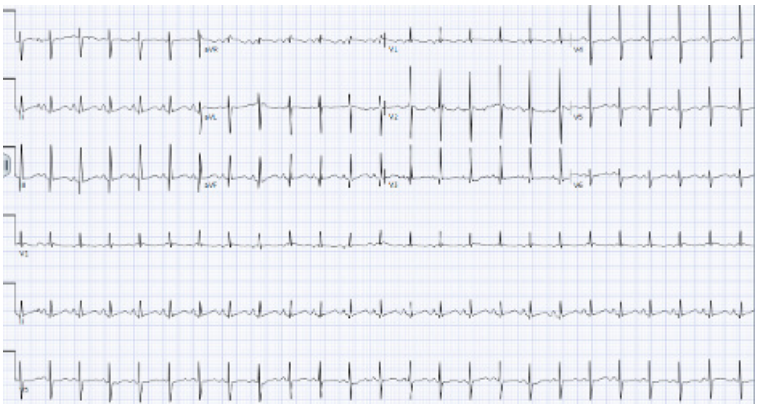


FIGURE 4B *ECG at day 1 of life*
ECG after birth showing nonspecific ST/T wave abnormalities and prolonged QTc secondary to T wave abnormalities.



Discussion

Although the incidence of congenital varicella has decreased due to vaccination, sporadic cases continue to occur in unvaccinated populations. Fetal myocarditis due to VZV is exceedingly rare but should be considered in cases of maternal varicella with abnormal fetal cardiac findings. Fetal myocarditis involves an inflammatory process of the myocardium, which may be acute or chronic with an infectious or non-infectious etiology. Infectious etiologies are typically viruses, including CMV, rubella, parvovirus B19, coxsackie virus, among others. Non-infectious etiologies include hereditary and metabolic diseases.[2] It is a rare finding in the fetus, and therefore, no large studies are available to determine the incidence or to establish a clear cause.[3] Fetal myocarditis may present as fetal arrhythmia, depressed function, pericardial effusion, fetal hydrops, or sudden fetal death. On fetal echocardiogram, fetal myocarditis typically shows as thickened, hyperechogenic, and poorly contractile myocardium with impaired ventricular systolic function.[2] Fesslova *et al*. reported the development of isolated dilated cardiomyopathy (CM) after infectious myocarditis. Infective cases in this study were entirely due to CMV and Coxsackie virus.[4] Similarly, in another manuscript by Pedra *et al*., biventricular dilatation and systolic dysfunction were found in subjects with confirmed CMV infection.[5] Hichijo and Morine wrote a case report describing parvovirus-related hydrops and terminal heart failure which led to fetal demise.[6] Herpes simplex virus infection has also been identified as a potential source of fetal CM, revealing a hyperechogenic myocardium with poor function of both ventricles.[2]

Varicella infection during pregnancy, particularly in the first and second trimesters, can lead to congenital varicella syndrome (CVS). The virus can cause direct cytopathic effects and inflammation in developing fetal tissues, including the myocardium. CVS presents with limb hypoplasia, cutaneous scars, microcephaly, cortical atrophy, chorioretinitis, and cataracts. Serious complications including pneumonia, encephalitis, nephritis, and although rare, myocarditis, have also been reported.[7] As a cardiotropic virus, it can infect endothelial cells and cardiomyocytes, triggering an inflammatory cascade, lymphocytic infiltration, and sometimes necrosis of myocardial tissue—hallmarks of viral myocarditis.

Varicella myocarditis, first described in 1953, has been rarely documented in children and has not previously been reported in a live fetus.[8] Documented cases include a 12-year-old girl who presented with myocarditis after a varicella exanthem, which resulted in severe ventricular dysfunction refractory to medical management needing cardiac transplantation.[9] Another described case is of a 15-year-old male who presented with sudden onset chest pain associated with ST-segment elevation and a positive troponin. His presentation was, at first, confused with acute myocardial infarction, but the diagnosis of varicella myocarditis was confirmed by the finding of raised IgM against varicella zoster on enzyme-linked immunosorbent assay. He had URI symptoms five days prior to presentation, and was vaccinated, however had contact with a child with chickenpox a few weeks prior. He was treated conservatively, and his condition rapidly improved with resolution of his pain and normalization of his ECG within two days.[10] Due to the rarity of this disease,

there are no standardized therapeutic guidelines for varicella myocarditis.

