Vascular Trauma

Chapter 1

Extremity Vascular Trauma in a Resource Poor Setting

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1. Introduction

Vascular trauma occurs in 1-2 % of civilian injuries [1] and 6.8% to 10.8% in war related injuries (Afghanistan, Iraq and Lebanon war) [2,3]. After the end of civil war in Sri Lanka in 2009 there is a reduction in the number of war related vascular injuries. But due to the increase in the number of road traffic accidents, the number of vascular trauma also increased i.e. from 1938 to 2013 road traffic injuries increased from 35.1 to 98.6 per 100 000 population in Sri Lanka. One of the reasons for this is the increased number of vehicles on the road especially two and three wheeled vehicles which are involved more with accidents [4]. Vascular injuries are potentially life or limb threatening. Trauma and vascular injuries often involve young males [5]. They are often the breadwinners of the family in this part of the world where the author works and when they lose the limb or life as a result of vascular injury it results in severe financial burden to the family and the country. In a study done at the Teaching Hospital Anuradhapura Sri Lanka, on patients presenting with major vascular injuries, 93.8% of the patients were males with the mean age of 35.0 years [5].

"Vascular injury" is a nonspecific term. Because this is used to describe the injuries to blood vessels in all parts of the body that includes neck vascular injuries, thoracic and abdominal vascular injuries, and injuries to the blood vessels of the limbs. And in addition vascular injury also describes injuries to the arteries and veins. Injuries to the blood vessels of the limbs are called extremity vascular injury. This chapter will concentrate on the extremity vascular injuries.

2. Causes and Mechanisms of Vascular Injuries

Causes of extremity vascular injuries vary from country to country. And also depending

on the presence of armed conflicts, status of the roads and the traffic, degree of violence, and any other unique things specific to the area of country. For example injuries by Trap gun accounts for 33.3% of major lower limb vascular injuries at the Teaching Hospital Anuradhapura, which is situated in the North Central province of Sri Lanka [6].

In general causes of extremities vascular injuries include;

- Road Traffic Accidents
- Fractures and dislocations
- Gunshot injuries
- Explosive injuries
- Stabs
- iatrogenic

The commonest cause of major lower extremity vascular injury at our center is the road traffic accidents, which accounts for 54.2% of the cases [7].

Iatrogenic vascular injury is defined as a vascular injury which occurs when a vessel is accessed for a diagnostic or therapeutic purpose. Incidence of iatrogenic vascular injury is on the rise due to the increase in the proportion of percutaneous intravascular interventions [8]. In a study done in Sweden the commonest artery injured due to an iatrogenic cause was the common femoral artery. Other common arteries injured were; External iliac, Superficial femoral, Brachial, Subclavian/axillary, Radial and Common carotid arteries [9]. In our practice common femoral arterial injury following accidental cannulation while attempting femoral venous line insertion is the commonest presentation after iatrogenic vascular injury [10].

The above mentioned causes are classified into either a blunt or penetrating (sharp) type of injuries. The definition of a blunt injury is that there is no penetration of the skin. Sharp injury means there is penetration wounds on the skin [11]. Any how this classification does not explain the type of injury which occurs at the vascular level. For example following a blast injury the pellets can cause multiple penetrating wounds on the skin. But the artery may be lacerated or contused. Similarly following fractures of the bones the sharp bone ends can cause penetrating type of injury, even though the mechanism was blunt by definition. Therefore a practical way of classifying the type of vascular injuries would depend on the findings at the vascular level. If the injured vessel wall is irregular or if the wall is contused it can be considered as a blunt mechanism. Whereas if there is a sharp cut with regular edges it can be considered as a sharp mechanism. Because the injury at the vascular level is the main factor which influence the management rather than the division of causes into ‘penetrating’

and ‘blunt’ injuries.

At the vascular level the following injuries occur (**Figure 1**) ;

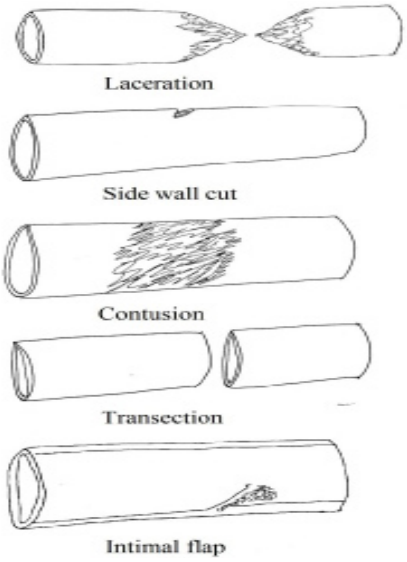

Figure 1: Types of injuries at the vessel level

a) Transection

The blood vessel continuity is interrupted. In blunt injuries the edges of the transected vessel is irregular and often tapered with thrombus at the proximal and distal ends (Figure 1). The transected ends may be held with a thin band of adventitia. When the transaction occurs due to sharp instrument the vascular ends are regular and completely separated (Figure 1)

b) Contusion

This often results from blunt injuries. The vessel wall is damaged. The resulting intimal flap (**Figure 3**) causes thrombosis leading to distal ischemia. Externally the damaged wall appears hemorrhagic and discolored (**Figure 2**).

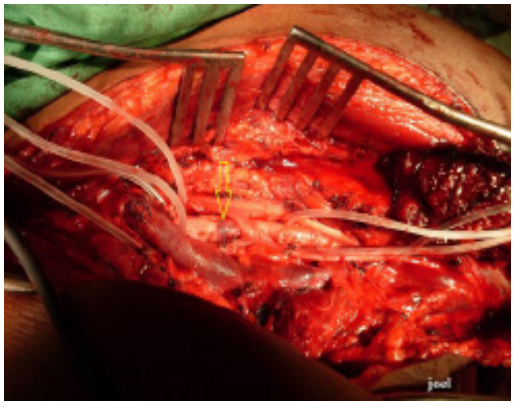

Figure 2: Blunt injury with contused (arrow) arterial wall

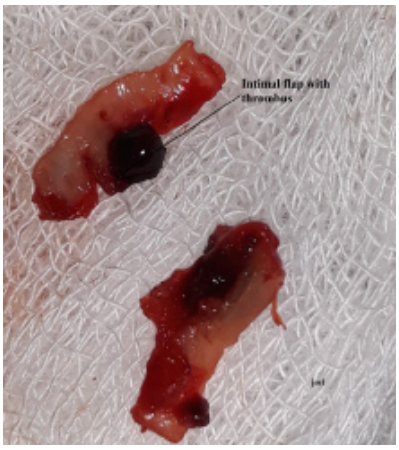

Figure 3: Intimal flap causing thrombosis

c) laceration

Side wall laceration is often caused by bone fragments and shrapnel. The part of the arterial circumference is open and the edges are irregular. This kind of injury often results in bleeding or false aneurysm formation. Also the vessel may thrombose later resulting in ischemia.

d) Side wall cut injury

This is often caused by knives and glass pieces. The cut edges are regular. Again this kind of injuries results in bleeding or false aneurysm formation (**Figure 4**).

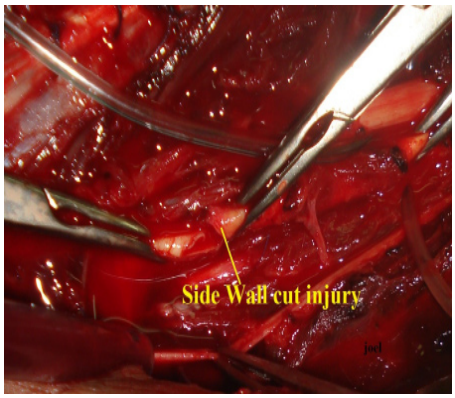

Figure 4: Side wall cut injury

e) Kinking

This happens when the vessel is caught between the fractured bone ends or caught between bones forming the joints. This results in distal ischemia due to interrupted circulation.

