Isolated Ventricular Septal Defect Following Blunt Chest Trauma

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**Introduction:** The spectrum of injuries resulting from blunt cardiac trauma is as broad as the clinical presentation (1, 2). We present a case of traumatic VSD. A previously healthy 16 year old, 80-kg white male, was involved in a MVA. He was a restrained driver and the airbag was reportedly deployed. He was diagnosed with bilateral open tibia, fibula, and femur fractures. He received 3L of NS and 3 units of packed red cells at an outside hospital and was air lifted to our hospital for further management. In the emergency room (ER), the patient was awake, alert and in severe pain. His blood pressure was 100/50 mm Hg, heart rate of 140 beats/min, respiratory rate of 24/min and his oxygen saturation was 99% on a non-rebreather oxygen mask. His physical examination was unremarkable except for bilateral lower extremity fractures and bruising on his chest. He had a negative Focused Assessment with Sonography for Trauma (FAST) in the ER. FAST is a limited ultrasound examination directed solely at identifying the presence of free intraperitoneal or pericardial fluid. CT scan of the chest showed air space disease in the left lower lung suspicious for lung contusion. CT scan of the abdomen and pelvis showed multiple liver lacerations and free fluid in the upper abdomen. Head and c-spine CT scans were normal. The patient was taken to the operating room for emergency exploratory laparotomy due to increasing abdominal distension and continued relative hypotension despite fluid resuscitation. Anesthesia was induced with etomidate and succinylcholine. Immediately after, he became profoundly hypotensive (SBP of low 50s) and ST depression was noted on the EKG. He was aggressively resuscitated with fluids, blood products, phenylepherine and epinephrine, with good response. Initial CVP was 24 cm H2O. Exploratory laparotomy revealed a liter of free blood in the abdomen and multiple liver lacerations, one of which was bleeding and was controlled with packing. Due to continued hypotension, a TEE was obtained which revealed a large mid-muscular VSD with a large left-to-right shunt. There was no pericardial effusion and the biventricular function was normal. The orthopedic surgeons then proceeded to quickly stabilize the lower extremity fractures. The patient became progressively more hypotensive requiring escalating doses of dopamine and epinephrine infusions. Cardiac surgery service was consulted which recommended immediate repair of the VSD. Before he was put on cardiopulmonary bypass, a TEE was done which confirmed the diagnosis (Fig.1 and 2). The right ventricle (RV) was severely distended, tense and hypokinetic. A large 2.5 cm x 2.5 cm VSD was repaired with difficulty due to the ragged edges and minimal septal remnants. The patient required sequential AV pacing for complete heart block. High doses of epinephrine, norepinephrine and milrinone were used as well as inhaled nitric oxide to reduce RV afterload. Although he weaned successfully from CPB, his cardiac, renal and pulmonary function continued to worsen necessitating extracorporeal membrane oxygenation (ECMO). His hospital course was further complicated by renal failure, vancomycin resistant enterococcus sepsis, and no recovery of cardiac function. The patient expired on the 10th postoperative day due to multi-system organ failure and sepsis.

**Discussion:** Reports of blunt cardiac trauma in the pediatric setting are rare (3,4). This may be because in cases of blunt trauma, cardiac injury is the most commonly overlooked injury. Traumatic VSD occurs most commonly in the muscular portion of the interventricular septum near the cardiac apex (5). The mechanism of injury of ventricular septal rupture is thought to occur when there is an anteroposterior compression vector that serves to crush the heart between the sternum and the vertebrae. The spectrum of physical presentation can be quite wide. Signs of associated trauma to the chest may or may not be visible. The pulmonary examination may either be normal or consistent with pulmonary edema. With larger shunts, usually associated with defects larger than 2 cm, jugular venous distention may become apparent (6). Delay in the diagnosis often occurs because of the focus toward commonly injured organs
and delayed septal rupture secondary to myocardial necrosis and perforation in an area of contusion (7). Echocardiography has proven to be one of the most effective screening tools to aid in the diagnosis of traumatic VSD in the acute setting (5). Initial stabilization consists of measures to reduce left-to-right shunting across the VSD such as reducing systemic vascular resistance and increasing pulmonary vascular resistance. An intra-aortic balloon pump can be helpful in reducing left ventricular afterload and improving coronary blood flow.

The decision to perform surgical repair is made on the amount of left-to-right shunt and presence of heart failure (7). Trans-catheter closure of a posttraumatic VSD can be an alternative to surgery in symptomatic patients with a systemic-to-pulmonary ratio > 1.5 following a blunt chest trauma or persistent shunt after surgical repair. A conservative approach has been recommended for small traumatic VSD with a pulmonary-to-systemic ratio < 2:1 because these defects may be well tolerated and close over time (8). Prognosis after traumatic VSD is directly related to the size of the defect and concomitant injuries (25 percent mortality rate with VSDs less than 2 cm versus 71% mortality rate if the defect was greater than 2 cm)(6). CHF often develops secondary to increased pulmonary arterial pressures in association with defects larger than 1 cm in diameter.

**Conclusion:** A high index of suspicion, with early application of echocardiography or cardiac catheterization, remains critical to prompt diagnosis of traumatic VSD. Serious cardiac injury should be included in the differential diagnosis of any patient who shows rapid hemodynamic deterioration after blunt trauma.

**Refs:**

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