Isolated Traumatic Membranous Interventricular Septal Rupture: A Rare Complication Following a Motor Vehicle Accident

Aashna Deep, BMedSc, Christina Chan, MBChB, MmedSci, FASE, Philip D. Adamson, MBChB, PhD, MPH, Richard Troughton, MBChB, PhD, FRACP, and Ellen Woodcock, PhD, *Christchurch, Canterbury, New Zealand; and Edinburgh, United Kingdom*

INTRODUCTION

Blunt cardiac injury is most commonly caused by motor vehicle accidents. The most common form of injury is contusion of the anteriorly situated right ventricle (RV), often resulting in transiently impaired function of the chamber. The total incidence of blunt cardiac injury varies significantly in literature, with occurrence of a traumatic ventricular septal rupture (VSR) secondary to blunt chest trauma being a rare complication. The incidence of this is likely underestimated due to the severity of the injury and the potential for rapid hemodynamic decline and death prior to diagnosis. This report details the presentation of a patient involved in a high-speed, multivehicle collision who was found to have a traumatic membranous VSR on transthoracic echocardiography (TTE) after the development of complete heart block.

CASE PRESENTATION

A 19-year-old previously well woman with no prior cardiac history presented via the ambulance service after a high-speed motor vehicle accident (traveling at approximately 100 km/h or 62 mph). The patient was unrestrained by a seatbelt, had been ejected through the vehicle windshield, and was found unresponsive 40 meters (>130 feet) from the vehicle with traumatic injuries to the head, central chest, torso, and an upper limb. An adrenaline infusion was started at the scene for bradycardia and hypotension. The patient was intubated and transferred to hospital. Trauma computed tomography (CT) performed after arrival at the hospital demonstrated a severe traumatic brain injury, including bilateral frontal lobe contusions, and left frontal lobe subcortical hemorrhage resulting in a 6 mm leftward midline shift and left uncal herniation.

The thoracic injuries included left rib fractures and pulmonary contusion, which was confirmed on the chest radiograph (Figure 1). Upon admission to the intensive care unit, the patient developed complete heart block, which at the time was attributed to neurological injury and autonomic dysfunction.

A bedside TTE to assess for cardiac contusion or pericardial effusion was performed with the patient intubated, ventilated, and lying supine. Parasternal imaging demonstrated a tear to the interventricular septum (IVS) anteromedially at the junction of the membranous and muscular septum below the aortic root (measuring approximately 12 mm). A thin linear band of tissue, considered to be a remnant of RV endocardium, that had been stretched rather than torn, was noted extending superiorly and parallel to the anterior of the aortic root (Figure 2, Video 1). Color-flow Doppler revealed a significant left-to-right shunt at the level of the defect (Figure 3, Videos 2 and 3), with a peak velocity of approximately 3.8 m/sec (peak gradient 58 mm Hg) on continuous-wave Doppler (Figure 4). Imaging in the parasternal short and apical window demonstrated the defect coursing anteriorly through the membranous septum (Figure 5, Video 5), leading to and terminating at the right ventricular outflow tract (RVOT), approximately 1.0 cm below the pulmonary valve. The RV was visually estimated to be mildly dilated with impaired function, and the mid-to-basal RV free wall was hypokinetic. The right ventricular systolic pressure was very mildly elevated at approximately 31 mm Hg above a mildly elevated right atrial pressure (the inferior vena cava was of normal size without respiratory collapse). Other findings included a normal left ventricle (LV) size (LV end-diastolic dimension: 42 mm) with hyperdynamic function (LV ejection fraction 65%-70%). The aortic valve structure and function were normal (there was no significant aortic regurgitation). All other cardiac valves were normal. There was no pericardial effusion. The aortic arch and descending thoracic aorta were not assessed due to time and access limitations; however, they appeared intact on the trauma CT. Considering the normal left-sided chamber dimensions as well as the impairment of the RV, these findings were consistent with a moderately sized traumatic membranous VSR. As this defect resulted in a physical disruption of the conduction system, it also explained the development of complete heart block. The VSR was also identified retrospectively on the trauma CT (Figure 6), after the defect had been identified on TTE. A targeted cardiac CT was not performed as the patient did not survive their noncardiac injuries.

