

การปิดรูรั่วของผนังกันหัวใจห้องล่างเนื่องจากได้รับอุบัติเหตุกระดูกแตกทรวงอกผ่านทางสายสวนหัวใจและหลอดเลือด : รายงานผู้ป่วย 1 ราย

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Percutaneous Transcatheter Closure in Blunt Chest Trauma with Traumatic Ventricular Septal Defect: A Case Report

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หลักการและวัตถุประสงค์: ภาวะบาดเจ็บของหัวใจอันเกิดจากอุบัติเหตุกระดูกแตกบริเวณทรวงอกพบได้ไม่บ่อย และมักจะไม่ได้รับการวินิจฉัยซึ่งสามารถทำให้เกิดภาวะทุพพลภาพและเสียชีวิตตามมาได้ รายงานนี้นำเสนอกรณีผู้ป่วยชายอายุ 17 ปี 1 ราย ซึ่งได้รับอุบัติเหตุจากการขับขี่รถจักรยานยนต์ทำให้เกิดการกระดูกแตกบริเวณทรวงอกอย่างรุนแรงจนเกิดรูรั่วของผนังกันหัวใจห้องล่างบริเวณส่วนกล้ามเนื้อร่วมกับการฉีกขาดของ papillary muscle ของลิ้นหัวใจไตรคัสปิดซึ่งทำให้ลิ้นหัวใจไตรคัสปิดรั่วอย่างรุนแรงร่วมกับนำเสนอการวินิจฉัยและการรักษาภาวะผนังกันหัวใจห้องล่างบริเวณส่วนกล้ามเนื้อรั่วซึ่งหลงเหลืออยู่ภายหลังการรักษาด้วยการผ่าตัดด้วยการรักษาผ่านทางสายสวน

วิธีการศึกษา: เป็นการศึกษาเรียบเรียงจากเวชระเบียนผู้ป่วย

ผลการศึกษา: ผู้ป่วยได้รับการวินิจฉัยว่ามีผนังกันหัวใจห้องล่างรั่วบริเวณส่วนกล้ามเนื้อขนาด 15 มม. ร่วมกับมีกล้ามเนื้อ papillary muscle ของลิ้นหัวใจไตรคัสปิดฉีกขาดแม้จะได้รับการผ่าตัดปิดรูรั่วและซ่อมลิ้นหัวใจแล้วผู้ป่วยยังคงหลงเหลือรูรั่วที่ผนังกันหัวใจห้องล่างอยู่ขนาด 10 มม. ร่วมกับอาการหัวใจล้มเหลวที่ยังไม่ดีขึ้นแม้จะให้ยารักษาแล้วก็ตาม ผู้ป่วยจึงได้รับการสวนหัวใจเพื่อปิดรูรั่วดังกล่าวด้วย Amplatzer muscular VSD occluder หลังการสวนหัวใจผู้ป่วยหลงเหลือรูรั่วขนาด 2 มม. ร่วมกับอาการหัวใจล้มเหลวที่ดีขึ้นจนหยุดยาได้และติดตามไปครบหนึ่งปีพบว่ารูรั่วดังกล่าวปิดสนิทในที่สุด

สรุป: ภาวะรูรั่วที่ผนังกันหัวใจห้องล่างที่เกิดตามหลังอุบัติเหตุกระดูกแตกที่ทรวงอกพบได้ไม่บ่อย การรักษาโดยการ

Background and Objective: Cardiac injury following blunt chest trauma is uncommon and usually undiagnosed, which could eventually lead to morbidity and mortality. This case report describe a 17-year-old male who developed a muscular ventricular septal rupture and severe tricuspid regurgitation due to disruption of all its papillary muscles causing by direct blunt chest trauma as a consequence of motorcycle accident. Diagnosis and management of residual moderate size traumatic muscular ventricular septal rupture by percutaneous transcatheter closure were discussed.

Method: A descriptive study from patient's medical record.

Result: The patient had been diagnosed to have muscular VSD (15 mm) with disruption of all papillary muscles of the tricuspid valve with severe tricuspid regurgitation. After surgical repaired of VSD and tricuspid valve, he remained had 10 mm. muscular VSD with congestive heart failure without responsive to medical treatments. Transcatheter VSD closure with Amplatzer muscular VSD occluder was done successfully with residual 2 mm. VSD shunt. All of the medication treatments of congestive heart failure were stopped with dramatic improvement of congestive heart failure symptoms. One year after that, the VSD was completely closed.

