Traumatic tricuspid regurgitation and right-to-left intra-atrial shunt--an unusual complication of a horse-kick.

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TRAUTAMATIC TRICUSPID REGURGITATION AND RIGHT-TO-LEFT INTRA-ATRIAL SHUNT – AN UNUSUAL COMPLICATION OF A HORSE-KICK

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Abstract
A 63 year old male presented with sudden onset chest pain and dyspnoea following a kick to the praecordium while castrating a horse. Physical examination revealed evidence of horse-print on the anterior chest wall (Figure 1A), and a harsh pan-systolic murmur loudest at the left sternal edge. Transthoracic echocardiography showed evidence of flail tricuspid valve leaflets, severe tricuspid regurgitation and a widely patent foramen ovale with a right-to-left shunt. Due to progressive severe systemic hypoxemia the patient underwent emergent surgical intervention. Operative findings confirmed rupture of the anterior and septal tricuspid valve papillary muscles. Successful papillary muscle reattachment was performed in association with tricuspid annuloplasty and suture closure of his patent foramen ovale. Disruption of the tricuspid valve is well described as a consequence of blunt trauma to the chest wall and is often well tolerated, coming to light many years post injury. Valve disruption due to rupture at the papillary muscle level, however, typically results in greater severity of tricuspid regurgitation and the abrupt rise in right-to-left shunting across a patent foramen ovale. Where hemodynamic compromise ensues, prompt surgical intervention is mandated.

Case Report
A 63 year old male with a background history of known coronary artery disease and coronary artery bypass surgery 20 years previously, presented with sudden onset chest pain and dyspnoea after suffering a kick to the praecordium while castrating a horse. Physical examination revealed evidence of horse-print on the anterior chest wall (Figure 1A), and a harsh pan-systolic murmur loudest at the left sternal edge. Transthoracic echocardiography showed evidence of flail tricuspid valve leaflets (Figure 2A), with severe tricuspid regurgitation and a widely patent foramen ovale (Figure 2B) with a right-to-left shunt. Coronary angiography revealed severe native 3-vessel disease and bypass graft studies revealed 2 arterial vein grafts and a patent internal mammary artery graft to the left anterior descending artery. Three dimensional echocardiography demonstrated a patent foramen ovale (pO2 5.8 kPa [43.5 mm Hg], FIO2 1.00), the patient proceed to emergent surgery. Operative findings confirmed rupture of the anterior (figure 1B, arrow) and smaller septal tricuspid valve papillary muscles, as well as the presence of a patent foramen ovale. Successful papillary muscle reattachment was performed in association with tricuspid annuloplasty, suture closure of his patent foramen ovale and 2-vessel coronary bypass grafting. He was discharged from hospital well on day 7 post-op and has made uneventful progress to date.

Figure 1: (A) Photograph of anterior chest wall showing evidence of horse-print. (B) Per-operative photograph with right atrium opened to expose tricuspid valve annulus and prolapsed papillary muscle

Figure 2: Two-dimensional echocardiography images (A, apical 4-chamber; B, subcostal views)

Discussion
Valve injury is a rare complication of blunt cardiac injury. Valve disruption in association with patent foramen ovale and a secondary rise in right atrial pressure is often well tolerated, coming to light many years post injury. Valve disruption due to rupture at the papillary muscle level, however, typically results in greater severity of tricuspid regurgitation and the abrupt rise in right-to-left shunt across a patent foramen ovale. Where hemodynamic compromise ensues, prompt surgical intervention is mandated though in clinically stable cases the role of intervention is less certain.

Isolated tricuspid valve injury at the level of the papillary muscle, on the other hand, is less frequently reported and, as in this case, may present with hemodynamically well tolerated and relatively clinically silent, first coming to light many years post injury. The proposed mechanism of injury is a forcible compression of the right ventricle resulting in a transient outflow obstruction accomplished by an abrupt rise in right ventricular pressure leading to acute papillary muscle rupture. A secondary rise in right atrial pressure occurs, in a patient with a patent foramen ovale, resulting in a right-to-left shunt, potentially causing severe hypoxemia.

Left-sided AV valve disruption due to rupture at the papillary muscle level, however, typically results in greater severity of tricuspid regurgitation and the abrupt rise in right-to-left shunting across a patent foramen ovale. In cases associated with hemodynamic compromise, prompt surgical intervention is mandated though in clinically stable cases the role of intervention is less certain.

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References

Transthoracic echocardiography showed evidence of flail tricuspid valve leaflets, severe tricuspid regurgitation and a widely patent foramen ovale with a right-to-left shunt. Due to progressive severe systemic hypoxemia the patient underwent emergent surgical intervention. Operative findings confirmed rupture of the anterior and septal tricuspid valve papillary muscles. Successful papillary muscle reattachment was performed in association with tricuspid annuloplasty and suture closure of his patent foramen ovale. Disruption of the tricuspid valve is well described as a consequence of blunt trauma to the chest wall and is often well tolerated, coming to light many years post injury. Valve disruption due to rupture at the papillary muscle level, however, typically results in greater severity of tricuspid regurgitation and the abrupt rise in right-to-left shunting across a patent foramen ovale. Where hemodynamic compromise ensues, prompt surgical intervention is mandated.