The above mentioned mechanisms result in; interruption of distal flow resulting in ischemia, hemorrhage or hematoma formation, false aneurysms and traumatic arteriovenous fistula formation (when both artery and vein walls are injured).

3. Clinical Features and Diagnosis

Traditionally the clinical signs and symptoms following extremity vascular injuries are

categorized into “hard” and “soft” signs.

Hard signs include;

- Active haemorrhage
- Expanding Hematoma
- Thrill or bruit at the site of injury
- Signs of distal ischemia. which includes 6 'P's

    Absent distal pulse

    Perishing coldness of the distal limb (poikilothermia)

    pallor of hands and feet

    Pain

    Paresthesia or anesthesia

    paresis or paralysis

Soft Signs include;

History of bleeding at the time of injury.

Injury close to a known neurovascular bundle or injury to the nerve in a neurovascular bundle.

Non expanding Hematoma.

In the presence of trauma assessing the hard signs and soft signs may be difficult and should be interpreted carefully. For example,

a) Absence of pulse maybe due to the fact that the patient is in hemodynamic shock due to hemorrhage. Therefore the pulse of the non-injured limb should also be examined. In addition the other signs of shock should also be looked for. In addition patients with proximal arterial injury may have distal pulses in 5 to 15% of patients [12], due to the presence of collaterals or because the arterial injury is only a sidewall injury and resulted in a false aneurysm or traumatic arteriovenous fistula and the distal flow is maintained. Again in these instances comparing the volume of the pulse with the opposite limb is helpful. And measuring the Ankle Brachial Pressure Index (ABPI) in both limbs will help (ABPI = Systolic blood pressure of the distal leg artery (either dorsalis paedis or posterior tibial whichever has the highest value) /

Systolic blood pressure of the brachial artery. Ankle brachial pressure index of less than 0.9 is significant especially when compared with non-injured limb and it should be investigated with Computed Tomographic Angiography (CTA) or Doppler Ultrasound evaluation. In elderly patients when they have associated occlusive peripheral arterial disease ankle brachial pressure index alone may not be accurate. In this instant arterial pressure index (API) i.e. dividing the systolic blood pressure of the injured limb by the systolic blood pressure of the non-injured limb will be useful. An arterial pressure index of 0.9 or less is significant. Arterial pressure index has a sensitivity of 87% and specificity of 97% [13].

b) The paresis or paralysis, paresthesia or anesthesia may occur as a result of associated nerve, head or spinal injury. Therefore the presence of these signs should be evaluated carefully and documented.

Studies show that the presence of a hard sign indicates nearly 100% chance of having a major vascular injury [14]. Therefore urgent intervention should be done when a hard sign is present. Whereas the presence of a single soft sign indicates the probability of having a vascular injury of about 10% and presence of two soft signs indicates the chance of having a major vascular injury of about 25% [15].

In addition paresthesia or anesthesia, paresis or paralysis are late signs indicating that the limb viability is in immediate threat and again urgent intervention should be carried out.

In case of vascular trauma it is essential to document the clinical findings which have clinical as well as legal implications. But the documentation is often poor in our local setting. In a retrospective study done recently on clinical notes of the vascular trauma patients at The Teaching Hospital Anuradhapura Sri Lanka, the cause of injury was properly documented only in 82.6%, and the limb viability on initial assessment was documented only in 30.7% [16]. This indicates that the importance of documentation of these findings should be taught starting from medical school curriculum and computerized documentation system with web-based links would make this documentation procedure easier in a busy emergency department. Retrieval and auditing will also be easier if the system is computerized.

Pre Hospital Care

Vascular trauma is associated with other injuries therefore the patient should be assessed completely and managed. The main concern in pre hospital care is the bleeding.

The options available for control of hemorrhage in the pre hospital setting includes pressure bandages, tourniquet and various hemostatic mechanisms(balloons, glue, etc.). Unfortunately in Sri Lanka there are no pre hospital care systems and the injured patients are brought directly to the hospital without adequate pre hospital care.

4. Initial Management of an Injured Patient

Trauma patient should be assessed and managed according to the Advanced Trauma Life Support (ATLS) protocol (ABCD). Airway and breathing takes priority before circulation. But if there is an ongoing bleeding, control can be achieved temporarily by direct pressure and then proceed with the rest of the resuscitation and assessment (cABCD).

5. Temporary Intravascular Shunt Insertion

Temporary intravascular shunt insertion includes insertion of a plastic shunt through the damaged ends of the artery to temporarily achieve blood flow. Initially temporary intravascular shunts were used for victims who were physiologically unstable and who needed transport for a long time to reach the medical facility where vascular repair can be done. At present the indications for shunt insertion after vascular trauma include;

- In unfit patient as a measure of damage control until patient can be resuscitated to become physiologically fit for definite vascular repair.
- When the patient needs a long transfer time for a definite vascular repair which will compromise the viability of the limb.
- Gustilo IIIC fractures needing complex orthopedic intervention before vascular repair, where the shunt is used until the orthopedic fixation is over [17].

The disadvantages of shunting include; Damage to the arterial ends by the shunt will compromise the chance of performing a direct repair later.

The procedure includes identification of damaged arterial ends and then the shunt with the largest diameter possible is inserted. The Shunt is inserted for about 1 to 2 centimeters into to the arterial ends and secured with ties. Post Shunt insertion heparinization may be considered if there are no extensive local, solid organ, cerebral or intra-abdominal injuries.

In our local setting temporary shunt tubes e.g. Carotid Artery Shunt, are not available in most of the peripheral hospitals. And the expertise to insert a temporary shunt is also not available. Therefore even though the transfer of the patient takes a long time, temporary intravascular shunting is not practiced.

Investigations

If the hard signs are present and the viability of the limb is threatened (presence of paresis, paralysis, paresthesia or anesthesia), the patient needs immediate exploration and repair of the vessels. But imaging may be needed even in these situations especially if the injuries are at multiple levels i.e. multiple fractures, trap gun injury with multiple penetrating

sites, etc.

Computed Tomographic Angiography (CTA) is the "gold standard" investigation for the vascular trauma. Because it can be rapidly done, it can scan the whole arterial system including the intra-abdominal, intra thoracic and the extremity vessels (Figure 5). It is non-invasive. And in addition it can show the associated injuries e.g. fractures. Also a 3D reconstruction would give a better idea about the site of the injury (**Figure 6**). The disadvantages of CTA include; it is not freely available, artifacts may disturb the visualization of the site of injury e.g. bone spicules, external fixators of the bone. Also poor timing of the imaging may interfere with the interpretation e.g. non visualization of the vessels. The administration of contrast agent may predispose to renal impairment in an already compromised patient.

