Introduction

Trauma is a major health problem world-wide. Vascular trauma is an important component of this critical scenario. Incidence of vascular trauma is on rise. Vascular injury is common in poly trauma. Peripheral vascular injuries may result from penetrating or blunt trauma to the extremities. If not recognized and treated rapidly, injuries to major arteries, veins, and nerves may have disastrous consequences resulting in the loss of life or limb (**Frykberg , 1995)**.

Peripheral vascular injuries account for 80% of all cases of vascular trauma. Most of the injuries involve the lower extremities. The great majority of patients are young males. Most injuries are caused by high-velocity weapons (70% to 80%), followed by stab wounds (10% to 15%) and blunt trauma (5% to 10%). The incidence of vascular trauma in the military is comparable to the civilian areas and varies from 0.2% to 4% of injured patients (**Hoyt & Coimbra, 2001)**.

Vascular injuries are classified as: contusion, intimal disruption, puncture, lateral disruption, transaction, arterio-venous fistulae and pseudoanerysm. The lower extremities are involved in two thirds of all patients with vascular injuries **(Frykberg , 1995)**.

Classically, injury mechanisms are divided into penetrating or blunt type. Following blunt trauma, tissue injury is produced by local compression or rapid deceleration. In penetrating trauma, the injury is produced by crushing and

separation of tissues along the path of the penetrating object (**Hoyt & Coimbra, 2001)** .

In World War I, most of the vascular injuries were treated with vascular ligation, there was 49% rate of limb amputations. This was reduced to 35.8% in World War II thanks to increased attempts at vascular repairs. In Korean and Vietnam conflicts major arterial repairs were carried out and amputation rate further declined to 13%. During Afghan war, vascular reconstructions were successfully carried out even in field hospitals saving many precious limbs and lives **(Hafez et al., 2001) .**

The management and outcome of vascular repair has remarkably improved over past decade due to, better understanding of the trauma mechanism, early detection of the nature and extent of vascular injury and speedy surgical intervention aiming to revascularization **(Hafez et al., 2001).**

Vascular reconstruction is carried out by different means and ways depending upon the nature and extent of vascular injury, size and caliber of injured vessel, its area of supply, nature of concurrent trauma, general condition of the patient and available resources including expert vascular services. In modern days surgeries, 95% limbs are successfully salvaged by early surgical intervention and revascularization **(Hafez et al., 2001) .**

The continued advances in imaging and stent -graft (SG) technology have considerably expanded the indications

for endovascular approach in vascular trauma, with the potential advantage of avoiding part of the challenging problemsof conventional repair **(Nicholson, 2004).**

However, the use of intra luminal arterial stents in peripheral vessels has been described in the treatment of angioplasty induced or penetrating injuries, but their use in the management of blunt trauma has rarely been reported **(Carillo et al., 2002).**

Mechanisms and Incidences of Traumatic Vascular Injuries

Classically, injury mechanisms are divided into penetrating and blunt type. Following blunt trauma, tissue injury is produced by local compression or rapid deceleration. In penetrating trauma, the injury is produced by crushing and separation of tissues along the path of the penetrating object **(Gupta et al., 2001)** .

Injury severity is proportional to the amount of kinetic energy (KE) transferred to tissues, which is a function of the mass (M) and velocity (V) : This is valid for both blunt and penetrating mechanisms. Changes in velocity alter the kinetic energy transfer more significantly than changes in mass. This is critical when evaluating high- and low- velocity gunshot wounds and their corresponding injury potential **(Raul** et al., **2005)**.

Blunt force injuries can generally be explained by 3 mechanisms:

The **first** is when rapid deceleration causes differential movement among adjacent structures. As a result, shear forces are created and cause hollow, solid, visceral organs and vascular pedicles to tear, especially at relatively fixed points of attachment. For example, the distal aorta is attached to the thoracic spine and decelerates much more quickly than the relatively mobile aortic arch. As a result, shear forces in the aorta may cause it to rupture. Similar situations can occur at

the renal pedicles and at the cervicothoracic junction of the spinal cord **(Wisner & Hoyt, 1997).**

The second is when intra-abdominal contents are crushed between the anterior abdominal wall and the vertebral column or posterior thoracic cage. This produces a crushing effect, to which solid viscera (eg, spleen, liver, kidneys) are especially vulnerable **(Wisner & Hoyt, 1997).**

The third is external compression forces that result in a sudden and dramatic rise in intra-abdominal pressure and culminate in rupture of a hollow viscous organ (ie, in accordance with the principles of Boyle law (**Wisner& Hoyt, 1997)**.