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ผ่าตัดปิดรูรั่วเป็นการรักษาที่ได้มาตรฐานถ้าผู้ป่วยมีภาวะหัวใจล้มเหลวจากเลือดไหลล้นตั้งจจากหัวใจห้องซ้ายไปห้องขวา การรักษาโดยการสวนหัวใจเพื่อปิดรูรั่วดังกล่าวพบว่าเป็นการรักษาที่ประสบผลสำเร็จและปลอดภัยดังรายงานผู้ป่วยรายนี้

คำสำคัญ: การสวนหัวใจเพื่อปิดภาวะผนังหัวใจห้องล่างรั่ว, ผนังหัวใจห้องล่างรั่วซึ่งเกิดจากอุบัติเหตุกระแทกที่ทรวงอก, อุบัติเหตุกระแทกที่ทรวงอก

Conclusion: Traumatic VSD consequent from blunt chest trauma is uncommon. The surgical treatment of VSD closure is the standard treatment for the patient with congestive heart failure symptom from left to right shunt lesion. Transcatheter VSD closure is safely and successfully closed as described from this report.

Keywords: Percutaneous transcatheter closure, traumatic ventricular septal defect, blunt chest trauma

ศรีนครินทร์เวชสาร 2558; 30 (3): 305-310. ♦ Srinagarind Med J 2015; 30 (3): 305-310.

Case report

A previously healthy 17-year-old man, had motorcycle accident collided with paddy wagon. He was admitted at a province hospital with acute deterioration of his functional class from class I (normal) to class III. He was referred to Maharat Nakhon Ratchasima Hospital (the tertiary care hospital). Physical examinations reveal newly-identified pansystolic murmur gr III/VI at left lower parasternal border with active precordium, left ventricular (LV) heave, leftward & downward shift of point maximal impulse (PMI), and hepatomegaly. Chest radiographic finding demonstrated cardiomegaly with increased pulmonary vasculature and pulmonary venous congestion (Figure 1). Then transthoracic echocardiography was performed and demonstrated muscular ventricular septal rupture (VSR) 15 mm with left to right shunt, moderate tricuspid regurgitation (TR) with peak systolic pressure gradient across VSD of 32 mmHg (Figure 2) and severe left ventricular enlargement (left ventricular internal diameter in diastole, LVIDd=7cm [LVIDd z score = 5.5 cm]). The left ventricular systolic function was in the normal limit (left ventricular ejection fraction, LVEF = 58%). His cardiac enzyme tests including creatinine kinase (CK-MB): 0.5 ng/ml (0.6-6.3) and troponin-I: 0.05 ng/ml (0.03-0.5) were normal. The authors decided to close the traumatic VSR along with open heart surgery with tricuspid valve (TV) repair. The intraoperative findings demonstrated moderate cardiomegaly, disruption of all papillary muscles of the TV with severe TR and traumatic VSR at mid interventricular septum

diameter of 1.5 cm. Operation included patch closure of VSR along with commissuroplasty at anteroseptal commissure and TV annuloplasty with CE tricuspid ring no. 32). After the operation he still had residual pansystolic murmur gr II/VI at LLSB with functional class II-III. The echocardiography demonstrated the residual defect from his repaired VSR at the mid-muscular part [which from now called residual muscular ventricular septal defect



Figure 1 Chest X-ray in postero-anterior view on admission

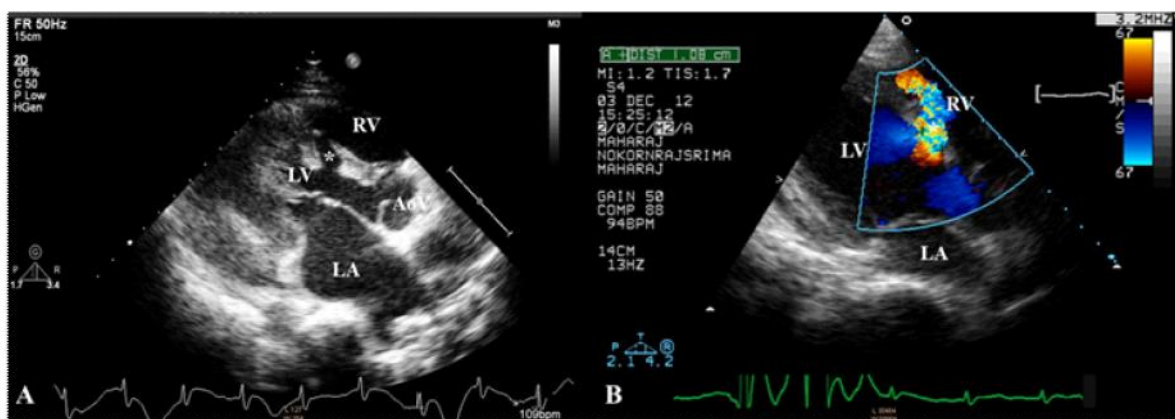


Figure 2 A 2-dimension echocardiography in parasternal long axis view demonstrated (A) echocardiographic drop-out in mid-muscular area represented VSR (*) and color Doppler (B) demonstrated left to right shunt across VSR

