

Case Report

Trauma of the Heart and Great Vessels

Zan Mitrev¹, Tanja Anguseva^{2*}, Zvonko Atanasov³

¹Surgery Department, Special Hospital for Surgery Fillip II, Macedonia

²Intensive Care Unit, Special Hospital for Surgery Fillip II, Macedonia

³Radiology Department, Special Hospital for Surgery Fillip II, Macedonia

***Corresponding author:** Tanja Anguseva, Special Hospital for Surgery Fillip II, Skopje, Macedonia. Tel: +389 23091500; Fax: +38923104947; E-Mail: tanja.anguseva@filipvtori.com

Citation: Mitrev Z, Anguseva T, Atanasov Z (2017) Trauma of the Heart and Great Vessels. Cardiol Res Cardiovasc Med 2: 115. DOI: 10.29011/2575-7083.000015

Received Date: 10 May, 2017; **Accepted Date:** 26 May, 2017; **Published Date:** 2 June, 2017

Abstract

Thoracic great vessel and cardiac trauma are characterized by anatomic location and mechanism of injury: blunt or penetrating. Management strategies are also directed by the extent and mechanism of injury. Advances in imaging and catheter-based technologies have allowed easier and more accurate diagnosis and less-invasive treatments. Although the advantages of endovascular techniques are attractive, open surgical repair remains the definitive treatment for many of these thoracic injuries. Given the increasing sophistication of these technologies and the demonstrated usefulness of a disease-oriented approach toward patient management, trauma centers have adopted a multidisciplinary team model for management of multitrauma victims. In this review, the authors detail the diagnosis and management of blunt or puncture aortic, non-aortic great vessel, blunt cardiac, and penetrating cardiac injuries.

Background

In modern society and frequent crush accidents, blunt trauma has become a major health problem. Improvements in the techniques of transporting injured patients and in the care given outside the hospital have increased the number of patients with severe injuries who reach the emergency room alive. Nevertheless, chest injuries are the cause of many deaths [1-3]. Injury to the heart is involved in 20 percent of road-traffic deaths, and the thoracic aorta or arch vessels in 15 percent [4-7]. In clinical series of patients with blunt trauma to the chest, the rate of cardiac injury varies widely, depending on diagnostic criteria, but then incidence would be around 15 percent; for injury to the thoracic aorta or arch vessels, the figure might be 4 percent (Table 1).

Population and Type of Injury	Incidence	
	Autopsy studies	Clinical series
	percent	
Cardiac injury	15-20	16-76
Persons with blunt chest trauma	5	Some case reports
Cardiac trauma		
Persons with cardiac trauma	2	Some case reports
Valve rupture		
Coronary-artery rupture	36-65	0,3-0,9
Chamber rupture		
Right atrium	10-15	36-65
Right ventricle	19-32	17-32
Left atrium	1-7	20-31
Left ventricle	17-44	11-15
More than one chamber	23-32	6-10

Injury to the great arteries and arch vessels	15-17	
Persons with blunt chest trauma	8-23	4
Injury to the great arteries	70-86	0-10
Persons with injury to the thoracic aorta	0-14	80-90
Ascending aorta and arch	2-20	0-5
Aortic isthmus	8 (Fulda et al.,9Kato et al.,10,and Baumgart et al.11)	5-10
Aorta at the aortic hiatus		20-61
More the one site (including arch arteries)		0-26
Persons with injury to the arch arteries	NA	
Innominate artery	NA	12-19
Left or right common carotid artery		13-19
Right subclavian artery	NA	
Left subclavian artery	NA	

Rates of incidence are from the medical literature. Specific criteria for inclusion in the studies varied. Figures for cardiac injury are from Shorr, et al., [1], Santavirta and Arajärvi, [3] Parmley, et al. [4]. Figures for injury to the great arteries and arch vessels are from Shorr, et al. [1] Williams, et al. [6] Strum, et al. [7].

Table 1: Incidence of Injury To The Heart and Great Vessels in Persons with Blunt Chest Trauma.

Cardiac Trauma

Mechanisms and Types of Injury

Massive compression of the chest may crush the heart, ascending aorta, or innominate artery between the sternum and the thoracic vertebrae. Direct injury by a fractured sternum may damage the right ventricle or the ascending aorta. Traction or torsion may tear the heart and the thoracic aorta at points of attachment: the junction of the venae cavae and pulmonary veins to the atria, the aortic annulus, the origin of the arch vessels, the aortic isthmus, and the aortic hiatus (Figure 1).