Conclusion

To date, there have been no reports of varicella infection leading to myocarditis in the live fetus. This case highlights the rare and dreaded cardiac manifestations of maternal varicella infection. This case is interesting in that it shows complete recovery of cardiac function with successful outcome even after onset of myocardial dysfunction, EFE, and rhythm disturbances on prenatal echocardiogram, findings usually associated with a poor prognosis. Early detection and multidisciplinary approach are the key components in managing this condition and improving outcomes. More research is needed to develop effective fetal interventions for in-utero myocarditis.

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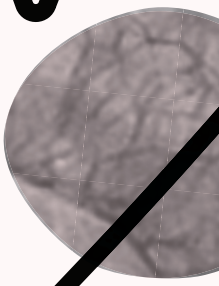

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Early Protein Signals May Help Predict Language Delays in Children with Single Ventricle Heart Disease

Kelly Wolfe, PhD; Benjamin Frank, MD; Jesse Davidson, MD, MPH

Key Takeaways

- Many children with single ventricle heart disease (SVHD) experience neurodevelopmental delays in areas such as language development, but the root cause is not well understood.
- Our researchers previously described widespread proteomic abnormalities in infants with SVHD, identifying 62 proteins known to be involved in neural growth, maintenance and repair.
- Blood sample analysis of children between their first and second SVHD corrective surgeries showed 27 of the 62 proteins were linked to lower-than-average language scores on developmental assessments later in toddlerhood.
- These findings suggest ongoing protein dysregulation may contribute to language delays in children with SVHD, highlighting the potential of blood-based biomarkers to guide personalized care to support neurodevelopment.

"These findings can bolster our ongoing advocacy efforts to expand access to developmental therapies in order to optimize neurodevelopmental outcomes in this vulnerable population."

– Kelly Wolfe, PhD

This study identified 27 proteins linked to below average early language development in infants and toddlers with single ventricle heart disease.

Research Study Background

In single ventricle heart disease (SVHD), neurodevelopmental (ND) impairment is the most common long-term complication, with more than 75% of children showing delays early in life. The causes of ND challenges in SVHD remain largely unknown. Acute brain injury biomarkers guide risk assessment in premature neonates, but with SVHD these injuries occur within the broader context of abnormal neural development beginning in the fetal period.

Experts in the Cardiac Neurodevelopmental Program at Children's Hospital Colorado sought to determine if combining biomarkers of abnormal neural development and neural injury with other risk factors could help predict the risk of ND challenges in kids with SVHD. In earlier work, the team examined biomarkers of pulmonary vascular development in infants with SVHD and found widespread proteomic abnormalities, including in 62 proteins enriched for neural development including neuron growth, maintenance and repair. In this JAMA Pediatrics report, researchers evaluated these proteins for associations with ND challenges in a cohort of 44 children between their first and second corrective SVHD surgeries.

When researchers reviewed how participants performed on standardized assessments of early childhood development delay, language scores were a standard deviation below population norms. An analysis of participant blood samples revealed that 27 of the 62 proteins were linked to language scores.

"Our findings suggest a potential shift in how we understand neurodevelopment in SVHD," says Kelly Wolfe, PhD, a neuropsychologist in the Neuroscience Institute at Children's Colorado and primary study author. "These children may experience persistent dysregulation of proteins vital for neuronal development and repair — proteins linked to language outcomes and detectable through routine blood testing."

"The results of this study are exciting on several levels," Dr. Wolfe continues. "From a research perspective, these findings informed the development of the CAN-DO study, which is a prospective observational study including biomarkers of neural development and neural injury before and after neonatal catheterization versus surgery for children born with certain types of critical CHD, including some with SVHD. From an advocacy perspective, SVHD is currently NOT an automatic qualifying condition to receive early intervention services in the state of Colorado."