AoV, aortic valve; LA, left atrium; LV, left ventricle; RV, right ventricle; VSR, ventricular septal rupture

(VSD)], diameter of 10 mm with left to right shunt and the peak systolic PG across VSD of 23 mmHg with residual mild TR. He was treated with medications [furosemide (40) 1x2, Spironolactone (100) ½ x 1, Enalapril (5) ½ x 2] for two weeks, but the clinical heart failure was not improved. After team evaluation, pediatric cardiologist decided to perform percutaneous transcatheter device closure for his residual muscular VSD. Under general anesthesia, the right femoral vein and artery were punctured and percutaneously inserted the femoral sheath no. 6 and 5, respectively. The transesophageal echocardiography was performed parallel with this procedure and confirmed the residual mid-muscular VSD with diameter of 12mm. and left to right shunt. Heparin (50 IU/kg) and cefazolin (50 mg/kg) were administered intravenously during the procedure. The right and retrograde left heart catheterizations were performed. The hemodynamic data showed moderately elevated right ventricular systolic pressure. (Table 1) Route of transcatheter was accessed through the right femoral vein and right femoral artery. Angiography in the left ventricle at antero-posterior (AP) and lateral were performed to profile and localize the VSD including its relationship to the surrounding area. The VSD was measured at the end-diastolic phase to measure its maximal diameter (12 mm.) by TEE and angiography. A 5 Fr right Judkin catheter was retrograde inserted from

right femoral artery sheath, the descending aorta, then passed from the left ventricle through the VSD into right ventricle and right atrium. An arteriovenous loop was set up through a right femoral vein sheath and right femoral artery sheath with a Terumo guide wire no 0.032, J tip, 260 cm. A 6 Fr Gensini catheter was advanced over the arteriovenous loop wire from the right femoral vein sheath, inferior vena cava, right atrium, right ventricle, through VSD into the left ventricle. Then, the arteriovenous loop wire was pulled out and this catheter was positioned in the left ventricle. The Amplatzer guide wire no 0.032, 260 cm. was inserted into the Gensini catheter until its tip positioned in the left ventricle, then, the Gensini catheter was pulled out. The AMPLATZER™ long delivery sheath (8 Fr) was introduced over this guided wire until its tip positioned in the left ventricle, then, its introducer was pulled out. The AMPLATZER™ Muscular VSD Occluder no. 14 was loaded in this long sheath. Through the long sheath, this occluder was deployed under fluoroscopic control and echocardiographic guidance to be in proper position.

Table 1 Hemodynamic data before and after device closure of the ventricular septal rupture

Site	Pressure (mm Hg)	
	Pre VSD closure	Post VSD closure
RV	45/0-11	25/0-10
LV	85/0-14	90/0-13

Angiography in the left ventricle demonstrates minimal left to right shunt across the central part of the occluded device (Figure 3). Right ventricular systolic pressure decreased after device closure (Table 1). Three days after, transthoracic echocardiography showed the residual small VSD (2 mm) at the lower rim of in placed AMPLATZER™ device. The left ventricular internal diameter in diastole:LVIDd decreased from 7 cm to 4.7 cm (LVIDd Z score = 5.5 cm). The LV systolic function was returned to normal (LVEF=68%, FS=38%). The tricuspid valve had mild regurgitation with peak systolic pressure gradient across tricuspid valve of 24 mmHg. There was no aortic regurgitation. The functional class improved from class III to class I (normal). The furosemide, spironolactone, and enalapril could be discontinued. Patient received only clopidogrel (75 mg) 1 tablet once daily for 6 months after the procedure. One month later, transthoracic echocardiography demonstrated residual small shunt at lower rim of device without significant hemodynamic left to right shunt (calculated Qp:Qs equal to 1.2:1) (Figure 4). Follow up echocardiography at one year after device closure demonstrated completely VSR closure (Figure 5).

Discussion

VSR following closed thoracic trauma is an unusual complication^{1,2}. Only 5% of closed thoracic traumas are complicated with a VSR^{1,3}. There are two mechanisms cause rupture of the ventricular septum. The first occurs when blunt trauma directs on the sternum in antero-posterior direction and compresses the heart against the spine in end-diastole to early systole while the ventricular chamber is at its maximal volume and the atrioventricular valves has just been closed. This could be direct injury to the ventricular septum and the atrioventricular valves at the same times^{1,3}. The second is caused by myocardial infarction which secondary from damage of coronary blood flow^{1,3}. Since patient in this case report had both VSR and rupture of the tricuspid valve's chordae tendinae and there was no evidence injury of myocardium by normal cardiac enzymes, the authors speculated that his injury to heart could be from the first mechanism.