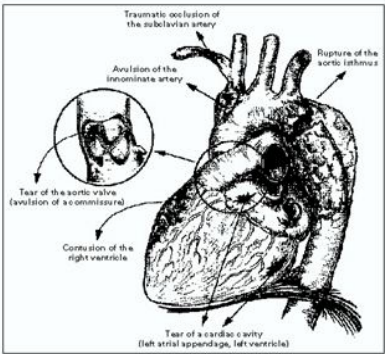


Figure 1: Heart Trauma.

Injuries to the heart and great arteries most commonly encountered in patients with blunt chest trauma [6]. A sudden rise in blood pressure during compression of the chest may injure the cardiac valves or lacerate the ventricular wall or septum [7]. The risk to the valves varies with the different events of the cardiac cycle. In early diastole, the aortic valve is vulnerable to injury because it has just closed and is not supported by the empty left ventricle. In early systole, a competent atrioventricular valve may be damaged because of an increase in ventricular pressure. Myocardial contusion is a common injury that may impair ventricular contrac-

tion and lead to arrhythmia. It is characterized by patchy areas of muscle necrosis and hemorrhagic infiltrate [7] that can be recognized at surgery or autopsy but not with conventional imaging studies. Therefore, the clinical diagnosis of cardiac contusion is an extremely vague and subjective one, often made only in patients with a history of chest trauma [6].

Natural History

The rupture of a cardiac cavity, a coronary artery, or the intrapericardial portion of a major vein or artery is usually instantly fatal because of acute tamponade. The few patients who survive usually have tears in a cavity under low pressure. In a few patients, the hemopericardium may initially be asymptomatic, become organized, and after a few months or years, lead to constrictive pericarditis. Myocardial contusion induces cardiac failure that usually improves with time. Areas characterized by dyskinesia recover proper function and most arrhythmias resolve within a few hours. Injury to a coronary artery can lead to myocardial infarction, either immediately or after a few hours, because of spasm or dissection of the arterial wall [4]. Regurgitation from valvular tears tends to worsen with time. Cardiac failure due to traumatic aortic insufficiency or mitral insufficiency develops within a few weeks; failure due to tricuspid insufficiency may appear only after several years.

Diagnosis

A patient with angina-like chest pain or progressive dyspnea after trauma must be suspected of having a cardiac injury. Many patients with cardiac injury, however, do not present with characteristic symptoms [2,7].The physical examination suggests a heart injury if complex arrhythmias, a precordial thrill, or a murmur is present, but these specific signs of heart injury are often lacking. In some patients, cardiac injury is reflected only by the hemodynamic status. Systemic hypotension and elevated venous pressure are important signs of cardiogenic shock. The jugular veins, however,

may not appear distended in patients who have bled from other lesions, and severe or refractory hypotension may be the sole sign of a cardiac injury.

Serial 12-lead Electrocardiography (ECG), Holter-monitor recordings, measurements of cardiac-enzyme levels, and radionuclide tests have not been proved to have diagnostic reliability in cardiac injury and hence are not suitable screening tests. Arrhythmias can have causes other than cardiac injury, and Creatine Kinase MB (CK-MB) isoenzymes can be produced by other injured organs [7]. Furthermore, because of the small amount of myocardial necrosis produced in a lacerated myocardium (or after myocardial contusion), enzyme measurements can still have normal results even though severe cardiac failure may ensue.

Bedside echocardiography enables rapid examination of the heart and thoracic aorta with little interference with other diagnostic or therapeutic procedures; it has become an important diagnostic tool in chest trauma. With multiplane analysis and color-flow Doppler imaging, echocardiography can be used to detect anatomical anomalies (pericardial effusion, areas of ventricular dyskinesia, and valvular dysfunction) and physiologic anomalies of the heart (abnormal blood-flow patterns). Transesophageal echocardiography frequently requires intubation, and cannot be performed in patients with severe trauma of the face or the cervical spine.