From the Department of Cardiology, Christchurch Hospital, Christchurch, Canterbury, New Zealand (A.D.,C.C.,P.D.A.,R.T.,E.W.); British Heart Foundation Centre for Cardiovascular Science, University of Edinburgh, Edinburgh, United Kingdom (P.D.A.,); and Christchurch Heart Institute, University of Otago Christchurch, Christchurch, Canterbury, New Zealand (P.D.A., R.T., E.W.).

Keywords: Transthoracic echocardiography, Ventricular septal rupture, Motor vehicle accident

Correspondence: Aashna Deep, BMedSc, Department of Cardiology, Christchurch Hospital, Private bag 4710, Christchurch, Canterbury 8140, New Zealand. (E-mail: *aashna.deep01@gmail.com*).

2468-6441
https://doi.org/10.1016/j.case.2024.01.005

VIDEO HIGHLIGHTS

Video 1: Two-dimensional TTE, parasternal long-axis view, demonstrates normal LV size and function with the VSR (echo-free space) in the basal IVS anteromedially at the junction of the membranous and muscular septum with a linear band of RV endocardium extending parallel to the aortic root.

Video 2: Two-dimensional TTE, parasternal long-axis zoom view with rightward sweep on the basal IVS, demonstrates the membranous VSR as an echo-free space below the aortic root at the junction of the membranous and muscular IVS.

Video 3: Two-dimensional TTE with color-flow Doppler, parasternal long-axis view, demonstrates a serpentine defect with left-to-right systolic flow through the membranous VSR terminating within the RVOT.

Video 4: Simultaneous two-dimensional TTE (*left*) and color-flow Doppler (*right*) color-compare display, parasternal short-axis sweep from base (aortic valve) to mid-LV (papillary muscle) view, demonstrates the VSR as a disruption of the membranous septum with left-to-right shunting coursing anteriorly into the RVOT.

Video 5: Simultaneous two-dimensional TTE (*left*) and color-flow Doppler (*right*) color-compare display, parasternal short-axis sweep from the mitral valve to the papillary muscles, demonstrates a disruption of the basal muscular septum (VSR) coursing anteriorly with left-to-right flow.

View the video content online at www.cvcasejournal.com.

Intervention to close the VSR was considered but not performed due to the patient's critical neurological condition. The patient succumbed to their brain injury and was palliatively extubated the following day.

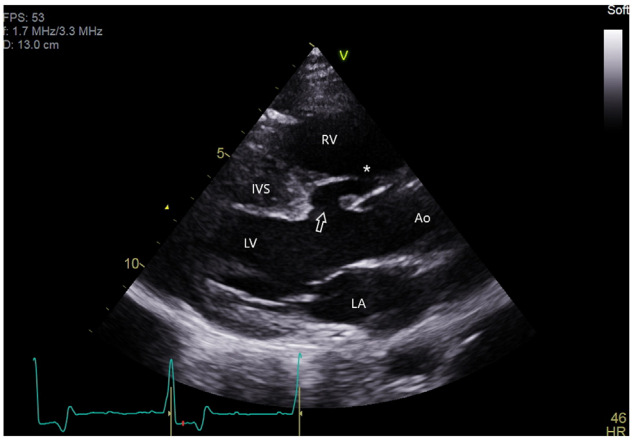

Figure 2 Two-dimensional TTE, parasternal long-axis mid-systolic view, demonstrates the VSR (*arrow*) in the basal IVS anteromedially at the junction of the membranous and muscular septum with a linear band of RV endocardium extending parallel to the aortic root (*). *Ao*, Aorta; *LA*, left atrium.

