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IMAGES IN EMERGENCY MEDICINE

Ultrasound

An unusual finding in a case of syncope

Taryn Clark MD[1,2,3] | Kyle Hoppens MD[4] | David Yang[5] | Lewis Hunt-Irving MD[4] | Christopher Cartmill MD[4] | Ellen Slaven MD[4] | Christine Butts MD[4]

[1] Department of Emergency Medicine, SUNY Downstate Medical Center & Kings County, Hospital Center, New York, New York, USA

[2] Biomedical Research Unit, A.B Prisma, Lima, Perú

[3] Department of International Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland, USA

[4] Section of Emergency Medicine, Louisiana State University Health Sciences Center, New Orleans, Louisiana, USA

[5] Department of Emergency Medicine, Yale School of Medicine, New Haven, Connecticut, USA

Correspondence
Taryn Clark, MD, Department of Emergency Medicine, SUNY Downstate Medical Center & Kings County Hospital Center, New York, NY, USA.
Email: tarynnclark@gmail.com

1 | PRESENTATION

A 42-year-old male presented to the emergency department (ED) for syncope. Shortly after an uncomplicated hydrocele aspiration in the urology clinic, he suddenly felt lightheaded and nauseated. He then became diaphoretic, vomited, and experienced a syncopal episode, witnessed by his wife. Per chart review, the patient had atrial fibrillation, but the patient did not convey this to us and was taking no medications. He had no significant family history. Upon arrival to the ED, he was alert and oriented, had a blood pressure of 100/62, heart rate of 78,

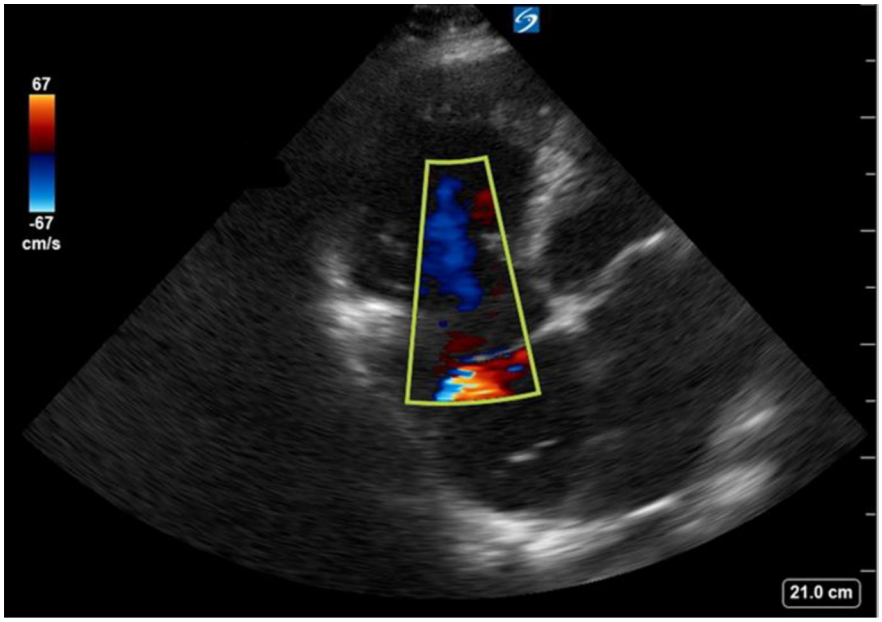

FIGURE 1 (Apical 4 chamber with flow): Shows intimal flap protruding from the atrial septum. Motion revealed rhythmic beating of this flap; however, it did not appear to be coordinated with remainder of the heart as evidenced by non-laminar flow

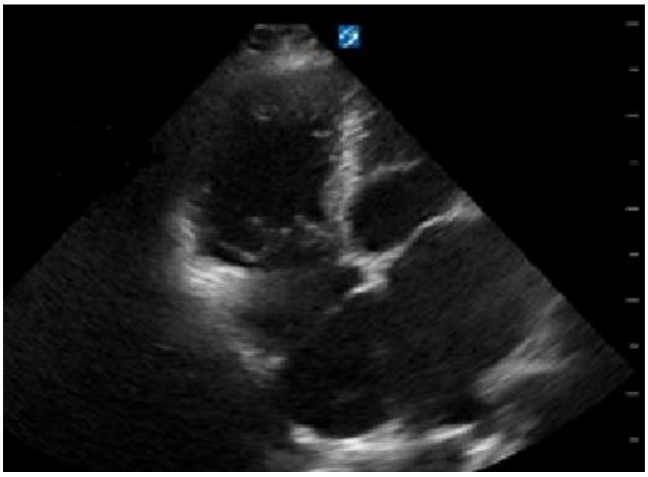

FIGURE 2 (apical 4 chamber): More clearly shows intimal flap protruding from the atrial septum splitting the right atrium (RA) and thus forming a triatrial heart. This view also clearly demonstrates the significant RA enlargement most likely secondary to turbulent flow

respiratory rate of 28, temperature of 96.8°F, and oxygen saturation of 100% on room air. Orthostatic vital signs were normal. EKG showed atrial fibrillation with a right axis. Bedside ultrasound echocardiography demonstrated the following images (Figures 1, 2, and 3).