The principle that at a constant temperature the volume of a confined ideal gas varies inversely with its pressure (**Wisner & Hoyt, 1997)** .

Another important concept in the understanding of the biomechanics of vascular injury is that of cavitation. Cavitation is a phenomenon that occurs as tissue recoils from the point of impact by a moving body, away from the object. After blunt trauma, the resulting transient tissue cavity may be caused by rapid acceleration or deceleration. Extreme strain occurs at points of anatomic fixation during the formation of these temporary cavities. Forces can be produced both along the longitudinal axis (tensile or compression strain) and across the transverse axis (shear strain). These types of forces cause deformity, tearing, and tissue failure or fracture. Following

penetrating trauma, temporary cavitation is caused by the transfer of kinetic energy from the projectile to adjacent tissue, which is followed by the formation of a permanent cavity caused by tissue displacement. This mechanism explains why vessels can be injured even without being in contact with oprojectiles frm firearms or bone fragments (**Hoyt et al., 2001).**

Incidence of vascular injuries in different body areas

1-Vascular Injuries in the Neck:

The most commonly injured structures in the neck are the blood vessels. The incidence of major vascular trauma following a penetrating neck injury is 20%. The incidence of neck arterial injuries following blunt trauma is extremely low, although in recent years, there has been an increase in reported blunt carotid injuries due to aggressive screening (**Beitsch et al., 1994).**

The mortality rate of blunt carotid injury varies from 20% to 40%, and permanent neurologic impairment occurs in 25% to 80% of the survivors **(Fabian et al., 1998**).

The incidence of vertebral artery injury following a penetrating mechanism varies from 1% to 7.5%. This variation is related to the indications for angiography, and some of these injuries may not need surgical intervention. The incidence of blunt vertebral artery injuries is low. These injuries are

commonly associated with cervical vertebral fractures (**Fabian et al., 1998**).

2- Thoracic Aortic Injuries:

Blunt aortic injury occurs following abrupt deceleration. This causes shear forces at points of anatomic fixation of the aorta and leads to transmural injuries. Most injuries are located distally to the take-off of the left subclavian artery (65%), although other segments of the thoracic aorta such as the arch (10%), the descending aorta (12%), or multiple sites (13%) may be injured. Frontal motor vehicle crashes as well as side-impact collisions are the most frequent mechanism (***Wall et al., 2004).***

Penetrating injuries to the ascending aorta are more commonly caused by stab wounds in survivors, whereas gunshot wounds are usually the mechanism of injury in the descending portion of the thoracic aorta (**Wall et al., 2004).**

3-Abdominal Vascular Injuries*:*

In contrast with military data, major abdominal vascular injuries are common in civilian practice. These injuries account for approximately 30% of all vascular injuries. Most injuries (90% to 95%) are caused by a penetrating mechanism. Approximately 10% of patients undergoing surgical exploration following a stab wound to the abdomen and 20% to 30% of those undergoing surgical exploration following a gunshot wound to the abdomen sustain a major vascular injury **(Asensio et al., 2003).**

Abdominal arterial and venous injuries occur with the same incidence. In a recent review of 302 abdominal vascular injuries, the incidence of arterial injuries was 49% and of venous injuries, 51%. The most commonly injured abdominal vessel was the IVC and it accounted for 25% of injuries, followed by the aorta (21%), the iliac arteries (20%), the iliac veins (17%), the SMV (11%), and the SMA (10%). Overall, patients with penetrating trauma had an average of 1.6 vascular injuries (**Asensio et al., 2000).**

Hospital mortality rates vary from 30% to 80% for abdominal aortic injuries and from 30% to 65% for inferior vena cava injuries. A high number of patients never reach the hospital alive, dying at the scene or during transport **(Coimbra et al., 1996).**

4- Subclavian and Axillary Artery Injuries*:*

Most injuries to the subclavian and axillary arteries occur following penetrating trauma. The incidence varies from 0.9% to 3% depending on the mechanism of injury (stab or gunshot wound). Blunt trauma is rare. These injuries occur following high-speed motor vehicle frontal crashes with significant deceleration and fracture to the clavicle or first and second ribs. The mortality rate of these injuries is high, and most patients do not reach the hospital alive (**Demetriades et al., 1999).**

5- Extremity Vascular Trauma:

The overall incidence of arterial injuries following penetrating injury to the extremity (upper or lower) is approximately 10%, in contrast with 1% following blunt trauma. The brachial, femoral, and popliteal arteries are the most frequently injured vessels in civilian as well as in military penetrating series (**Dennis et al., 1998)**. Most femoral artery injuries are the result of a penetrating mechanism, particularly gunshot wounds (**Carillo et al., 2002).**