Assessment and Management

In patients admitted to the hospital with blunt trauma to the chest or with multiple injuries, there should be a high degree of suspicion that a cardiac injury has occurred. Routine tests should include: base-line chest radiography, ECG, and measurement of cardiac-enzyme levels. The prognosis, however, is excellent if the patient's condition is stable. If details of the accident (for instance, bending of the steering wheel) and signs of thoracic trauma (numerous rib fractures) suggest particularly severe impact to the chest, one should monitor the patient closely for several hours. If their condition remains stable and the ECG reveals no abnormalities or only minor changes (nonspecific changes in the ST segments or T waves), these patients can be admitted to a regular ward. (A patient with angina-like chest pain, raised enzyme levels (a CK-MB level or a CK-MB fraction of more than 5 percent), or minor arrhythmias (sinus tachycardia, premature atrial beat, or unifocal premature ventricular beat) should be monitored in an intermediate care unit and evaluated with echocardiography if these symptoms persist longer than 12 hours [7]. A patient with progressive dyspnea, ischemic patterns on ECG, or complex arrhythmias, should be treated in an intensive care unit, receive specific therapy, and be investigated further, initially with echocardiography. Taking this approach to patients in stable condition does not exclude the possibility that a few cardiac lesions may not initially be recognized.

Shock in a patient after trauma is initially due to inadequate filling of the heart or acute ventricular failure. A cardiac cause of

shock should be suspected in any patient with severe chest trauma, with hypotension that is disproportionate to the estimated loss of blood, or with an inadequate response to the administration of fluid. The most frequent causes of cardiogenic shock after trauma are cardiac tamponade and ventricular akinesia [3]. These two conditions can often be identified with transthoracic echocardiography. This examination should be done rapidly in the emergency room in suspected cases and, if transthoracic studies do not permit diagnosis, transesophageal views should be obtained. The detection of a hemopericardium that impairs cardiac filling requires prompt drainage, ideally by means of a surgical subxiphoid approach in the operating room. The surgically created drainage can be profuse or persistent, in which case immediate thoracotomy is required to control bleeding. Shock from ventricular akinesia may respond to appropriate filling, to inotropic support, or to a reduction in afterload. If these measures fail, intraaortic balloon counter pulsation should be considered to enhance cardiac output (but only after a lesion of the aortic isthmus has been ruled out) [1]. Cardiogenic shock from a traumatic valvular or septal tear is rare and requires surgical correction. It may be wise to postpone this operation for one or two days if supportive measures allow it. Cardiopulmonary bypass is necessary for the repair of intracardiac lesions, and the required anticoagulation may precipitate bleeding from other injuries.

Thoracic-Aorta and Arch-Vessel Trauma

Mechanisms and Types of Injury

Deceleration and traction are the classic wounding mechanisms of the thoracic arteries. Horizontal deceleration creates shearing forces at the aortic isthmus, the junction between the relatively mobile aortic arch and the fixed descending aorta [6]. Vertical deceleration displaces the heart caudally and into the left pleural cavity and acutely strains the ascending aorta or the innominate artery. Sudden extension of the neck or traction on the shoulder can overstretch the arch vessels and produce tears of the intima, disruption of the media, or complete rupture of the arterial wall [6]. These injuries may in turn lead to dissection, thrombosis, pseudo aneurysm of the involved vessel, or hemorrhage.

Natural History

Minor lesions of the arterial wall (e.g., a mural hematoma or a limited intimal flap) have a benign course and frequently regress spontaneously. One must be aware of these lesions because, with the improved resolution of radiologic and echocardiographic studies, they are increasingly diagnosed. Pseudoaneurysms, even small ones, have an insidious course. Very few remain stable over time. They tend to expand and rupture, but may also lead to thrombotic embolization, fistulization to adjacent organs, or the compression of nearby structures.

Rupture of the thoracic aorta immediately leads to death in 75 to 90 percent of cases [5,6]. The few survivors owe their lives

only to fragile perivascular hematomas and surrounding tissues that maintained blood within the vascular lumen. The natural history of aortic rupture has been extrapolated from autopsy reports; without repair, 30 percent of initial survivors die within 24 hours after rupture and more than 50 percent die within one week [5]. These grim statistics have led aortic rupture to be considered a time bomb that often creates an atmosphere of alarm in emergency rooms. This sense of alarm, however, is excessive. Autopsies have never proved that aortic rupture itself was the cause of death, and many patients may have died of associated injuries. The results of nonoperative management of aortic rupture, initially undertaken in patients with extremely severe neurologic conditions and then used on patients in stable condition by a few teams, have confirmed that the immediate prognosis for patients who initially survive aortic rupture is less dismal than previously thought [1]. Although good results are reported by those who advocate delaying repair by a few days, no evidence currently validates delaying the repair of aortic rupture beyond the time required for the evaluation and treatment of other emergency conditions. In recent studies, death from the sudden hemorrhage of an aortic tear or from associated injuries occurred during the first hours after admission in 8 to 13 percent of patients with aortic rupture.