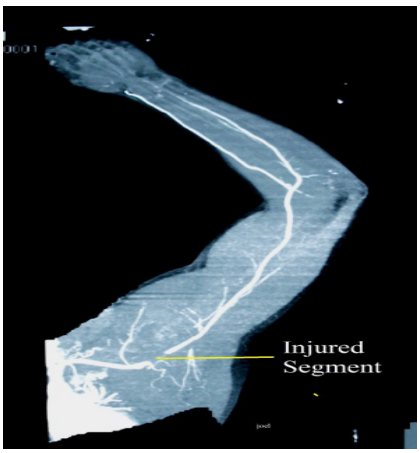

Figure 5: Computed Tomographic Angiography showing the injured segment

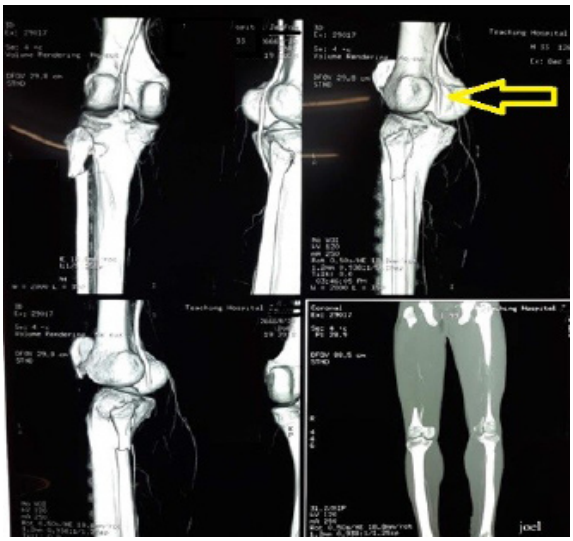

Figure 6: 3D reconstruction showing Popliteal arterial injury (Arrow)

Doppler ultrasonography (Duplex Scan) combined with color (Color Duplex) is a useful tool in case of extremity vascular trauma. Duplex is easily accessible, it visualizes the injured segments. But duplex scanning is operator dependent, and the limb with extremity vascular injury is covered with dressings which interfere with scanning and the patient may not be cooperative and the scanning probe induces pain. Also finding an expert ultra-sonographer in the nights when vascular injury commonly presents is difficult in our hospital. For these reasons in the center where the author works, Duplex Ultrasound Scanning is not routinely done on

an urgent basis in patients with extremity vascular injuries. But patients who develop false aneurysms and traumatic arteriovenous fistula often present late. In these patients ultrasound scanning is useful in confirming the diagnosis and follow up.

Conventional catheter angiography is done when the patient is on the operating table (On-Table Angiography). This is done when the limb viability is threatened but there are multiple level injury to the limb (if the patient has multiple level fractures and in trap gun injuries which produces multiple pellet injuries- (**Figure 7**). In this situation the CT Angiogram is not done because it is time consuming, but the site of vascular injury has to be visualized to plan the exploration and repair. The angiogram needle is directly introduced into the artery and contrast is injected. But performing an on-table angiogram needs a radiolucent operating table and a radiographer available during the time of the procedure. In addition it involves injection of contrast agent which is associated complications. And it is a relatively invasive procedure.

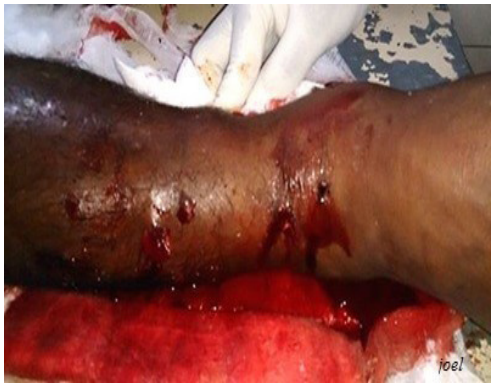

Figure 7: Multiple pellet injuries

6. Management of Vascular Injuries

The Management and the outcome of the vascular injuries have improved dramatically over time. Experience gained from various war injuries has contributed dramatically to the improvement of the management and the outcome of the vascular injuries. During World War 1 and 2 the vascular injuries were managed with ligation. This resulted in very high amputation rate. For example during world war 2 the amputation rates were 40% [18]. This situation improved during Vietnam and Korean wars (amputation rate of 15%), mainly due to rapid evacuation and repair [19]. During the Iraq and Afghanistan wars the amputation rate reduced to 8%. In addition to the advances in vascular repair methods, antibiotics, advances in trauma care have contributed to the improved outcome.

6.1 Principles of vascular repair in vascular trauma

Following vascular injury patients with ‘hard signs’ should undergo immediate surgical exploration and intervention. Patients who present with ‘soft signs’ of vascular injury and patients who present late with false aneurysms and traumatic arteriovenous fistulas, can undergo further imaging and can be managed later.

Patients following vascular injury may have other associated injuries. The patient maybe hemodynamically unstable. Therefore a detailed assessment according to the Advanced Trauma Life Support (ATLS) protocol should be done. All patients should be resuscitated before the patient is transferred to the operating room for the vascular intervention.

Following vascular injury and ischemia, vascular repair and reperfusion should be done as soon as possible. With long ischemic time the muscles become progressively nonviable and there is a high chance of having severe reperfusion effects. Miller and welch reported in 1947 in canine vascular occlusion / limb ischemia model, that the limb salvage rate was 90% if the ischemic time is less than 6 hours. And the Salvage rate reduced when it was more than 6 hours. Therefore they suggested a "Golden period" of 6 hours as a threshold for intervention. In practice in the center where the author works, the patients are transferred from long distance hospitals and often reach the vascular center well beyond 6 hours (5) (6). Traditionally in lower limbs if more than two of the four leg compartments are non-viable the limb was not revascularised, due to the risk of reperfusion effects. But at the center where the author works, we receive patients after long delays and about a third of them have two or more compartments non-viable. In this situation revascularization of such patients is attempted despite having longer than 6 hours of ischemia, provided the patients are consenting, systemically well, there were no severe soft tissue injuries and there was no sepsis [20]. Because outcome after a vascular trauma not only depends on ischemic time but on other factors such as associated soft tissue and skeletal injuries, contamination, systemic status of the patient, etc.

The vascular surgical repairs are generally done under general anesthesia. The whole limb is cleaned and prepared. The whole limb should be kept exposed (to assess distal circulation after the intervention and to perform fasciotomy before or after the vascular repair and to facilitate the skeletal stabilization). The contralateral groin and the thigh is also cleaned and prepared for the harvest of the saphenous vein graft (RSVG). In case of lower limb injury the contralateral groin and the thigh is used to obtain the reversed saphenous vein graft. Because in an already injured limb the deep veins may also be injured therefore compromising the superficial veins may impair the venous drainage further.

Longitudinal incisions are made using the known landmarks for major arteries. Longitudinal incisions will allow extension along the whole arterial and venous system if needed and in addition it will allow fixation of the bone. Access to the junctional vessels i.e. the subclavian and the iliac vessels may need additional incisions.

Exposure of the femoral artery is done by a longitudinal incision at the mid inguinal point or in line with the femoral pulse, starting from about 1-2 centimeters superior to the inguinal ligament and extending inferiorly along the superficial femoral artery (a line extending from the mid inguinal point to the adductor tubercle). This incision can be extended inferiorly about

a hand breadth posterior to the medial border of the patella to about a centimeter posterior to the medial border of the tibia to expose the popliteal vessels.

In case of upper limb arterial injury repair the arm is kept at 90 degrees on an arm board. The incision for brachial artery is made medially on the arm along the groove between the medial border of the biceps and the triceps. And this incision is extended along the brachial artery, and at the elbow crease a lazy "S" extension is made.

The subclavian artery arises from the arch of the aorta on the left side and from the brachio cephalic trunk on the right side. It runs behind the scalenus anterior on the first rib and enters the neck. The subclavian artery is divided into three parts based on its relationship to the scalenus anterior. The part of the subclavian artery medial to the muscle is the first part; the part which lies posterior to the scalenus anterior is the second part and the third part is lateral to the scalenus anterior. The exposure of the second and third part of the subclavian artery is through a supraclavicular incision. And the exposure of the first part needs a thoracotomy or sternotomy.

The iliac artery arises at the aortic bifurcation. Exposure to the iliac artery is either extra peritoneal (through a curved supra inguinal ‘hockey stick’ incision) or Trans peritoneal (through a midline incision).

During the surgical exploration of the injured arteries, the proximal pulse will act as a guide to the presence of proximal end of the injured vessel. The proximal and distal control should be achieved (especially the proximal) before exposing the injured segment to avoid dislodgement of the clot and inducing bleeding. After achieving proximal and distal control the damaged area is exposed and the damage to the blood vessels is assessed. Also at the time of surgical exploration the damage to the soft tissues, nerves are also assessed. Unhealthy soft tissues foreign bodies and contaminants are removed. After removal of the unhealthy tissues the patient may need a flap to cover the repaired blood vessel.