DISCUSSION

Cardiac injury sustained from blunt force trauma is most commonly seen following motor vehicle accidents (with incidence ranging between 20% and 76%), but can also occur secondary to assault, sporting-related injuries, and crush injuries.[1,2] Manifestations of cardiac injury can include myocardial contusion, coronary artery injury, valvular injury, VSR, and pericardial injury.[1,3] Clinical presentation varies from asymptomatic patients to patients in cardiogenic shock, and is dependent on other concurrent injuries as well as the level of hemodynamic compromise.[2] The presence of conduction disturbances or a murmur can allude to the presence of a traumatic VSR.[4] In the case of this patient, a VSR specifically was not initially suspected, and the primary

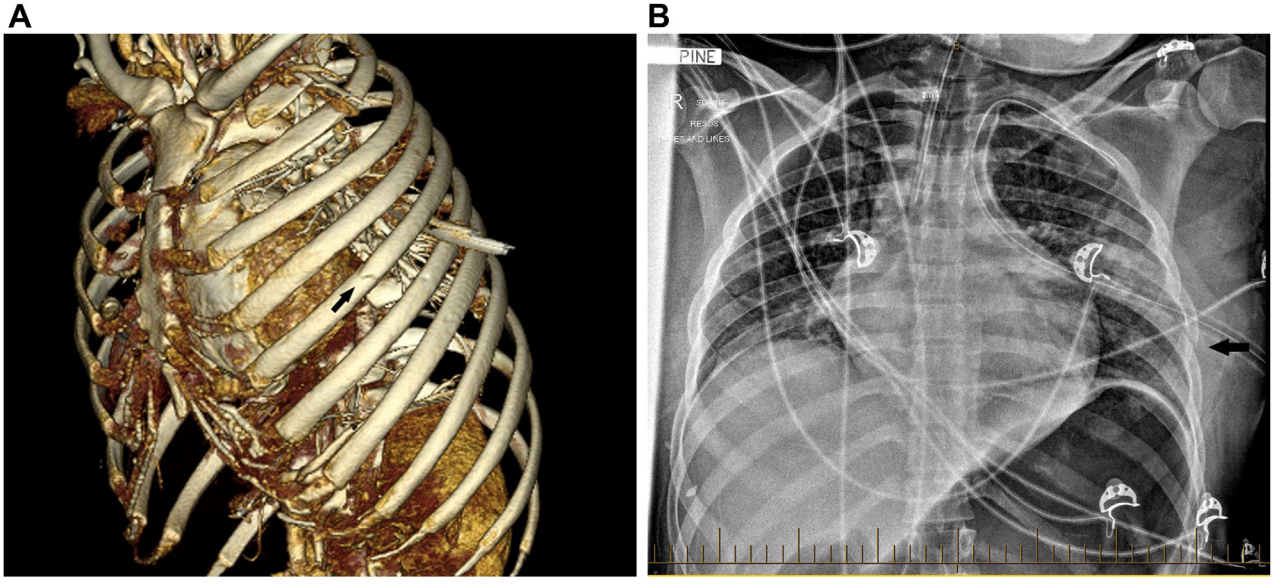

Figure 1 **(A)** Three-dimensional chest CT, trauma protocol, oblique view, demonstrates a fracture in the lateral fifth left rib (*arrow*). **(B)** Chest radiograph, anteroposterior projection, supine position, demonstrates lateral fifth left rib fracture (*arrow*) and increased density in the mid zone of the left lung and lower zone of the right lung, consistent with a pulmonary contusion.