In contrast, a blunt mechanism accounts for 20% to 75% of popliteal artery injuries. These injuries encompass 19% of all extremity arterial injuries (**Frykberg et al., 1991).**

The incidence of vascular injuries below the popliteal fossa is difficult to determine because most of these injuries, when isolated, cause no vascular compromise. One study analyzing 755 patients sustaining gunshot wounds below the knee reported 136 injuries below the popliteal fossa identified on angiography, an incidence of 18%. **(Ordog et al., 1994).**

Pathological Effects of Vascular Trauma

Types of vascular injuries: Figure (1) and table (1) (Nigel et al., 2005).

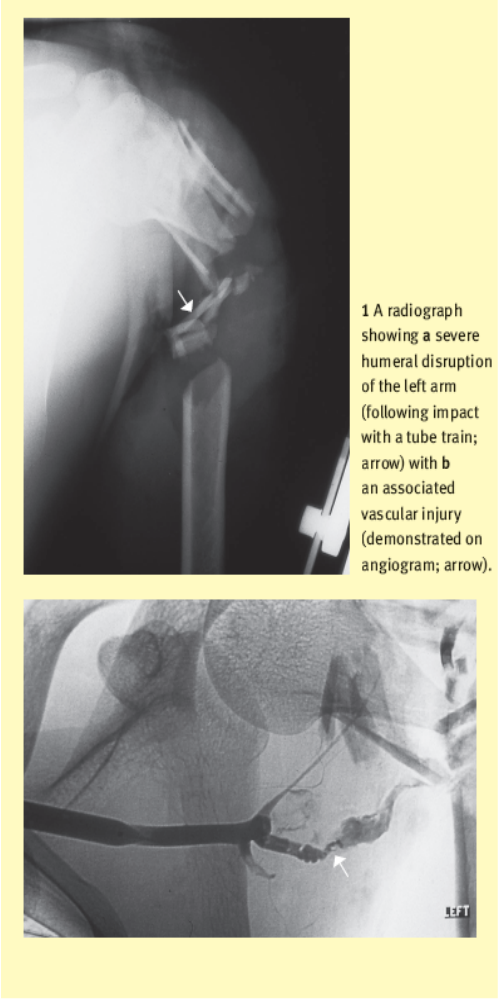
Vessel disruption is the most common vascular injury and may be partial or complete. Partial disruptions usually cause active bleeding if an open wound is close to the disruption. It may present as an expanding or pulsating haematoma.. Partial disruptionsmay not present with ischaemia because a channel for blood flow can be maintained. False aneurysms may develop if a partial disruption is unrecognized.decreases as the vessel goes into spasm and a clot develops

Intimal injuries are the second most common type of vascular injury and may lead to thrombosis of the vessel or the formation of an intimal flap, progressing to distal ischaemia. Occasionally,an intimal flap may cause a dissection, which extends with time and may become apparent later.

Small arterial contusions with limited intimal flaps may not cause distal hemodynamic compromise and may be undiagnosed. These are sometimes classified as “occult” or “minimal” arterial injuries when seen on angiography. Although these injuries carry a small risk of thrombosis, several studies have documented spontaneous healing. Concomitant arterial and venous injuries may lead to the formation of an arteriovenous fistula and partial lacerations may cause pseudoaneurysm formation.

Arteriovenous fistula formation occurs when an artery and its adjacent vein are injured. They are commonly seen after penetrating trauma and tend to present late.

Arterial spasm is rare and should not be considered as the cause of limb ischaemia after trauma.