The main principles of vascular repair after vascular injury are that the healthy arterial walls should be anastomosed without tension in a non-contaminated on infected field. Therefore the lacerated arterial ends are trimmed until the healthy intima is seen (**Figure 8**). After trimming the proximal and distal arterial ends, Fogarty embolectomy / thrombectomy catheter is inserted into the distal and the proximal arterial ends to remove the thrombus. Thrombus may have formed during the time when the blood flow was absent or stagnant following vascular injury. Back flow from the distal end is noticed. And the proximal end should have pulsatile bleeding. Absence of normal pulsatile bleeding from the proximal end indicates that there may be further the injuries proximally. Therefore in case of multiple level injuries further exploration proximally or an on table angiogram may have to be performed. The distal arterial end is injected with heparin saline (10 IU /ml concentration). The distance

between the trimmed arterial ends are assessed. After trimming, the arterial ends retract and the distance between the ends increases. General principle is that if the arterial ends can be approximated without tension a direct end to end anastomosis repair can be done (less than 1 cm gap). If this is not possible after trimming an interposition graft repair is done (**Figure 9**). In authors experience the saphenous vein graft (after reversing - RSVG) is commonly used (6).

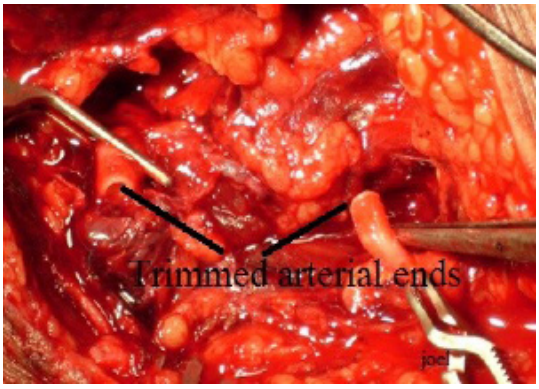

Figure 8: Trimmed brachial artery showing healthy intima

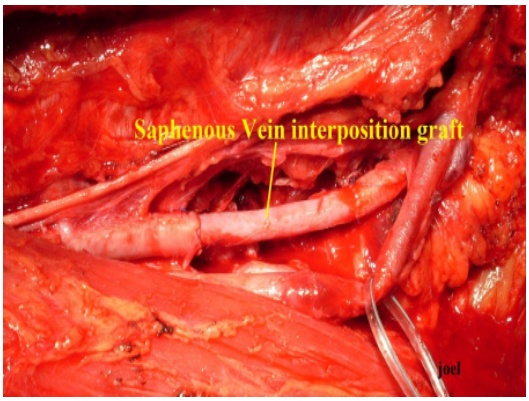

Figure 9: saphenous vein interposition graft repair

Other grafts which can be used include;

- Short saphenous vein (SSV)
- Arm veins – Cephalic / Basilic veins
- Superficial femoral vein (SFV) - if there is a mismatch in size of the saphenous vein graft in larger arteries e.g. subclavian, iliac arteries.
- Biological grafts - Bovine graft
- Synthetic grafts (PolyTetraFluoroEthylene (PTFE) – antibiotic impregnated)

In trauma vascular repair, synthetic grafts are not preferred. Because the operation field is already contaminated and may be infected which results in graft infection and poor outcome. And also long term patency of the grafts are poorer then the native grafts.

For large arteries (e.g. subclavian, axillary, brachial, iliac, common and superficial femoral), anastomosis is done with 6/0 or 5/0 polypropylene sutures. Two anchoring sutures are

applied in diagonally opposite directions (180 degrees) and the far stay suture is tied (figure). The near suture is kept untied to facilitate the suturing. Stay sutures are stabilized with rubber shods (**Figure 10**). The anastomosis is done with continuous suturing. When a reversed vein graft is used, the distal end is anastomosed first to get the larger diameter of the vein graft. Before completing the anastomosis the vessel ends are flushed with heparin saline and some blood is allowed to bleed out to make sure the clots and the debris are flushed out. When tying the knot tightening is avoided to prevent narrowing at the anastomotic site. For radial, ulnar and tibial arteries, a 7/0 polypropylene suture is used. And interrupted suturing technique is done.

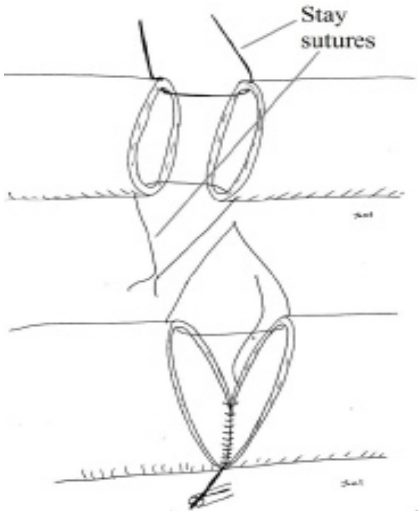

Figure 10: Repair technique

After completion of the anastomosis the distal pulses are assessed. And the compartments of the leg and the forearm are re assessed to check for the evidence of reperfusion injury. Sometimes the distal pulse may not appear especially in the foot. In this instant saturation, the change in quality of the doppler flow can be used to assess the circulation. A completion angiogram (on table) can also be performed. Again performing an on table angiogram has practical difficulties.

When there is a side wall laceration, the lacerated edges are trimmed and then the defect is repaired with a venous patch (**Figure 11**).

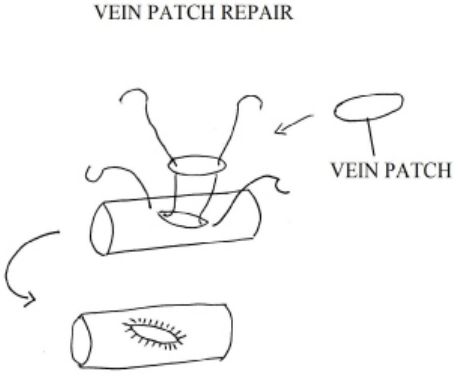

Figure 11: Repair with a venous patch

6.2 Injuries to the peripheral veins

Injuries to the peripheral veins usually do not result in limb loss or death. But it may result in hemorrhage or thrombosis. Thrombosis may result in increased compartmental pressure in the already injured limb.

Generally major lower Limb veins (iliac, femoral and popliteal) are repaired. And major upper Limb veins (subclavian and axillary) are also repaired. Small injured veins can be ligated. Repair methods of veins include;

- Direct suturing of the lateral wall - lateral phleborrhaphy
- Venous patch repair
- Interposition grafts
- Direct repair
- And if synthetic grafts are used reinforced grafts should be used to prevent collapse and thrombosis.

7. Reperfusion Effects

With the onset of ischemia, changes start to occur at cellular level (Reversible and Irreversible). With prolonged ischemia irreversible changes progresses and finally results in cell death. But before cell death some changes which occur at the cellular level produces unwanted side effects when an ischemic limb is reperfused. These are called reperfusion effects.

There are two types of reperfusion effects

- Local
- Systemic.

Local effects are called reperfusion injury i.e. paradoxical death of already dying muscles after reperfusion of the limb. The systemic effects which occur as a result of release of substances from dying and dead muscle cells is called reperfusion syndrome.

7.1 Pathophysiology

During ischemia there is a reduction in the production of adenosine triphosphate (ATP) in the cells. This result in;

- Increase in anaerobic metabolism

- Dysfunction of the sodium potassium ATPase pumps (Na+ - K+ ATPase) and calcium ATPase pumps (Ca2+ ATPase)

- Detachment of the ribosomes.