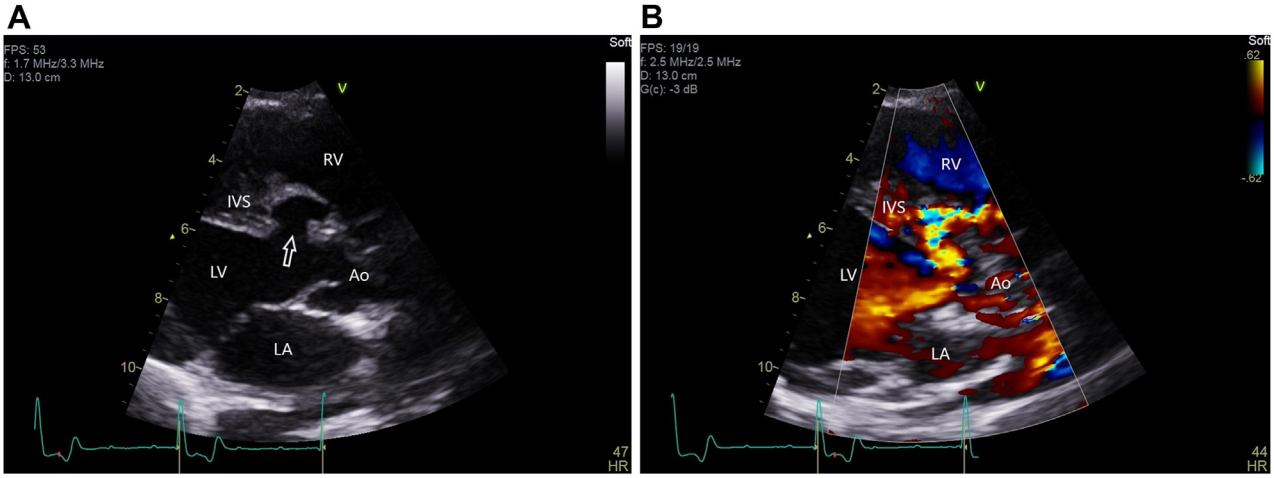

Figure 3 Two-dimensional TTE, zoomed parasternal long-axis mid-systolic view, without **(A)** and with **(B)** color-flow Doppler, demonstrates the VSR (*arrow*), with a serpentine defect noted by the turbulent left-to-right shunt. *Ao*, Aorta; *LA*, left atrium.

indication of cardiac compromise was complete heart block on the patient's electrocardiogram.

While isolated traumatic VSR is a rare sequela of blunt force trauma, evidence of its occurrence can be found in literature dating back to the mid-19th century. Hewett describes a case in 1847 of a young male patient with an isolated muscular VSR found at postmortem following blunt chest trauma.[5] The true incidence of VSR following blunt chest injuries is unknown and likely underestimated. This is in part due to a large proportion of patients being diagnosed with the defect postmortem, as well as the varying incidence of blunt cardiac injuries reported in literature.[4] The most common location for a VSR to occur is in the muscular apical IVS.[6]Therefore, our patient presents a unique case, not only due to the occurrence of a traumatic VSR following blunt force trauma but also due to the location of the rupture within the membranous IVS.

A VSR can develop immediately or over multiple days post-trauma. The proposed mechanism of immediate traumatic VSR following blunt force trauma is thought to occur due to a sudden increase in end-diastolic/early systolic intracardiac pressure, caused by mechanical compression of the heart (between the vertebral column and sternum). In this instance, the IVS can become susceptible to acute rupture as the intracardiac pressures are raised at a time when the ventricles are filled and the atrioventricular valves are closed.[7,8] In contrast, delayed development of a traumatic VSR is thought to occur due to microvascular injury caused by myocardial contusion, leading to tissue necrosis and subsequent perforation/rupture of the septum.[8,9] In the case of our patient it was thought that the