Bleeding from an arch vessel is usually contained by local tissues. In rare instances, the avulsion of the origin of an arch artery causes massive bleeding into a pericardial or pleural cavity. Acute occlusion of the innominate or subclavian artery rarely leads to ischemic symptoms of the arm or hand, owing to the rich collateral network of vessels. Acute occlusion of the common carotid artery, however, may result in brain ischemia. This artery is also prone to traumatic dissection, which may extend distally to occlude the internal carotid artery after a few hours and thus produce a delayed neurologic deficit. Because of the low blood pressure inside the vessel, the tearing of a thoracic vein does not lead to major hemorrhage unless it bleeds into the pleural cavity, which can occur with the azygos or pulmonary veins [1].

Hematomas in the mediastinum from a vein injury are common and, although generally innocuous, can be troublesome if they produce a widening of the mediastinum visible on chest radiography. This sign is a hallmark of thoracic arterial injury and should be followed up with further testing and imaging.

Diagnosis

Severe deceleration may have torn a thoracic artery despite the absence of external signs of chest trauma. The circumstances of the accident may be the sole clue to such an injury. Specific signs of an injury to the thoracic aorta are rarely present. Pseudo-coarctation or decreased blood pressure in the left arm occurs in only 5 percent of patients with rupture of the aortic isthmus. Clinical signs of injury to an arch artery are more common and include

cervical or supraclavicular hematomas, bruits, and diminished peripheral pulse. Coma or a hemisindrome may occur in cases of the rupture of a common carotid artery. The denervation of an arm due to laceration of the brachial plexus is frequently associated with rupture of the subclavian artery.

Chest radiography is essential to screen for injuries of the thoracic aorta and arch arteries (Figure 2 Anteroposterior Chest Radiograph Showing Enlargement of the Upper Mediastinum after Avulsion of the Innominate Artery.). The most reliable radiographic signs of an arterial injury are a widening of the superior mediastinum, blurring of the aortic-knob contour, and enlargement of the paratracheal stripe.

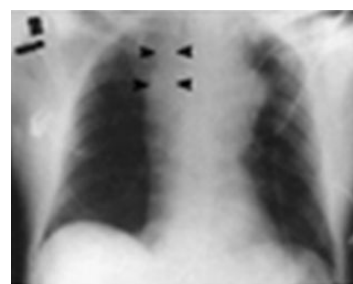


Figure 2: Enlargement of the Upper Mediastinum After Avulsion of the Innominate Artery.

Aortic angiography provides images of the entire thoracic aorta and arch vessels (Figure 3) Angiogram of the Thoracic Aorta Showing a Traumatic Pseudoaneurysm of the Aortic Isthmus (Arrows) that are easy to read, but the procedure requires some time [4]. Transesophageal echocardiography is rapid and accurate in the evaluation of the aortic isthmus [3], but does not precisely analyze the distal part of the ascending aorta and the arch vessels, because of the interfering presence of the respiratory tract.

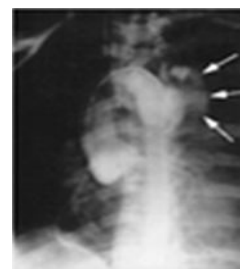


Figure 3: Traumatic Pseudo-Aneurysm of the Aortic Isthmus-Showed by Angiogram of the Thoracic Aorta.

Computed Tomography (CT) of the chest has a better predictive ability than does standard radiography, but a physician must frequently rely on indirect signs, such as a periaortic hematoma, to diagnose a rupture of the aorta, [4] and CT can miss many arch-vessel injuries. Helical CT more precisely delineates arterial lesions and has excellent accuracy (Figure 4).

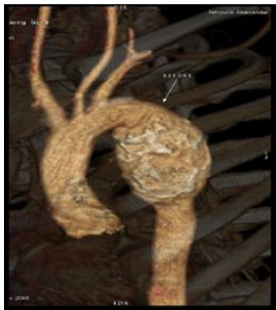


Figure 4: CT Image of the Posttraumatic Aneurysm of the Proximal Part of Descending part of Thoracic Aneurysm.