Dysfunction of the Na+ - K+ ATPase pumps on the cell membrane results in accumulation of intracellular sodium. And dysfunction of calcium ATPase pump results in impaired reuptake of calcium into the endoplasmic reticulum and increased cytosolic calcium ion concentration. And accumulation of sodium in the cells results in dysfunction of the sodium hydrogen exchanger (Na+ - H+) pumps. Accumulation of sodium and calcium within the cell results in hyperosmolarity, which in turn results in accumulation of water within the cell resulting in intracellular edema. Accumulation of intracellular calcium results in activation of enzymes e.g. Phospholipases and Cal pains. Activation of these enzymes results in degradation of membrane phospholipids and cytoskeletal proteins. In addition increase in the mitochondrial calcium level leads to influx of water into the mitochondria resulting in mitochondrial swelling and uncoupling of oxidative phosphorylation process. Increased anaerobic metabolism and dysfunction of the sodium hydrogen exchanger pump result in intracellular acidosis leading to enzyme dysfunction. If the ischemia continues progression of these changes leads to cell death. In addition neutrophil migration and accumulation within the ischemic tissues are also increased [21]. Also during ischemia there is an increased endothelial permeability due to cytoskeletal changes and mediators secreted by the neutrophils [22].

Endothelial ischemia results in in expression of leukocyte adhesion molecules on the surface of the endothelial cells. And also the complements are activated (C3, C5a) which further enhances the inflammatory response after reperfusion.

And during ischemia, there are other changes which occur at the intracellular level which results in production of oxygen free radicals and hydrogen peroxide (O2•/ H2O2) when oxygen becomes available during reperfusion. This results in further damage to the cells. These changes are in;

- Xanthine Oxido-Reductase Enzyme System

- NADPH Oxidase (Nox) System

- Nitric Oxide Synthase (NOS) System

Xanthine Oxido-Reductase Enzyme System

In cells the ATP is metabolized to hypoxanthine and then to xanthine and finally to uric acid by xanthine oxido reductase enzyme system (xanthine oxidase / xanthine dehydrogenase). Xanthine dehydrogenase uses Nicotinamide Adenine Dinucleotide (NAD) as electron acceptor

and this is reduced to NADH. Xanthine oxidase uses oxygen as electron acceptor, during this reaction oxygen free radicals (O_2•) are formed. During ischemia the level of xanthine dehydrogenase reduces and the level of xanthine oxidase increases. Therefore after reperfusion when the oxygen becomes available xanthine oxidase uses oxygen as an electron acceptor and this result in formation of oxygen free radicals and hydrogen peroxide. Thus resulting in increased oxidative stress to the cell and cell damage.

Nicotinamide Adenine Dinucleotide Phosphate (NADPH Oxidase) (Nox) system

NADPH oxidase (Nox) system is a family 7 protein enzymes [23] which are involved in reactive oxygen species production during ischemia reperfusion injury. It is found in many cells but mainly in phagocytes e.g. neutrophils. Ischemia activates the NADPH oxidase through several factors e.g. hypoxia inhibitory factor-1α, phospholipase A2, etc. This in turn results in production of oxygen free radicals when oxygen becomes available during reperfusion.

Oxygen free radicals cause cellular damage by;

- Damaging the membranes by lipid peroxidation
- Increasing the leukocyte adhesion molecules on the surface of the endothelium
- Promotion of inflammatory mediator secretion by endothelium [24].

Nitric Oxide Synthase (NOS) System

In normal state endothelial nitric oxide synthase (NOS) produces nitric oxide which is a vasodilator. In reperfusion state when there is oxidative stress by oxygen free radicals, nitric oxide synthase components are oxidized resulting in uncoupling of nitric oxide synthase. This mechanism leads to more formation of oxygen free radicals.

7.2 Clinical features of reperfusion injury / and reperfusion syndrome

As mentioned above the post perfusion / reperfusion effects are;

- local - called reperfusion injury and
- Systemic - called post perfusion syndrome.

The local effects include;

- Further death of already dying muscles (reperfusion injury) - Due to accumulation of reactive oxygen species resulting in oxidative stress of the reperfused cells. Oxygen free radicals causes membrane peroxidation, cytoskeletal destruction, and stimulation of the inflammatory cell migration into the reperfused tissues. This results in further the damage to the cells leading

to the death of more cells.

• Swelling of the muscle compartments - During ischemia there is endothelial cell cytoskeletal restructuring and destruction, which results in increase gap between the endothelial cells resulting in increased permeability. In addition migration of neutrophils into the tissues also increased during the ischemic period. This is further aggravated following reperfusion due to oxidative stress. Accumulation of fluid and inflammatory cells within the compartments results in swelling and compartment syndrome.

The systemic effects of reperfusion (post perfusion / reperfusion syndrome) includes systemic abnormalities, multi organ dysfunction syndrome and death of the patient. It occurs as a result release of mediators in to the circulation after reperfusion. The main organs affected are the lungs. Resulting in Acute Respiratory Distress Syndrome (ARDS). In addition there is myocardial dysfunction resulting in hypotension and coagulation abnormalities (Disseminated Intravascular Coagulation (DIC). Other effects are renal impairment and hepatic dysfunction. In addition hyperkalemia, acidosis, myoglobinuria are some other features [25].

7.3 Prevention and management of reperfusion effects

As mentioned above the undesirable effects of reperfusion are more when ischemic time increases. Therefore reperfusion as soon as possible is the mainstay of treatment in minimizing the reperfusion effects. The treatment strategies are aimed at counteracting the cellular changes, which occur during the ischemic period as well as after reperfusion.

The multi organ failure is aggravated if there are associated conditions like severe injury, sepsis, hemorrhagic shock, etc. The following interventions are carried out to manage reperfusion syndrome;

a) Hemodynamic stabilization

Hypotension worsens multi organ failure. Maintaining hemodynamic stability with oxygenation is important in preventing further worsening of the organ failure as well as in promoting the recovery. Adequate blood pressure should also be maintained to keep the repaired artery patent.

b) Hydration

Patients after trauma are often dehydrated. Multi organ dysfunction syndrome which results after reperfusion is worsened by dehydration. Therefore adequate hydration is essential to maintain the cardiovascular and the renal systems. Monitoring of the hydration status is essential. Patients should maintain high urine output.

c) Antibiotics

Patients with limb vascular injury often have associated musculoskeletal injuries which are contaminated. And in addition sepsis worsens the effects of reperfusion. Patients are administered with broad spectrum antibiotics.

d) Antioxidant Therapy

Reactive oxygen species plays a major role in ischemic re perfusion injury. Therefore various antioxidants and free radical scavengers have been tried in experimental models. This includes mannitol, allopurinol (xanthine oxidase inhibitor), superoxide dismutase and apocynin (interferes with the action of NADPH oxidase). Beneficial effects of these agents are mainly in clinical trials [26]. The author uses mannitol for the first 48 hours after reperfusion. Mannitol is a free radical scavenger. It has been shown to reduce the acute respiratory distress syndrome after re perfusion [27].

e) Anti-complement agents

Compliments play a major role in post perfusion inflammation. Anti-complement agents like C3 convertase inhibitors and antibodies to various components of the complements have been used in animal models and shown benefits [28]. Widespread uses of these agents are not practiced in clinical settings.

8. Compartment syndrome

Compartment syndrome is increased intra compartmental pressure resulting in reduced tissue perfusion within the compartment. In lower limbs there are osseo-fascial compartments. The deep fascia of the lower limb is unyielding, therefore if there is an increase of the contents within the compartment the intra compartmental pressure increases sharply. When the compartmental pressure is more than the capillary pressure there will be ischemia at the tissue level. This results in reduced tissue perfusion resulting in compartment syndrome. This was first described by Volkmann in 1881 [29].