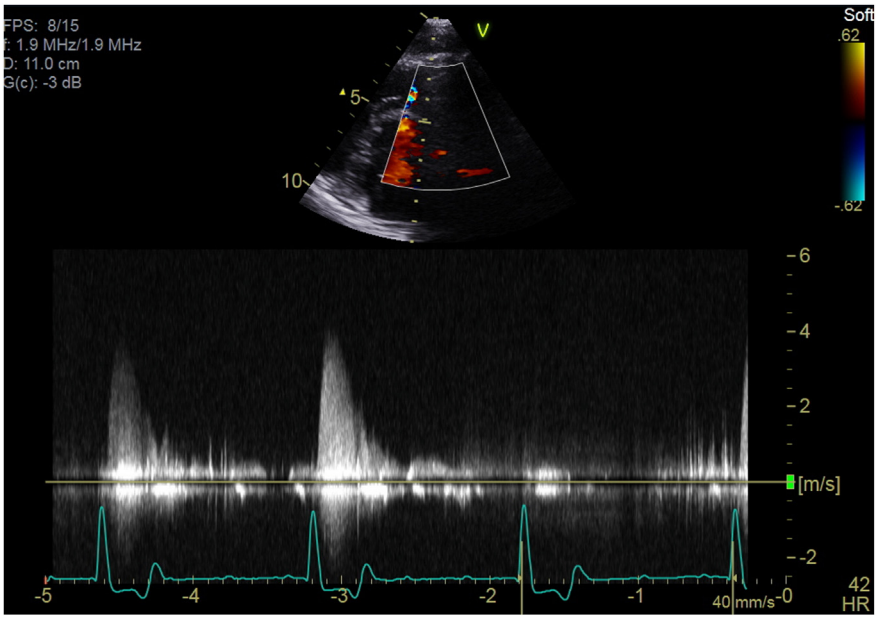

Figure 4 Two-dimensional TTE with color-flow Doppler, parasternal short-axis view, continuous-wave spectral Doppler display with the cursor through the VSR, demonstrates the peak velocity at 3.8 m/sec (peak gradient = 58 mm Hg). Doppler profile quality affected by the patient's respiration.

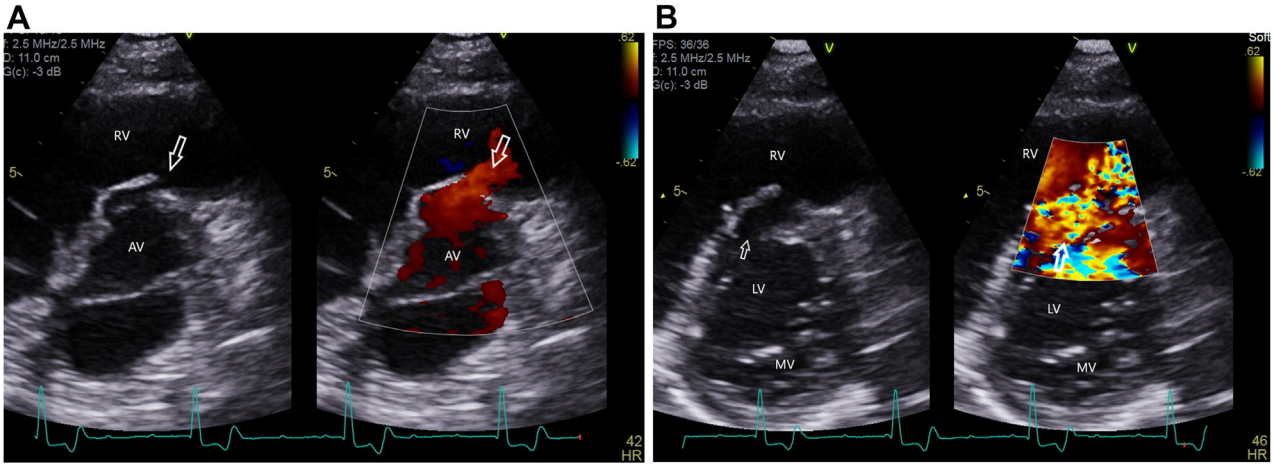

Figure 5 **(A)** Simultaneous two-dimensional TTE (*left*) and color-flow Doppler (*right*) color-compare display in early diastole, basal parasternal short-axis view, demonstrates the VSR as a disruption of membranous septum (*arrow*), with left-to-right shunting coursing anteriorly into the RVOT (*arrow*). **(B)** Simultaneous two-dimensional TTE (*left*) and color-flow Doppler (*right*) color-compare display in mid-systole, mid-parasternal short-axis view, demonstrates the VSR as a disruption of the basal muscular septum (*arrow*), with left-to-right shunting coursing anteriorly. *AV*, Aortic valve; *MV*, mitral valve.

membranous septal site of the rupture likely reflects the motion of the septum in relation to the magnitude and direction of the deceleration force directed through the thorax, relative to the semirigid cardiac skeleton.