Clinical Implications

While previous research has uncovered biomarker evidence of neural injury after cardiac surgery in SVHD, this study is the first to examine biomarkers of neural development and demonstrate protein abnormalities present before stage II repair. Future research should assess whether these abnormalities appear at birth, stem from genetic or epigenetic changes, and are influenced by postnatal factors such as medications, duration of hospitalization or social determinants of health.

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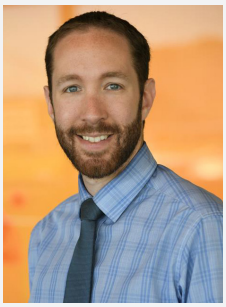

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New Guideline Addresses Barriers to Lifetime Continuous Care for Congenital Heart Disease

Scott Buzby, Cardiologytoday

Key Takeaways

- The American Heart Association and ACC unveiled an updated guideline for managing Adult Congenital Heart Disease.
- The guideline addresses many specific heart defects as well as barriers to lifetime care.
- Professional cardiology societies have issued an updated guideline for the management of adult patients with Congenital Heart Disease, which replaces its 2018 iteration.

The updated guideline addresses a variety of specific Congenital Heart Diseases as well as strategies to mitigate barriers to lifetime continuous care, especially during the transition from childhood to adulthood.

The 2025 American College of Cardiology/American Heart Association Guideline for the Management of Adult Congenital Heart Disease was developed in collaboration with and endorsed by the Heart Rhythm Society, International Society for Adult Congenital Heart Disease and Society for Cardiovascular Angiography and Interventions.

"There is a growing body of literature regarding adults with congenital heart disease, and we are gaining more evidence for our therapies and treatment strategies," Michelle Gurvitz, MD, MS, FACC, associate professor of pediatrics and program director of the Adult Congenital Heart Disease fellowship at Harvard Medical School, well-being director in the department of cardiology at Boston Children's Hospital and chair of the guideline writing committee, told Healio. "We thought it was a good time to bring the literature together and update the prior guidelines. We still have a long way to go in learning about the adult congenital heart disease patient population and how to provide optimal care for all of our patients, but we are moving forward every day."

In synthesizing these new recommendations, the writing committee conducted a systematic review of randomized controlled trials, registries, nonrandomized comparative and descriptive studies, case series, cohort studies, systematic reviews and expert opinion.

"There are dozens of new recommendations in the 2025 adult congenital heart disease guidelines addressing general topics as well as specific conditions," Gurvitz said.

Topics and special conditions with new or revised recommendations included plans of care for adult congenital heart disease programs, expertise for noncardiac surgery, management of cyanosis, heart failure, transplantation, genetic screening, reproductive health, exercise and physical activity, atrial and ventricular septal defect, anomalous pulmonary venous connection, cor triatriatum sinister, congenital valvular disease, Ebstein's anomaly and more.

"Broader themes of the 2025 guidelines include trying to address the challenges related to accessing care."

– Michelle Gurvitz, MD, MS, FACC

"Some of the broader themes of the 2025 guidelines include trying to address the challenges related to accessing care, recommendations for collaborative care between adult congenital specialists and other health care professionals and ensuring we continue to care for the whole patient not just the specific congenital heart condition," Gurvitz told Healio.

The guideline recommends adults with congenital heart disease undergo routine care at dedicated adult congenital heart disease centers, and with multidisciplinary care teams to help guide decision-making.

In addition, the committee wrote that adult patients with anatomic or physiologically moderate or complex Congenital Heart Disease who undergo procedures — cardiac or noncardiac — should have an Adult Congenital Heart Disease cardiologist as part of their care team to provide guidance for procedures, anesthesia and postprocedural management.

"While our management of adult congenital heart disease conditions has improved over time, the variety of anatomic congenital heart defects and the changing surgical approaches over time remain challenges to developing robust clinical outcomes data," Gurvitz told Healio. "Further multicenter research collaboration and advanced research methodology will be required to continue to move the field forward. On the clinical side, there continue to be barriers to accessing care with a limited number of Adult Congenital Heart Disease specialty cardiologists and challenges with insurance coverage and costs."