2 | DIAGNOSIS AND TEACHING POINTS

This patient has cor triatrium dextrum (CTD) as seen on ultrasound. CTD is a rare congenital heart anomaly where the right atrium is divided into 2 chambers by an incomplete membrane usually made of fetal eustachian and thebesian valves.[1,2] It represents 0.1% to 0.4% of all congenital heart diseases and can be associated with other congenital heart defects with the most common being ostium secondum atrial septal defect and patent foramen ovale. Although it is typically diagnosed in childhood, a delay in symptom presentation can lead to later diagnoses.[3] The majority of CTD is asymptomatic and is discovered incidentally during surgery for another pathology or after death in post-mortem exams.[4] When symptomatic, the presentation may include heart failure, arrythmia, syncope, cyanosis, pulmonary embolism, and sudden cardiac death.[5-7] Pediatric literature suggests that syncope in CTD may be owing to pulmonic valve obstruction.[8,9] Atrial fibrillation, although more common in CTD's counterpart, cor triatriatum sinister, has been reported previously and warrants consideration of anticoagulant agents.[10] Management can be expectant, symptomatic treatment or surgical resection.[11]

In a patient with syncope, the ED differential diagnosis should consider cardiogenic causes of syncope. CTD can be screened through use of echocardiography during standard ED protocols.[12] The ED where this patient was seen uses the 5 Es as a standardized approach

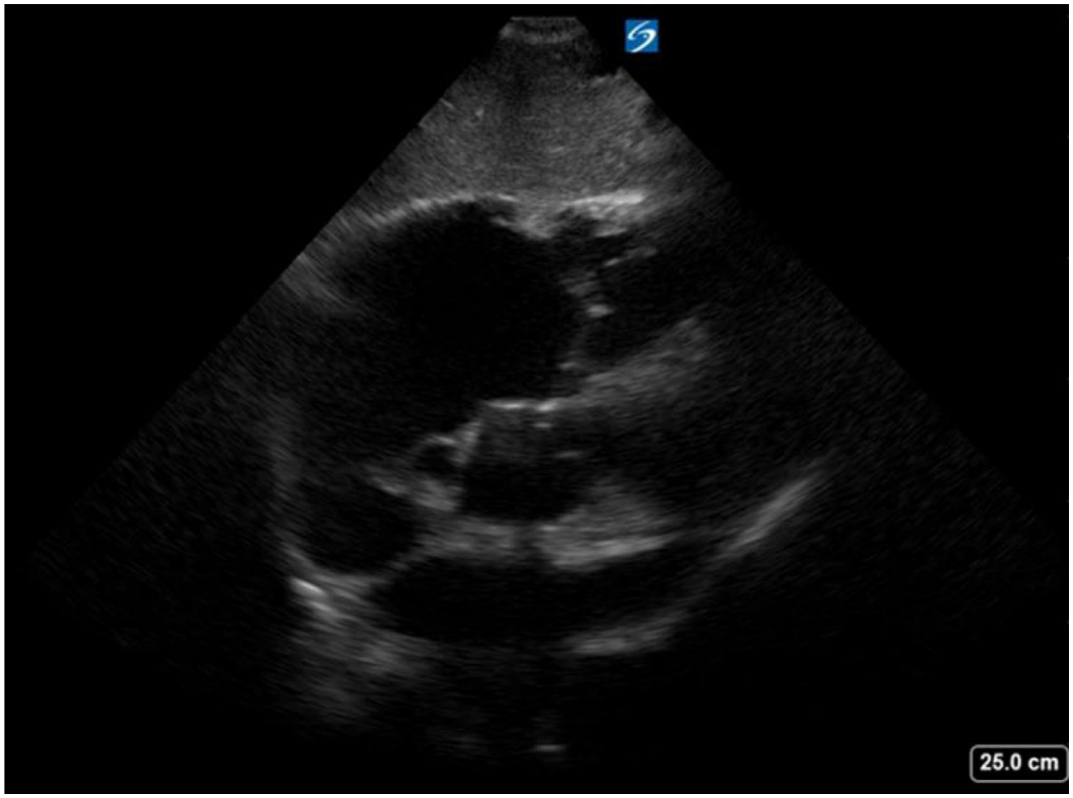

FIGURE 3 (subxiphoid): Shows extremely enlarged right atrium, which can also be seen in other views. It does not clearly show the location of the extra atrium in this image; however, the image appears to be grossly abnormal. This view also shows the aortic outflow tract. In all views, both ventricles appear to be within normal limits with adequate contractility and no significant chamber enlargement; the only obvious abnormalities were noted to be supraventricular

to echocardiography. In this approach, Effusion, Ejection fraction, Equality of the ventricle, Exit, and Entrance are assessed, as previously described in literature.[13] As seen in our report, CTD can best be screened for in the apical 4 chamber and subxiphoid views of the heart. If visualized, cardiology consult is recommended and discharge of the stable patient should occur only if the patient has close follow-up.

Our patient was initially treated with 1 liter of normal saline, 4 mg of IV ondansetron, and 5-325 mg of hydrocodone-acetaminophen. Although his presentation was most consistent with vasovagal syncope, the rare findings seen on echocardiography prompted cardiology consultation and evaluation for high-risk syncope. Further cardiac workup revealed a chest X-ray with cardiomegaly with mild pulmonary vascular congestion, troponin <0.02 ng/mL, and B-type natriuretic peptide 368 pg/mL. Complete blood count and metabolic panel were within normal limits. Cardiology felt that this syncopal event was likely a vasovagal response and unrelated to the patient's CTD. The patient required promethazine 12.5 mg for nausea and an additional dose of hydrocodone for pain control with complete resolution of symptoms. Our patient was discharged with close cardiology follow-up and referral to cardiothoracic surgery.

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