Cardiovascular imaging plays an integral role in the diagnosis and management of cardiac injury after chest trauma. Not only is cardiovascular imaging required to establish a diagnosis, but it also guides intervention and is used for surveillance after intervention. Point-of-care ultrasound (POCUS) is often the first imaging modality used for suspected traumatic cardiac injury in an acute setting. While POCUS can provide rapid assessment of a life-threatening injury (including cardiac rupture or aortic injury), it is operator dependent and is often limited by acoustic window access and quality.[10,11] Detailed two-dimensional TTE provides a noninvasive, quick, and accessible means to screen for a hemodynamically significant defect, proving it to be one of the most effective diagnostic tools in the acute setting.[2] In addition to identifying the presence of a defect, TTE provides assessment of the defect's severity and impact through assessment of cardiac structure and ventricular function. Additionally, TTE can be used to identify any further complications, such as a pericardial effusion.[12] Transesophageal echocardiography (TEE) is also a useful modality in the assessment of such lesions. Due to its high image quality, TEE can provide enhanced visualization of the location and course of a VSR, the relationship of the defect to other cardiac structures, and the nature and extent of the intracardiac shunt.[12] Transesophageal echocardiography also has an advantage over TTE when there are limitations to noninvasive imaging, such as limited access to acoustic windows and suboptimal imaging due to the patient's position.[12] A TEE was not obtained in the case of this patient due to their noncardiac injuries.

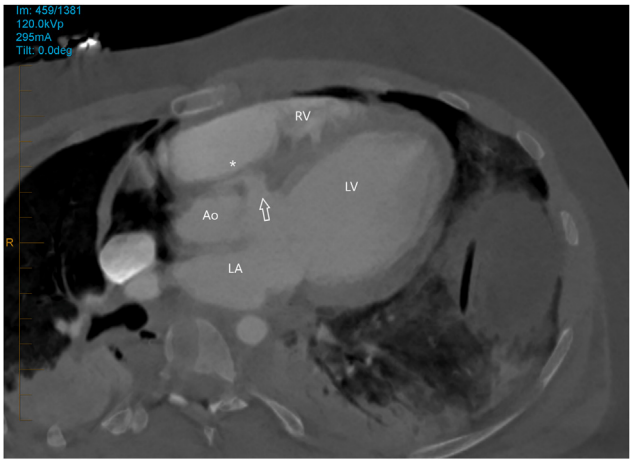

Figure 6 Computed tomography, nongated, contrast-enhanced trauma protocol, axial view, demonstrates the VSR as a disruption of the basal IVS immediately below the aortic root (*arrow*) with remnant RV endocardium extending parallel to the aortic root (*). *Ao*, Aorta; *LA*, left atrium.

Cardiac catheterization as well as electrocardiogram-gated CT can also be used in such cases to characterize the defect. In trauma patients, the use of CT is extensive and is often performed prior to a TTE. Comprehensive evaluation of even a noncardiac focused CT may lead to early identification of a VSR (or other hemodynamically significant lesions), which can facilitate a rapid focused cardiac assessment (via formal TTE) if the lesion has not been identified on POCUS (or if a POCUS has not been performed).[13] Cardiac catheterization can be useful in identifying the hemodynamic significance of the VSR by quantitating the shunt ratio.[2] In the case of our patient, the VSR can be seen on the trauma CT; however, as a hemodynamically significant cardiac lesion was not suspected, it was not identified at the time of the scan. Further assessment to characterize the traumatic VSR was unable to be conducted due to the patient's extensive injuries.