Barriers to seamless, high-quality, lifelong adult congenital heart disease care include lack of education and organization for patients, clinicians and congenital heart disease programs during the transfer from pediatric to adult care as well as lack of dedicated clinicians and programs for this patient population, according to the document.

To reduce these barriers to care, all patients with Congenital Heart Disease receive structured, patient-centered education during transition from childhood to adulthood to reduce loss to care, the authors wrote.

In addition, the guideline recommended that Congenital Heart Disease programs have in place transfer-of-care procedures to ensure effective handoffs of patients from pediatric to adult cardiologists.

"We are hopeful that clinicians and patients find the recommendations informative and useful and help improve care and stimulate ideas for additional research," Gurvitz told Healio.

ASCEND Cardiovascular Unveils Comprehensive Pediatric Cardiology Offering to Modernize Congenital Heart Disease Management

ASCEND Cardiovascular, the leader in cardiovascular IT solutions, today announced the launch of its re-imagined pediatric cardiology offering, a comprehensive solution specifically designed to meet the unique, lifelong demands of Congenital Heart Disease (CHD) care – from fetal through adulthood. By combining modernized Mullins diagrams with an integration-first architecture, ASCEND is ending the era of "good enough" legacy systems for pediatric programs.

For decades, pediatric cardiology has been underserved by rigid, imaging-centric platforms that fail to capture the complexity of congenital heart disease. ASCEND's new offering replaces these dated workflows with a multi-modality reporting platform that connects the entire cardiovascular ecosystem – from cardiac catheterization and EP to fetal and pediatric echocardiography.

A Modern Approach to Clinical Depth

At the heart of the pediatric offering is a modernized, interactive Mullins atlas. Unlike static legacy illustrations, ASCEND's SVG-designed diagrams allow clinicians to:

- Tailor Patient Anatomy: Add, resize, and edit devices like ASD/VSD closure devices, stents, and coils to reflect patient-specific conditions.
- Automate Data Entry: Populate diagrams with hemodynamic measurements (pressures, saturations, and calculations).
- Utilize Advanced Editing: Use specialized tools for documenting conduits, shunts and ligatures to accurately capture complex procedural anatomy and outcomes.

One Solution, One Lifetime

Congenital Heart Disease is a lifelong journey, and ASCEND's architecture ensures that care is never compromised as patients transition through different life phases. By consolidating data from the moment of fetal or neonatal diagnosis throughout adulthood, ASCEND creates a rich, longitudinal data foundation that enables proactive and targeted care that can be leveraged to:

- Streamline Workflows: Support multi-modality workflows across fetal, pediatric, and adult congenital care to deliver seamless, specialized care throughout every stage of life.
- Manage Aging CHD Populations: As pediatric patients reach adulthood, the system tracks historical surgical data that can be used to

recommend adult-congenital procedures, such as TAVR or complex electrophysiology mappings for arrhythmia management.
- Simplify Device Integrations: Automate data ingestion directly from DICOM SR, VMS+, hemodynamic systems, and other specialized pediatric devices to improve accuracy and allow clinicians to spend more time with patients and less time on documentation.

"Cardiology deserves more than adaptation; it deserves intention," said Dr. Jeff Soble, CEO of ASCEND Cardiovascular. "We believe pediatric cardiology programs should never have to choose between their clinical history and modern capability. Our solution provides the clinical depth required for CHD while leveraging existing enterprise investments in EHR and imaging systems."

Live Telepresence for Remote Collaboration

Recognizing that pediatric expertise is often concentrated in specialized centers, ASCEND has created Catalyst telepresence, which transforms how pediatric care can be delivered across distances, providing:

- Real-Time Specialist Consultation: Clinicians can consult with remote pediatric sub-specialists in real time, as if they were physically present in the room. This enriches clinician-to-clinician collaboration and allows for more meaningful, immediate interactions between specialists and the patient's family.
- Remote Imaging Oversight: Cardiologists can remotely oversee image acquisition for pediatric procedures.