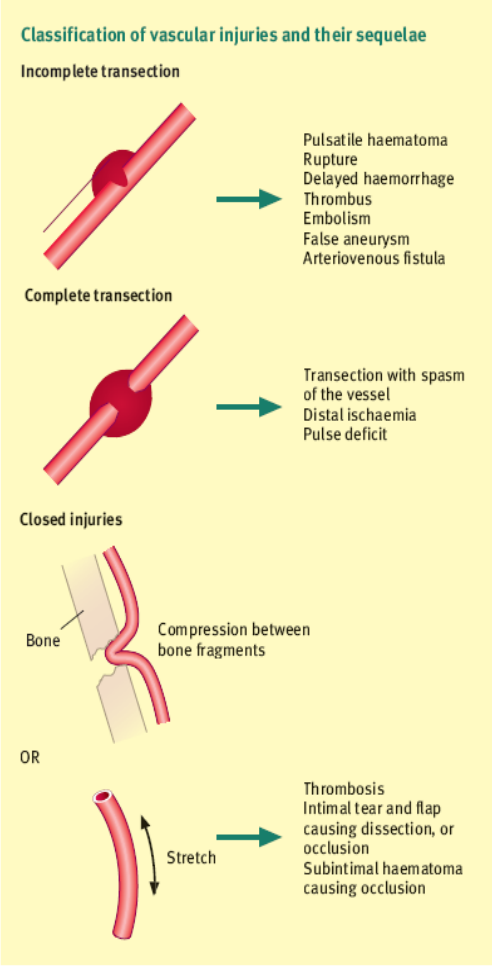


Fig (1): Classification of vascular injuries (Nigel et al., 2005).

Table (1): Types of Arterial Injury and Possible Clinical Presentation

TYPE OF INJURY	CLINICAL PRESENTATION
Partial laceration	Decreased pulse, hematoma, hemorrhage
Transection	Absent distal pulses, ischemia
Contusion	Initially, examination may be normal, may progress to thrombosis
Pseudoaneurysm	Initially, examination may be normal, bruit or thrill, decreased pulses
AV fistula	Same as pseudoaneurysm
External compression	Decreased pulses, normal pulses when fracture aligned
AV, arteriovenous.	

(Fred et al., 2005)

As noted by the preponderance of penetrating injury in the published medical literature, the vascular tree, both arterial and venous, appears to have some limited natural protection from stretching and bending, which results in fewer blunt injuries to the extremity vasculature following trauma. The smooth muscle of the arterial media protects the patient from both stretch-type injuries and minor puncture wounds, which heal spontaneously in most cases. The smooth muscle layer also offers mild protection from death due to ongoing hemorrhage (**Nanobashvili et al., 2003).**

When the arterial vessel is transected, vascular spasm coupled with low systemic blood pressure appears to promote clotting at the site of injury and to preserve vital organ perfusion better than that which occurs with ongoing uncontrolled hemorrhage. This partially explains the prehospital finding that, in the subset of penetrating trauma, limited or no fluid resuscitation until arrival at the hospital may improve patient survival and outcome (**Harkin et al., 2001).**

The importance of the pathophysiology of interrupted arterial inflow and venous outflow should be understood.Reduced tissue oxygenation depletes high energy phosphates essential to cell metabolism and increased levels of purine metabolites and cellular acidosis create an intracellular environment which, on reperfusion, richly favours the production of oxygen reactive species. These species, namely, superoxide anions, hydrogen peroxide, and hydroxyl radicals bring about IRI. Failure of local anti-oxidant scavenging systems in the face of overwhelming production of oxidants is central to injury associated with oxidative stress. These oxidants promote lipid peroxidation and lysis of cell membranes metabolising arachidonic acid to generate a cascade of potent vasoactive eicosanoids such as thromboxane A2 and leukotriene B4 which have both local and systemic pro-inflammatory effects. Local recruitment, activation and sequestration of neutrophils also release oxygen reactive species and proteases, further compounding IRI. Asystemic inflammatory response syndrome (SIRS) affects the bowel and

generates cytokine release which has implications for the liver, lungs, heart, brain, and kidneys (**Harkin et al., 2001).**

It appears that both the extent and duration of ischaemia is crucial to this process, and, therefore, any measure initiated which curtails the ischaemic period should offer some clinical benefit. Reports also suggest that gene therapy can be helpful in managing the chronic ischaemic limb and it would not be unreasonable to expect such therapies of the future to tackle the harmful effects of acute limb ischaemia (**Isner et al., 1996).**

In the lower limb, endothelial cell injury promotes increased microvascular permeability, transcapillary filtration and tissue oedema thereby gravely reducing tissue perfusion, and contributing to the compartment syndrome, endothelial activation and swelling is associated with microvascular stasis, thrombosis, aseptic muscle necrosis, Volkmann's contracture, ischaemic nerve palsy and amputation (**Harkin et al., 2001).**

Compartment syndrome (CS) is a limb-threatening and life-threatening condition observed when perfusion pressure falls below tissue pressure in a closed anatomic spacReperfusion injury refers to damage to tissue caused when blood supply returns to the tissue after a period of ischemia. The absence of oxygen and nutrients from blood creates a condition in which the restoration of circulation results in inflammation and oxidative damage through the induction of oxidative stress rather than restorae. The current body of knowledge unequivocally reflects that untreated compartment