The causes for compartment syndrome in cases of trauma and vascular injury include;

- Swelling of the muscles
- Muscle ischemia
- Fractures and Hematoma formation
- Reperfusion injury
- Inflammation of the muscles

- Infections
- Capillary leak as a result of Systemic Inflammatory Response Syndrome (SIRS) and excessive fluid resuscitation
- Tight bandages and Casts

The rise in intra compartmental pressure leads to ischemia at the capillary level which further induces swelling of the tissues, which in turn results in the increase in the intra compartmental pressure. This vicious cycle continues and finally resulting in death of the contents of the compartment e.g. muscles and nerves.

The clinical features of compartment syndrome include;

- Excessive pain not explained by the degree of trauma or the fracture
- Swollen, tense, tender compartments
- Pain on passive movements
- Numbness in the area of the nerve distribution running through the compartment. For example in case of leg compartment syndrome the anterior compartment is often the first compartment to be involved. And numbness occurs in the first interdigital cleft as a result of deep peroneal nerve involvement.
- Disappearance of pulse and other signs of distal ischemia as a result of compression of the arteries running through the compartment, e.g. Dorsalis paedis and posterior tibial pulses. This is a late sign. Therefore presence of distal pulse should not be taken as an exclusion criterion for the compartment syndrome in the leg.
- Weakness of muscles

Diagnosis is mainly clinical. But in patients who have other associated factors which interferes with diagnosis of the compartment syndrome (e.g. Unconscious patients, patients with associated nerve injury) intra compartmental pressure measurement can be used as an adjunct to diagnosis. In addition intra compartmental pressure measurement is useful in monitoring the above patients.

8.1 Intracompartmental pressure measurement

There are several techniques of measuring intra compartmental pressure. All of these techniques involve injection of a small amount of fluid into the compartment and transducing the intra compartmental pressure on to a manometer. The classic technique was described by whitesides, et al. in 1975 [30] .The other devices available are portable Stryker system and

slit or wick catheter system. Generally diastolic blood pressure - intra compartmental pressure difference (Delta pressure) of 30 mm/ Hg is taken as a threshold for diagnosing compartment syndrome and the indication for fasciotomy [31].

Recognition and immediate removal of the cause and fasciotomy are the main modes of treatment of the compartment syndrome.

8.2 Fasciotomy

Fasciotomy is done by releasing the fascial walls of the compartments.

Fasciotomy is performed for the following purposes;

- Therapeutic

This is done when the patient already has an established compartment syndrome and the fasciotomy is done as a treatment.

- Prophylactic

This is done on a patient who has high chance of developing compartment syndrome. Therefore the fasciotomy is done prophylactically to prevent the compartment syndrome and its consequences e.g. before transferring a vascular injury patient over a long distance a prophylactic fasciotomy is done.

- Diagnostic

This is done when there is a doubt about the viability of the compartments. Fasciotomy is done to perform a direct assessment of the viability of the muscles.

8.3 Fasciotomy of the leg

Leg is a common site of vascular trauma and compartment syndrome. It has four compartments namely (**Figure 12**);

1. Anterior compartment

2. Lateral compartment

3. Deep posterior compartment

4. Superficial posterior compartment

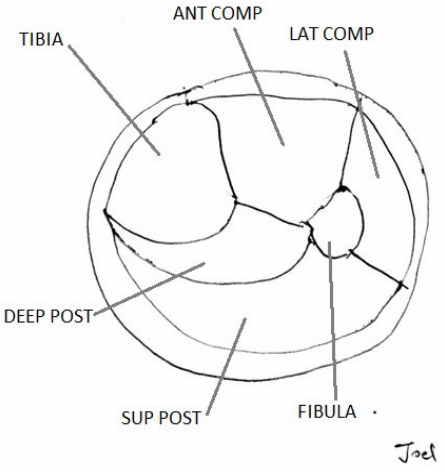

Figure 12: Compartments of the leg; Ant comp - Anterior compartment, Lat comp - Lateral compartment, Sup post - Superficial posterior compartment, Deep post - Deep posterior compartment

Anterior and the deep posterior compartments are the first compartments to be involved by the compartment syndrome.

Types of leg fasciotomy

There are two methods of fasciotomy in the leg i.e.

- Single incision fasciotomy
- Double incision fasciotomy

The author prefers single-incision fasciotomy done through an incision on the lateral aspect of the leg. The double incision fasciotomy is done through two incisions on medial and lateral aspects of the leg. The disadvantages of double incision fasciotomy in case of trauma include;

- Additional length of wounds for the patient
- The popliteal artery is often accessed from the medial side therefore if there is medial incision on the leg it may cause problems with closure of the wound

The main criticism regarding the single incision fasciotomy is the concern about the inadequate release of all compartments. Experience and sound knowledge of anatomy of the leg compartments are needed to overcome this. In any case if there is any concern about the inadequate release of all compartments, the single incision fasciotomy should be converted to a double incision fasciotomy.

Sometimes excision of the fibula is done with single incision fasciotomy. This increases the trauma. The author does not perform excision of the fibula even though we usually perform single incision fasciotomy.

8.3.1 Single incision fasciotomy

We prefer to perform single incision fasciotomy at our center. The line of incision is along the fibula. The full length of the incision extends from two inches below the head of the fibula to two inches above the lateral malleolus. But often it is not necessary to make the full length of the incision. The adequacy of the incision is assessed during the fasciotomy and the lengthening is done to completely reduce the pressure within the compartment.

The incision is deepened up to the deep fascia (**Figure 13**). This will be over the lateral compartment. A small opening is made on the deep fascia and a blade of a half open dissecting scissor is inserted and the fascia is split open longitudinally to the length of the incision.

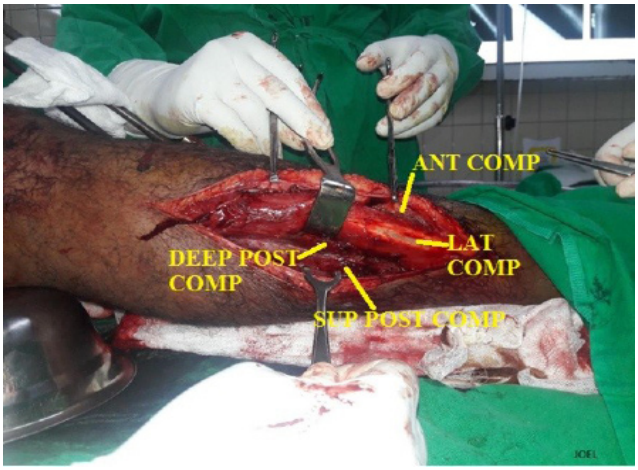

Figure 13: Single incision fasciotomy

Two tissue holding forceps are applied to the anterior fascial flap and it is lifted by the assistant. The dissection is carried out between the deep fascia and the muscles to create a fascio cutaneous flap. Dissection is carried out until the anterior inter muscular septum, separating the anterior and the lateral compartment is encountered. The superficial peroneal nerve runs anteriorly two pierce the deep fascia to enter the subcutaneous tissue at about the middle and lower thirds of the leg. Damage to this should be avoided. The anterior intermuscular septum is split open longitudinally. Now the tissue holding forceps are shifted to the posterior facial flap and it is lifted. The dissection is carried out between the muscles of the lateral compartment and the posterior intermuscular septum. The posterior intermuscular septum is split open to enter the superficial posterior compartment. The dissection between the muscle of the lateral compartment and the posterior intermuscular septum is continued until the fibula is encountered and dissection is continued further posterior to the fibula. This will expose the thin fascia covering the deep posterior compartment which lies anteriorly on the dissection plane. The fascia covering the deep posterior compartment is also split open. The viability of all compartments is assessed and documented.

