SURGICAL MANAGEMENT OF COMPLEX VASCULAR EXTREMITY TRAUMA

Thesis

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بسم الله الرحمن الرحيم

(وما أوتيتم من العلم إلا قليلا)

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- **API:** Arterial Pressure Index
- **ATLS:** *Advanced trauma life support.*
- **CT:** Computed Tomography.
- **CTA:** Computed Tomography Angiography.
- **CK:** Creatine kinase.
- **DCO:** Damage control orthopedics**.**
- **DIC :** Disseminated Intravascular Coagulation.
- **DVT:** Deep venous thrombosis**.**
- **Factor VIIa:** Factor VII activated.
- **GSV:** great saphemous vein.
- **HFS:** Hanovour fracture scale.
- **LD:** Lattismus Dorsi.
- **LEAP:** Lower extremity assessment project.
- **LSI:** Limb salvage index.
- **MCA**: Motor car accident.
- **MESI**: Mangled Extremity Syndrome Index.
- **MESS:** Mangle Extremity Severity Score
- **NIRS:** Near Infra Red Spectroscopy**.**
- **NISSSA:** Nerve, Ischemia, Soft tissue, Skeletal, Shock, Age.
- **NS**: Normal saline**.**
- **PHTLS:** Prehospital trauma life support.
- **PSI**: Predictive Salvage Index.
- **PTFE**: PolyTetraFloroEthylene.
- **P VALUE:** probability value.
- **SD: Standard Deviation.**
- **SFA**: superficial femoral artery.
- **SFV:** superficial femoral vein.
- **SPSS:** Statistical package for the social science
- **X2: CHI square.**

Abstract

Aggressive resuscitation is of almost importance. Thorough clinical assessment of limb viability before reconstruction, as some patients will need a primary amputation because of non-viability or marked haemodynamic instability or inability to restore function. The use of interposition grafts rather than direct arterial repair or venous patches. Meticulus debridment, mandatory solid bone fixation, and proper soft tissue coverage. Routine anticoagulation in venous injuries. Early fasciotomy in compartmental syndrome suspicion, or pre-emptive in critical limbs. Extra-anatomical bypass, temporary shunts, or ligation after stump pressure measurement may salvage limbs in critical situations. The combination of delay and shock reflects grave prognosis. Respect the concept of damage control (life before limb).

Keywords:
Complex Vascular
Extremity Trauma

INTRODUCTION

Due to the combination of soft tissue, osseous, vascular, and nerve involvement, complex extremity trauma requires prompt and precise evaluation and management to attain optimal outcome. Patients sustaining these unique injuries are at high risk for ischemia, wound infection, delayed union or non-union and chronic pain, not only owing to the anatomy of their injuries but also the prevalence of associated multisystem trauma and systemic problems related to the mechanism of injury. While the treatment goal remains extremity salvage, these injuries carry a high potential for morbidity and amputation (**Robert et al, 2009**).

Complex lower limb vascular injuries in high-energy penetrating or blunt trauma are associated with an unacceptably high incidence of complications including amputation. Traumatic ischaemia and ischaemia-reperfusion injury of skeletal muscle often lead to limb loss, the systemic inflammatory response syndrome that affects remote organs and even the potentially fatal multiple organ dysfunction syndrome .

The literature shows that vascular injuries are present in 10–48% of cases of complex limb trauma and amputation rates in this group as high as 85% have been reported (**Barros D'Sa et al, 2006)**

A truly mangled extremity is one in which amputation is a potential outcome and one in which complex reconstructive efforts need to be incorporated for limb salvage **(Robert et al, 2009).**

Aim of work

1. To review the type of trauma and patients presenting with complex vascular extremity trauma.
2. To highlight points of recommendations in diagnosis and treatment.
3. To discuss some points of contraversaries

Review of literature

HISTORIC PREVIEW

Extremity vascular injuries have been documented during episodes of armed conflict as far back as the Greek and Roman civilizations and undoubtedly occurred before those eras. Extremity amputations were the most common procedure performed by military surgeons in the US Civil War and World War II. **(DeBakey et al, 1946)**

DeBakey and Simeone calculated the amputation rate from vascular injuries in World War II as greater than 40%. **(DeBakey et al, 1946)**
The amputation rate from vascular injury in the Korean War and the Vietnam War dropped to approximately 15%. (**Rich et al 1970)**

FREQUENCY:

Sherif et al ,**1992** reported 224 extremity vascular injuries

in18months during the Afghanistan War, roughly 150 per year. **Fasol et al, 1989** reported 94 patients in 3 months (ie, approximately 376/y) on the Thailand-Cambodia border. In both studies, antipersonnel mines caused the majority of civilian extremity vascular injuries.

At a university teaching hospital in Australia, **Tobin et al, 1988**; reported 10 cases per year of extremity vascular injuries in Tbilisi**. Razmadze, 1999** reported 10.5 cases per year; in Sweden**. Kjellstrom and Risburg, 1980** reported 8.2 cases per year.

In Oxford, United Kingdom, **Magee et al, 1996** reported 4.7 cases per year. Penetrating injuries, both violent and nonviolent, predominated as the causes of vascular injuries in these reviews**. Humphrey et al, 1994** reported 12.4 extremity vascular injuries per year at a rural trauma center in Missouri.

Feliciano et al ,1988 reported approximately 55 lower extremity vascular injuries per year at Ben Taub General Hospital (a high-volume

urban trauma center) in Houston. In both extremes, the predominant cause of injury, especially in isolated vascular injury, was due to penetrating trauma. .

World War I	72.5%
World War II	35.8%
Korean conflict	13.0%
Vietnam conflict	12.7%
Vietnam[4] (% of major limb injuries)	8.3%
OEF/OIF[4] (% of major limb injuries)	7.4%

OEF, Operation Enduring Freedom; OIF, Operation Iraqi Freedom.
Note that OEF/OIF rates may change after complete analysis is performed after war's end.
Adapted with permission of Edward Arnold (Publishers) Ltd. from Barros D'Sa et al.[10]

Table (1) showing war time amputation rates.

Patho-physiological considerations

The importance of the patho-physiology 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. **(Harkin et al, 2001)**