By ensuring that every necessary view is captured with diagnostic-grade quality the first time, Catalyst reduces overall procedure time, saves valuable cardiologist time, and—most importantly—eliminates the need for stressful re-imaging sessions for young patients.

Advancing CHD Care with Artificial Intelligence

The impact of this offering is further amplified when combined with advanced AI solutions to provide a complete, intelligent end-to-end reading and reporting workflow that addresses the most persistent challenges in clinical practice.

By pairing ASCEND's structured reporting and zero-footprint DICOM viewer with Ventripoint's AI-based diagnostic technologies, the combined solution delivers a level of clinical insight previously unavailable in standard workflows. This synergy powers the construction of a 3D model of the heart generated from 2D echo data, enabling calculation of volumes and ejection fractions for all chambers with accuracy comparable to MRI, providing a critical diagnostic alternative for young CHD patients who may not tolerate or have access to traditional cardiac MR. This partnership represents a shared commitment to novel solutions that ensure pediatric specialists have the technical expertise and research-grade capabilities required for complex heart analysis.

Rady Children's Health Co-CEO to Take Job in Atlanta

Departure leaves CHOC Co-CEO in charge of merged pediatric provider

Paul Sisson, The San Diego Union-Tribune

It is back to Atlanta for Dr. Patrick Frias, Co-Chief executive officer of Rady Children's Health.

Rady, which completed a merger with Children's Hospital of Orange County in early 2025 under Frias' leadership, announced that the executive accepted a job as the new CEO of Children's Healthcare of Atlanta.

Frias, a cardiologist and electrophysiologist, was the Chief Operating Officer of Children's Healthcare of Atlanta when Rady lured him to San Diego in 2018 to replace retiring CEO Dr. Donald Kearns.

With Frias' departure, Co-CEO Kimberly Chavalas Cripe will serve as the organization's sole president. Cripe was the chief executive of Children's Hospital of Orange County, becoming Co-CEO with Frias after the merger.

It is the opposite of the result that was anticipated during the merger. The previously shared plan was for the two executives to run their respective operations for two years with Chavalas Cripe retiring after a 30-year career, leaving Frias as sole CEO.

Through a spokesperson, Frias declined to discuss his tenure at Rady or his reasons for heading back to Atlanta instead of sticking with the succession plan that the CHOC merger put in place.

But he did express his feelings on the matter more deeply in a memo to all staff, deeming the decision "bittersweet."

"This was not an easy decision," Frias wrote. "Ultimately, it was guided by my desire to be closer to my family and return to my home state."

Frias also said that Rady will "begin a thoughtful national search for a future leader with the experience and passion to build on our progress."

Cripe, a hospital spokesperson said, still intends to retire.

S. Doug Hutcheson, Chair of Rady's Board of Directors, bid Frias farewell.

"I've had the honor of experiencing first-hand the transformational changes Patrick has made during his time in San Diego at Rady Children's Hospital and Health Center," Hutcheson said. "Over the past year, Patrick's and Kim's vision, strategic acumen and unwavering commitment to our mission have shaped the future of pediatric healthcare across Southern California and improved the lives of thousands of children in our region. We wish him continued success in this next chapter of his remarkable career."

In its own statement, Atlanta children's referenced Frias' 18 years of experience "as a cardiologist, Chief Physician Officer and Chief Operating Officer," as the reasons why it offered its former executive a round-trip ticket.

Patrick Frias, Rady Children's Hospital-San Diego President and CEO (Rady Children's Hospital)

Frias departs at a fragile moment for Rady Children's. Caring for an estimated 800,000 children in Southern California, a patient volume that puts it among the largest providers of pediatric care in the nation, Rady has found itself targeted by the Trump administration due to its gender-affirming care program.

On January 20th, Rady severely curtailed the program, stopping surgeries and prescriptions but continuing counseling and other mental health care services, citing nationwide threats to end all federal reimbursement.