Treatment for a traumatic VSR is dependent on multiple factors, including the size of the lesion, whether the lesion is hemodynamically significant (shunt ratio >2.0), and the presence of heart failure.[2,9] Small defects are often treated percutaneously, with a closure device, or left unrepaired for spontaneous closure, while surgical repair is required for large ruptures.[2,9] Unrepaired large VSRs have been associated with pulmonary arterial hypertension and rapid hemodynamic decline. In these instances, early surgical repair is indicated.[14] In

patients undergoing early surgical repair of VSR, there is a documented risk of the myocardial tissue being too fragile, nonviable, and unrepairable.[2] Early intervention to the IVS, either percutaneous or surgical, for our patient was not considered due to the severity of the neurological condition.

CONCLUSION

Ventricular septal rupture is a rare and potentially life-threatening consequence of blunt cardiac injury and commonly occurs within the muscular septum toward the apex. This case report highlights the importance of focused cardiovascular imaging in patients with blunt chest trauma; as in most cases, early diagnosis of a hemodynamically significant lesion is crucial for prompt intervention to stabilize a patient. The patient presented in this case experienced a blunt cardiac injury during a motor vehicle accident and was diagnosed with a membranous traumatic VSR. Intervention was not possible due to the patient's critical neurological condition.

ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans.

CONSENT STATEMENT

The authors declare that since this was a noninterventional, retrospective, observational study utilizing de-identified data, informed consent was not required from the patient under IRB exemption status.

FUNDING STATEMENT

The authors declare that this report did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

DISCLOSURE STATEMENT

The authors report no conflict of interest.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found online at https://doi.org/10.1016/j.case.2024.01.005.

REFERENCES

1. Schultz JM, Trunkey DD. Blunt cardiac injury. Crit Care Clin 2004;20:57-70.
2. Reinhardt SW, Popescu WM, Crandall I, Tantawy H, McNamara RL. Large traumatic ventricular septal defect: a rare complication of blunt chest trauma. Circulation 2019;12:e008783.
3. Nair L, Winkle B, Senanayake E. Managing blunt cardiac injury. J Cardiothorac Surg 2023;18:71.
4. Marcolini EG, Keegan J. Blunt cardiac injury. Emerg Med Clin 2015;33:519-27.
5. Hewett P. Rupture of the heart and large vessels: the result of injuries. Lond Med Gaz 1847;1:870.
6. Parmley LF, Manion WC, Mattingly TW. Nonpenetrating traumatic injury of the heart. Circulation 1958;18:371-96.
7. Hamdan-Challe M, Godin M, Bouchart F, Doguet F. Isolated ventricular septal rupture secondary to blunt trauma. Interact Cardiovasc Thorac Surg 2010;11:667-9.
8. Mello RP, Santana MV, Silva MA, Esteves C, Pedra C, Thom A, et al. Ventricular septal rupture following blunt chest trauma. Arq Bras Cardiol 2006;87:e243-6.
9. Kim NY, Kwon TJ, Park JR, Choi HC, Cheon YH, Kang MK, et al. Delayed ventricular septal rupture following blunt chest trauma. Korean Circ J 2011;41:625-8.
10. Batchelor TJ, Imperato NS, Wheel KL, Rennie AJ, Roth KR. Traumatic atrial septal defect diagnosed by bedside point-of-care ultrasound. Radiol Case Rep 2022;17:4546-9.
11. Zanza C, Longhitano Y, Artico M, Cammarota G, Barbanera A, Racca F, et al. Bedside cardiac pocus in emergency setting: a practice review. Rev Recent Clin Trials 2020;15:269-77.
12. Girón-Arango L, D'Empaire PP. Is there a role for transesophageal echocardiography in the perioperative trauma patient? Curr Anesthesiol Rep 2022;12:210-6.
13. Hammer MM, Raptis DA, Cummings KW, Mellnick VM, Bhalla S, Schuerer DJ, et al. Imaging in blunt cardiac injury: computed tomographic findings in cardiac contusion and associated injuries. Injury 2016;47:1025-30.
14. Al-Hijji MA, Kelle AM, Park JY, Dearani JA, Taggart NW. Delayed repercussions of blunt trauma: isolated muscular ventricular septal defect. CASE 2017;1:11.