Conservative treatment is recommended if the left to right shunt flow is lesser than 1.5:1, because small defect may be spontaneous closure^{3,4}. However, if there is significant left to right shunt flow (>1.5:1), this could

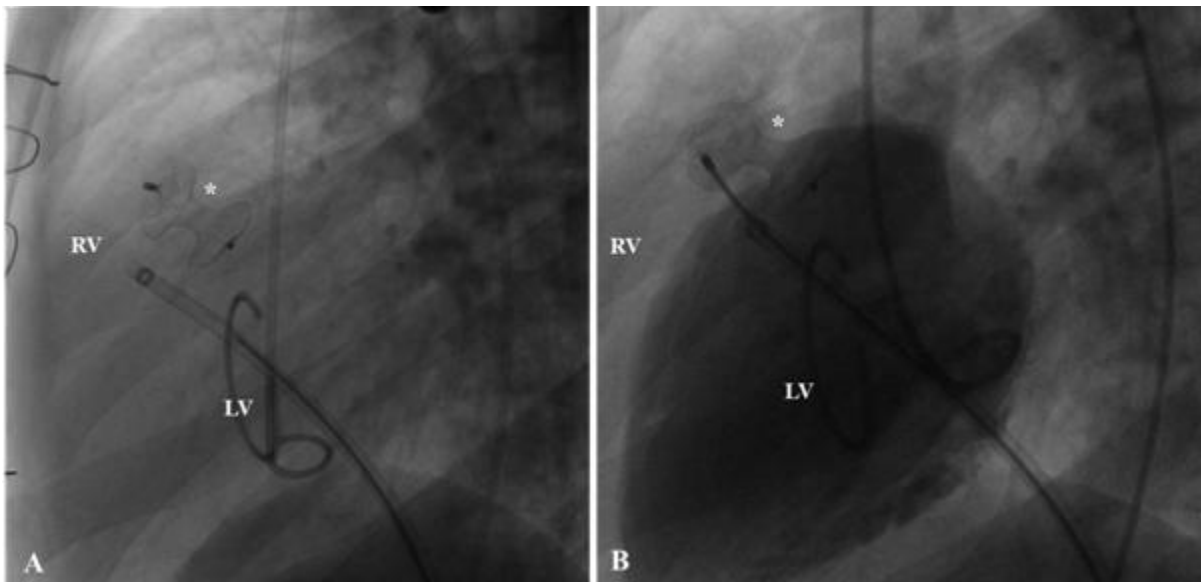


Figure 3 Fluoroscopic figure after device closure (A) demonstrated device (*) after full deployment and left ventricular angiography (B) demonstrated contrast dye filled in dilated left ventricle without minimal contrast flow across the device