That move spurred an immediate backlash, first from affected patients and their families who staged a protest in front of Rady's San Diego medical campus, then

through a lawsuit by California Attorney General Rob Bonta, who obtained a temporary restraining order that forced Rady to resume all gender care, save surgeries.

Gender care strife notwithstanding, Frias' tenure at Rady has been positive.

For three years running, Rady has been named among the nation's 10 best children's hospitals by U.S. News and World Report, a designation that ranks medical providers in specialty care such as cardiology, endocrinology and orthopedics.

In 2023, Rady broke ground on a seven-story medical tower, estimated to cost between $1.2 billion and $1.4 billion, replacing aging structures on its main Serra Mesa campus.

Dr. Stephen Kingsmore, Chief Executive of the Rady Children's Institute for Genomic Medicine, cited this project, as well as completion of the merger with CHOC, as defining accomplishments.

"Patrick was a dynamic leader who started two of the most ambitious long-term projects in the hospital's history," Kingsmore said in an email.

Rady has also maintained an ongoing collaboration with UC San Diego. Most of its physicians have dual appointments on the university's medical faculty, making it easier to undertake research studies. The organization lists 17 areas of inquiry and 200 active clinical trials on its website, ranging from allergy to urology.

Today, Rady Children's Health, including operations in Orange County, has more than 13,000 employees and more than 2,000 physicians on its medical staff.

Frias mentioned many of these accomplishments in his final message to staff, crediting the organization's successes to all of its employees.

"I am deeply grateful for all we have accomplished together and for the relationships built along the way," Frias wrote. "I leave here a better leader because of you, and I will always carry forward lessons and friendships from this chapter."

CAREER OPPORTUNITIES

Click the position title to view the full job description

Advanced Heart Failure - Cardiac Transplant Team
Phoenix Children's
Phoenix, Arizona

Pediatric Cardiologist
Phoenix Children's
Phoenix, Arizona

Pediatric Cardiologist
Loma Linda University
Children's Hospital
Loma Linda, California

Pediatric Electrophysiologist
Loma Linda University
Children's Hospital
Loma Linda, California

Assist/Assoc/Full Professor Interventional Cardiology
University of Alberta
Division of Pediatric Cardiology
Edmonton, Alberta, Canada

Assist / Assoc Professor of ACHD
Yale University
Yale New Haven
Children's Hospital
New Haven, Connecticut

Director of Pediatric Heart Failure / Heart Transplant Cardiology
Ochsner Children's Hospital
New Orleans, Louisiana

Pediatric Cardiologist Advanced Imaging with Cross-Sectional Focus
MaineHealth Maine
Medical Center
Portland, Maine

Pediatric Interventional Cardiology
Children's Hospital of Michigan
Detroit, Michigan

Regional Pediatric Cardiologists
Ochsner Children's Hospital
Baton Rouge, Lafyette, Monroe, Louisiana
Gulfport, Mississippi

Pediatric Cardiologist Preventative Cardiology
Children's Mercy Hospital
Kansas City, Missouri

Pediatric Cardiologist
The University of Utah
Primary Children's Hospital
Las Vegas, Nevada

Adult Congenital Cardiologist
Akron Children's Hospital
Akron, Ohio

Physician Director, ACHD
Akron Children's Hospital
Akron, Ohio

Pediatric Cardiologist
Tri-City Pediatric Cardiology, P.C.
Johnson City, Tennessee

APRIL

10TH-11TH

EuroACHD 2026
Madrid, Spain
https://www.escardio.org/

16TH-17TH

EPIC 2026 - Emory Percutaneous Intervention Course
Virtual
https://cerebria.org/ch/EPIC

MAY

16TH-19TH

AEPC 2026
Padua, Italy
https://www.aepc2026.org/

18TH-19TH

5th CME Cardiologists Conference
Athens, Greece
https://cardiologists.plenareno.com/

JUNE

16TH-19TH

ACHD 2026
Cincinnati, Ohio
https://www.cincyhearteducationseries.org/achd2026

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