8.3.2 Double incision fasciotomy

In double incision fasciotomy the medial incision is along a line running one centimeter

posterior and parallel to the medial border of the tibia.

The lateral incision is parallel to and a centimeter anterior to the fibula. The full length of the incision extends from about two inches below the tibial tuberosity to two inches above the malleoli. The length of the incision is determined intraoperatively, depending on the amount of swelling and the adequacy of the release of the tension.

On the lateral side the incision is deepened up to the deep fascia. Then the anterior intermuscular septum is identified. This will lie in line with the incision. Then a horizontal incision is made crossing the anterior intermuscular septum. This will confirm the position of the intermuscular septum. Then the dissection is carried out between the skin and the deep fascia for about a centimeter on either side of the anterior intermuscular septum. Then incisions are made on the deep fascia covering the anterior and the lateral compartments, parallel to and on either side of the anterior intermuscular septum, about a centimeter away from it. The length of the fascial incision is equal to the length of the skin incision. Initially a small opening is made on the fascia with the tip of the number 11 blade, and then it is extended superiorly and inferiorly by splitting the fascia with the blade of a half open dissection scissor. Towards a lower end care should be taken to avoid injury to the deep peroneal nerve emerging through the fascia.

On the medial side the fascia is opened longitudinally along the medial border of the tibia. This will expose the attachment of the soleus to the medial border of the tibia. This has to be separated to visualize the deep posterior compartment. After visualizing the deep posterior compartment the fascia over deep posterior compartment is incised longitudinally.

After entering each compartment the viability of the muscles is assessed. This is done by checking the color, consistency, capillary bleeding and contraction. Viable muscles are soft to firm in consistency with bleeding capillaries on the surface of the muscles and contracts to the electrical stimulation with a diathermy.

8.3.3 Forearm Fasciotomy

After the leg fasciotomy, forearm fasciotomy is a commonly done fasciotomy in our center. Unrecognized forearm compartment syndrome results in ischemic contractures later (Volkmann ischemic contracture).

There are two main compartments in the forearm i.e. Flexor (Volar) compartment and extensor (Dorsal) compartment. Flexor compartment is often involved with compartment syndrome. And releasing the flexor compartment alone may be adequate in most situations.

8.3.4 Flexor compartment fasciotomy

The incision is made starting from about an inch below the medial end of flexor crease of the elbow. The incision runs in a straight line towards the radial aspect of the wrist crease. The forearm fasciotomy is often combined with carpal tunnel release. For this the forearm incision is curved towards the ulna side horizontally along the wrist crease and continued into the palm along the ulna side of thenar crease for about 3 to 4 centimeters. The forearm incision is deepened and the fascia is incised in line with skin incision. After opening into the compartment, the epimysium surrounding individual muscles may have to be released. After completing the forearm fasciotomy carpal tunnel release is done.

The tension in the extensor compartment is reassessed after performing the flexor compartment fasciotomy. Often releasing only the flexor compartment is enough to reduce the pressure in the dorsal compartment as well. If the dorsal compartment is tense, then an incision is made starting about an inch below the lateral epicondyle running towards the middle of the dorsal wrist crease ending about an inch proximal to the crease. The fascia is opened and individual muscle epimysium may have to be released.

8.3.5 Thigh fasciotomy

Thigh compartment syndrome often occurs after closed fractures. There are three compartments in the thigh i.e.

- Anterior compartment
- Posterior compartment
- Medial or adductor compartment

The incision is made along the lateral aspect of the thigh, starting from 2 to 3 inches below the greater trochanter and extending towards lateral femoral epicondyle. The incision is deepened and the fascia lata is incised in line with incision. This will release the anterior compartment. Then the vastus lateralis is retracted anteriorly. This will expose the lateral intermuscular septum. The lateral intermuscular septum is incised longitudinally to release the posterior compartment. Releasing the anterior and the posterior compartments is often adequate unless there is a special need to release adductor compartment.

8.4 Closure of fasciotomy wounds

Soon after the fasciotomy closure is often not possible due to the presence of swelling, contamination and dead tissues. Therefore the wound is always left open and sequential inspection of the wound is done every 48 hours. When there are no swelling, infection or dead

tissues the skin can be approximated. Often the wound is closed with a skin graft (**Figure 14**).

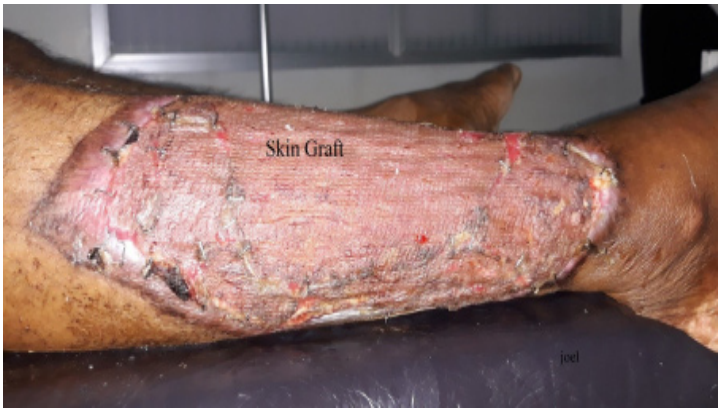

Figure 14: Delayed closure of fasciotomy wound with skin graft

9. Systemic Heparinization Following Traumatic Arterial Injuries

There is always controversy about systemic heparinization following traumatic arterial injuries.

The intended benefits of systemic heparinization are;

- Prevention of clotting of the stagnant vessels following vascular injury
- Prevention of thrombosis at the site of arterial repair
- Reduction of clotting tendency arising as a result of ischemia reperfusion injury of the endothelium.

Heparin is shown to reduce the skeletal muscle infarct size after ischemia reperfusion injury [32].

But the disadvantages of heparinization following trauma includes;

- The risk of bleeding into the injured tissue and Hematoma formation
- Bleeding into other internal organs which may have associated trauma e .g. splenic injury, intracranial injuries, etc.

Some studies report an increase in bleeding complications, hematoma formations and prolonged intensive care unit stay following administration of systemic heparin in patients with trauma [33]. Another study reported reduced amputation rate following administration of heparin in patients with popliteal arterial trauma [34]. But even in this study there were many other factors e.g. the mangled extremity score, associated with the amputation rates. Therefore I do not recommend systemic administration of heparin.

10. Complications of Vascular Injury

The main complication following the vascular injury of the extremities is the risk of amputation. In addition to that if improperly managed extremity vascular injury can lead to the death of the patient.

In addition traumatic pseudo aneurysm formation and traumatic arteriovenous fistula can present late. Traumatic pseudo aneurysm formation occurs when there is a side wall injury. This results in bleeding in to the surrounding tissues forming a hematoma. The center of the hematoma will have the flowing blood. Gradually the surrounding hematoma with the surrounding tissues forms a fibrous cap of the false aneurysm sac. Patients present with history of trauma or an intervention on the artery. And following that intervention or trauma there is hematoma formation which does not settle and keeps on increasing in size. The patient may have increasing pain or discomfort. The skin over the false aneurysm is discolored and shiny. And it is pulsatile on examination and could be warm if there is associated infection. In the center where the author works traumatic false aneurysms are commonly due to iatrogenic injury to the common femoral artery. This happens following common femoral arterial access for cardiac catheterization. Small false aneurysms arising from the common femoral artery (less than 2 centimeters), which are reducing in size, not infected and asymptomatic can be managed conservatively. Small traumatic false aneurysms spontaneously close.

Treatment options for symptomatic and complicated false aneurysms are;

- Open closure of the neck of the aneurysm
- Patch repair of the neck of the aneurysm
- Covered stenting
- Thrombin injection in to the sac.
- If the traumatic false aneurysm arises from an artery which is not an end artery or from an artery which has another companion artery e.g. radial or ulnar, the artery can be tied or embolised.