LV, left ventricle; RV, right ventricle

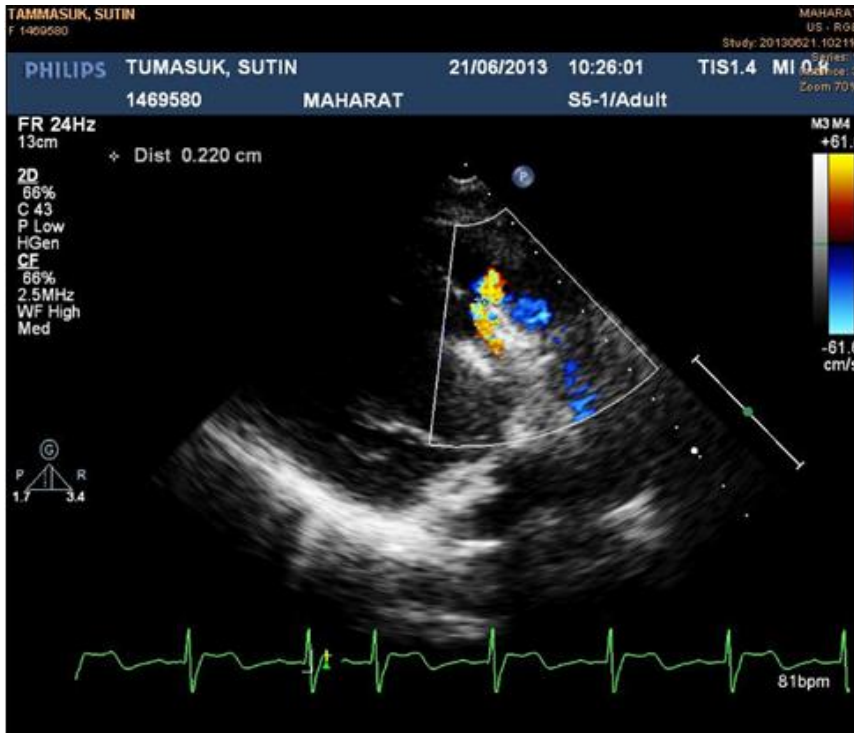


Figure 4 A 2-dimension echocardiography in parasternal long axis view demonstrated residual small shunt at lower rim of Amplatzer muscular VSD device occluder after transcatheter VSD closure 1 month later.

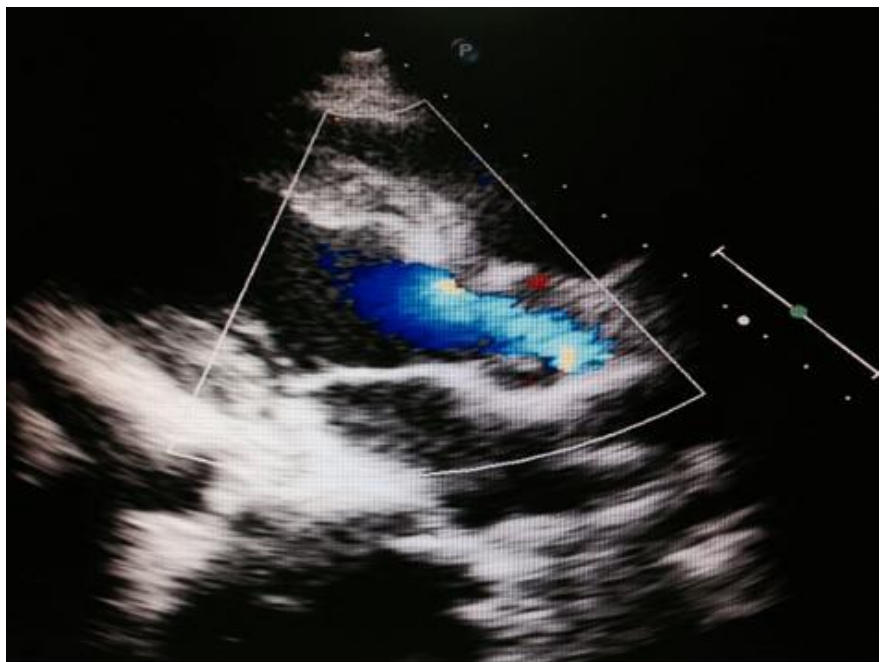


Figure 5 A 2-dimension echocardiography in parasternal long axis view demonstrated complete VSD closure after transcatheter VSD closure 6 month later.

lead to increased pulmonary blood flow, significant volume load on the left atrium and left ventricle, and developing of pulmonary venous congestion from increased left atrial pressure. Treatment to decrease the left ventricular afterload (decreased systemic vascular resistance) by using arterial vasodilators along with

inotropes and diuretics must be administered. If these treatments fail to improve the hemodynamic instability, the urgent surgery is required. If there are both valves injury and VSR, surgery would be recommended like in this case report. The surgical closure of VSR is standard treatment. However, in some situations,

especially when VSR is in the mid-muscular area, the demarcation of VSR might be challenging to delineate while approaching from the right ventricular surface. The alternative management is transcatheter closure. Studies reported 6 cases with Amplatzer muscular VSD occluder, 2 with Amplatzer ASD occluder, 1 with Amplatzer perimembranous VSD occluder and Amplatzer PFO device occluder with all successfully, respectively⁵⁻¹⁰. This patient underwent successful repair of the tricuspid valve with residual mild regurgitation, however, there was still large residual defect after VSR patch closure. The authors selected to use the device closure of this residual defect of the repaired VSR successfully without any complications.

We conclude the thorough physical examinations on cardiovascular system should be part of evaluation in all victims from blunt trauma. If the changing in functional class, newly heart murmur, and signs of congestive heart failure, further full investigations for associated cardiac injury should be warranted. Isolate rupture of the ventricular septum or residual defect of repaired ventricular septal rupture could be safely and successfully closed by VSD device occluder.

Conclusion

Traumatic VSD consequent from blunt chest trauma is uncommon. The surgical treatment of VSD closure is standard treatment for the patient with congestive heart failure symptom from left to right shunt lesion. Transcatheter VSD closure is safely and successfully closed as described from this report.

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