Assessment and Management

A particularly troublesome presentation of chest trauma occurs if the circumstances of the accident suggest injury due to severe deceleration but the results of chest radiography are normal. In a patient with a normal chest radiograph, the risk of an injured aorta is very low, but the diagnosis must be excluded. For this pur-

pose, CT is appropriate. Normal CT results almost certainly rule out an injury to the aorta and obviate the need for further examination. The patient should nevertheless undergo regular chest radiography (for up to six months after injury), with further investigation of any new mediastinal abnormality.

If the mediastinum is enlarged after trauma to the chest, the risk of a thoracic arterial injury is increased, and a diagnosis must be established. In this case, angiography is appropriate, because both CT and transesophageal echocardiography can miss certain vascular injuries, especially of the arch vessels. (Figure 3) shows an enlarged mediastinum caused by an avulsion of the innominate artery that was not detected by transesophageal echocardiography. Differential diagnosis obtained bleeding from spine trauma or from extremities' bonds.

Our Experiences

During 16 years, we have treated 22 patients with acute chronicle trauma consequences. All patients' data are shown in the (Table 2).

Age of patient	Type of trauma	Intervention	Outcome
18	Puncture of the heart during pericardial drainage	Urgent surgery	Exituslethalis - After 4 hours
21	Puncture of the heart during pericardial drainage	Urgent surgery	Exituslethalis - After 8 hours
67	Rupture of LAD with tamponade during angio	Urgent surgery	3 years' survival after surgery
32	Blind trauma-contusio- Constrictive pericarditis	Surgery-pericardiectomy	1 year survival after surgery
57	Blind trauma-contusio- Constrictive pericarditis	Surgery-pericardiectomy	7 months after surgery died due to gobal heart failure
58	Posttraumatic aortic arch aneurysm	Surgery	6 years' survival after surgery
59	Posttraumatic rupture of the descending aorta	Urgent surgery	4 years' survival after surgery
28	Posttraumatic dissection of the descending aorta	Conservative	5 years' survival after trauma
63	Posttraumatic dissection of the arch and descending aorta	Conservative	8 years' survival after accident
37	Acute dissection of the proximal part of descending aorta	TEVAR	7 months' survival after intervention
26	Acute dissection of the proximal part of descending aorta	Urgent surgery	6 years' survival after surgery

Table 2: Characteristics of Treated Patients in Our Hospital.

Medical treatment aimed at controlling or reducing stress on the aortic wall should be performed in any patient with a suspected or identified aortic tear. Such treatment should include the prevention and treatment of hypertension, adequate sedation and analgesia, and the prevention of movement of the thoracic vertebral column. Short-acting beta-blockers reduce blood pressure, the force of the arterial upstroke, and heart rate and are particularly suitable for reducing stress on the aortic wall.

Surgery is indicated for the repair of most arterial lesions. Repair of the ascending aorta and the aortic arch usually necessitates full circulatory support with extracorporeal circulation or by stent transcuteaneous stent implantation. In patients with trauma of descending aorta, transcuteaneous endovascular replacement is a

method of choice for treatment. CT scan control is obligatory for evaluation of graft position (Figure 5).



Figure 5: CT Control of Graft Positioning, in the Same Patient from (Figure 4).

Conclusion

All types of trauma in cardiothoracic surgery needs to be recognized on time, diagnosed. Multidisciplinary team model ensures adequate and prompt treatment with good clinical patient outcome.

References

1. Shorr RM, Crittenden M, Indeck M, Hartunian SL, Rodriguez A (1987) Blunt thoracic trauma: analysis of 515 patients. *Ann Surg* 206:200-205.
2. Galan G, Penalver JC, Paris F, Caffarena JM Jr, Blasco E, et al. (1992) Blunt chest injuries in 1696 patients. *Eur J Cardiothorac Surg* 6:284-287.
3. Santavirta S, Arajärvi E (1992) Ruptures of the heart in seatbelt wearers. *J Trauma* 32:275-279.
4. Parmley LF, Manion WC, Mattingly TW (1958) Nonpenetrating traumatic injury of the heart. *Circulation* 18:371-396.
5. Parmley LF, Mattingly TW, Manion WC, Jahnke EJ Jr (1958) Nonpenetrating traumatic injury of the aorta. *Circulation* 17:1086-1101.
6. Williams JS, Graff JA, Uku JM, Steinig JP (1994) Aortic injury in vehicular trauma. *Ann Thorac Surg* 57:726-730.
7. Sturm JT, Hankins DG, Young G (1990) Thoracic aortography following blunt chest trauma. *Am J Emerg Med* 8:92-96.