Traumatic Arterio Venous Fistula (TAVF) occurs when there is simultaneous injury to the artery and vein (**Figure 15**). This results in arterial blood flow getting into the vein. Patients with TAVF present with pulsatile mass at the site of trauma. Prominent pulsatile vessels are seen at the site with thrill and bruit over it. High volume, low resistance flow through the TAVF can result in inadequate distal blood supply to the limb resulting in distal ischemia (steal syndrome). Due to high volume return of blood to the heart there is high cardiac output resulting in hyper dynamic circulation which over long term results in heart failure.

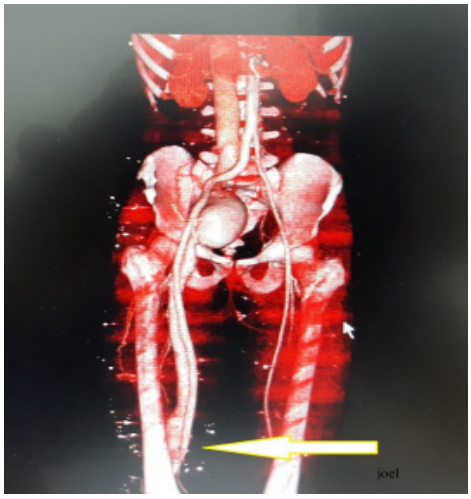

Figure 15: Traumatic AVF between Popliteal artery and vein (Arrow)

Management of traumatic arteriovenous fistula includes;

- Open interventions

  Ligation

  Excision and interposition graft repair

- Endovascular interventions

  Covered Stenting

  Embolization

Other complications of extremity vascular injuries include ischemic contracture. This commonly occurs in the forearm following prolonged ischemia and muscle death resulting in muscle fibrosis (Volkmann ischemic contracture). This was first described in 1881 by Volkmann [29].

And also complications occur due to reperfusion (Refer above).

11. Factors Affecting the Outcome of Extremity Vascular Injuries

After traumatic vascular injury in addition to the ischemic time there are other factors which influence the outcome (i.e. limb salvage / amputation, complications, death, etc.). Some of these factors include; age, presence of circulatory shock, bone injury, soft tissue injury, nerve injury, deep vein injury, amount of contamination, comorbid conditions, etc.

11.1 Mangled limbs and Mangled Extremity Severity Score (MESS)

A limb with severe soft tissue, skeletal and vascular injury with contamination is a "Mangled limb" (Figure 16). These types of limbs cannot be revascularised or reconstructed. There are various scoring systems developed over time to objectively predict the risk of

amputation after severe lower limb injuries with or without vascular injuries e.g. Mangled Extremity Severity Score (MESS), Predictive Salvage Index , Limb Salvage Index , etc.

Mangled Extremity Severity Score incorporates skeletal / soft tissue injury, limb ischemia, shock and age (**Table 1**).

Table 1: Mangled Extremity Severity Score

Factors		Points
Skeletal/ Soft Tissue Injury	Low energy - simple fractures, stabs	1
	Medium energy - open fractures, dislocation	2
	High energy - gunshot wounds, crush injuries	3
	Very high energy - injury with gross contamination and /or severe soft of tissue injury	4
Limb Ischemia (Points doubled if ischemia is more than 6 hours)	Reduced or absent pulse, no immediate limb threat	1
	Absent pulse, threatened viability	2
	Paralyzed / anesthetic limb	3
Shock	Systolic pressure always maintained above 90mm/Hg	0
	Transient hypotension	1
	Persistent shock	2
Age	Less than 30 years	0
	30 to 50 years	1
	More than 50 years	2

Mangled Extremity Severity Score of 7 or more predicts high chance of undergoing amputation. In other words patients who have multiple fractures with severe soft tissue injury, with highly contaminated wound, and clinically non-viable limb and who is old and systemically not well have a high chance of undergoing amputation. Therefore futile attempt at revascularization and reconstruction should not be considered endangering life in these patients.

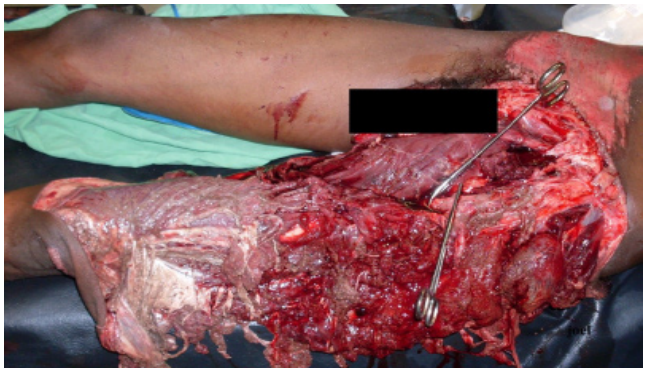

Figure 16: Mangled limb

12. Vascular Injuries to Popliteal Artery Trifurcation and Tibial Arteries

Popliteal artery begins at the adductor hiatus and ends by dividing into anterior tibial artery and tibio-peroneal trunk at the lower border of popliteus muscle. Popliteal artery is the commonest injured artery of the lower limb (6). The reason for this is because it is closely associated with the posterior aspect of the knee joint and therefore easily damaged during the injuries around the knee joint.

Injuries to the popliteal artery trifurcation / division are treated like other major lower limb arterial injuries. Repair methods include interposition graft between distal popliteal artery and the tibial arteries (generally posterior tibial artery) or direct anastomosis of the distal popliteal artery to the tibial arteries.

Injuries distal to this of a single tibial artery generally do not present with signs of distal ischemia. And single tibial arterial injury need not be repaired because the collateral circulation from the tibial and peroneal arteries is enough to maintain the distal circulation. If there is bleeding from a single tibial artery, it can be ligated.

However if the patient with leg injuries presents with signs of distal ischemia they should be assessed and revascularised if the situation allows. Often these patients with leg injury and distal ischemia have severe associated soft tissue and skeletal injuries i.e. mangled limb. Therefore the repair of the vessels in this situation becomes impossible. And the outcome of these limb injuries is often poor.

13. Use of Tourniquets in Extremity Vascular Injuries

Following injuries to the extremity vessels one of the important cause of death is bleeding. Tourniquet if correctly applied at the right time has been shown to reduce the mortality especially in battlefield trauma [35]. Also application of tourniquets is shown to reduce the need for blood transfusions [36].

But it has been shown that patients who had tourniquets had higher amputation rates, need for fasciotomy and acute kidney injury [37]. But this could be due to the fact that the limb needing a tourniquet is already severely injured i.e. mangled; therefore the patient would have anyway had the above mentioned complications attributed to the tourniquet. Therefore early decision in the field to apply tourniquet to patients who needs it will prevent the bleeding and death.

The tourniquet is used in the pre hospital care. Once the patient is in the hospital, if there is uncontrollable bleeding from the trauma site, direct pressure should be applied followed by immediate surgical exploration and tying or repair of the vessels should be done.

14. Conclusion

Extremity vascular injury involves young and fit individuals. They are the breadwinners of the family and patients with extremity vascular injuries often have associated injuries. All these patients should be assessed according to the Advanced Trauma Life Support protocol and resuscitated. All the clinical findings including the pulse status, the injuries and the viability of the limb should be documented. Outcome of extremity vascular injuries not only depends on the duration of ischemia but on many other factors as well. Therefore ischemic time alone should not be taken as the only factor when deciding on revascularization or amputation.

Outcome following extremity vascular injuries has improved over time including in our local setting. But prevention of injuries, establishment of dedicated trauma centers, improved transport of patients, training of staff in handling trauma including vascular trauma and improvement in post-operative care will improve the outcomes further in the future.

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