Arterial injuries at the elbow carry a high risk of muscle necrosis and warrant urgent revascularisation

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ABSTRACT

INTRODUCTION Revascularisation following axial arterial system injury is effective in upper limb salvage but necrosis of muscle, the tissue most sensitive to ischaemia, may still occur. We examined the frequency of necrosis, its related factors and its functional significance.

METHODS The clinical findings and operative management of 13 patients with injuries at the elbow referred to 2 plastic surgical hand surgery units over a 30-month period were reviewed. Good outcome was defined as minimal impairment with return to previous occupation, intermediate outcome as moderate impairment with change in occupation and poor outcome as major functional loss preventing work.

RESULTS Seven patients injured the brachial and six injured both the radial and ulnar arteries. Concomitant injuries were severe with nerve injuries in 11 and muscle damage in 12 patients. Functional outcome was good in four cases, intermediate in four and poor in five. Muscle necrosis developed in four brachial artery injuries. In all four cases, initial successful revascularisation failed post-operatively. Case review revealed delayed recognition in three cases where pain heralded ischaemia but distal skin circulation and pulses were adequate. Of patients with necrosis, three had a poor outcome and one had an intermediate outcome.

CONCLUSIONS The risk of muscle necrosis must be considered when managing these injuries, particularly if initial revascularisation is unsuccessful. Every effort should be made to optimise repair technique and post-operative monitoring. Limb salvage is no longer enough. Fully viable muscle is necessary to restore function and livelihoods.

KEYWORDS

Upper extremity – Vascular system injuries – Muscles – Necrosis

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Of the tissues of the upper limb, muscle is the most susceptible to ischaemia. In arterial injuries this ischaemia may result from a lack of proximal inflow or from direct muscle injury and may be compounded by compartment syndrome.^{1,2} As little as 2 hours of complete ischaemia has been demonstrated to result in necrosis with loss of a significant proportion of muscle bulk after 4–6 hours.^{3,4} This necrosis may affect individual muscles or groups of muscles as in Volkmann's contracture. The functional consequences may vary from mild to severe depending on the extent of necrosis and concomitant injuries.⁵

Injuries of the axial arterial system of the upper limb have a spectrum of presentations. Those occurring at a proximal level have a dramatic presentation with severe bleeding and a clear need for emergency surgical intervention to control haemorrhage and revascularise the limb. By contrast, in the distal forearm bleeding can generally be

controlled by pressure and elevation, and there is little risk of exsanguination or of tissue ischaemia due to the plentiful collateral circulation.

Between these anatomical extremes there are particular problems in the elbow region where injury of the axial vessels (brachial, radial and ulnar arteries) results in bleeding that may be controllable by pressure or by surgery. Distal pulses may be palpable in 25% of patients with a brachial artery injury and 50% of patients with an isolated radial or ulnar artery injury due to wave transmission or flow through collaterals.^{6–8} As the adequacy of distal limb circulation is often judged by clinical evaluation of the hand, the potential for muscle ischaemia and subsequent necrosis in the intervening forearm segment may be underestimated. We studied the frequency, associated factors and functional impact of necrosis in cases presenting to our units.

Methods

The management of all patients presenting with an axial arterial injury between the mid-arm and mid-forearm (brachial or both radial and ulnar arteries) over a 30-month period to 2 regional, plastic surgical hand units located in Manchester and Glasgow was analysed. Data on demographics, mechanism of injury, mode of referral, surgical technique, muscle necrosis requiring debridement, revisional surgery, complications and outcomes were collected. Good functional outcome was defined as no documented impairment with the ability to return to original employment. Intermediate outcome was defined as a minor functional impairment but little disability (patient able to work but not in his or her original employment if manual ability was important). Poor functional outcome was defined as a significant impairment and disability including inability to work. Cases of muscle necrosis were studied in detail. Statistical analysis was performed using Fisher's exact test and Student's t-test.

Results

Thirteen cases were identified (Table 1). Nine presented directly to the two plastic surgery units. Four presented secondarily, having undergone initial revascularisation elsewhere. There were 11 men (85%) and 2 women (15%). The median age was 28 years (range: 17–42 years). Seven patients sustained brachial artery injuries and six injured both the radial and ulnar arteries. There were eleven complete transections, one partial transection and one crush injury. Eight injuries were caused by glass, three by knives, one by machete and one by a dog bite.

All 13 patients suffered concomitant damage to either peripheral nerves or muscle (Table 1). Eleven

(85%) suffered peripheral nerve damage. Twelve (92%) suffered primary damage to muscle. The median number of muscle bellies injured was 4 (range: 0–9). In 10 patients (77%) there was damage to both nerve and muscle. The four patients referred for care after revascularisation in other units had all had macrovascular repairs. The remaining nine patients underwent microsurgical repair. Direct repair was performed in seven cases and vein grafting in six. In five cases (3/4 macrovascular and 2/9 microvascular) anastomotic revision was necessary. Additional revisional surgery was required in seven cases including revision of fasciotomies, nerve repair, further wound debridement, split skin grafting, and pedicled and free flap reconstruction.

Muscle necrosis occurred in four cases (31%). Patients with necrosis had a higher incidence of failure of the initial arterial repair ($p=0.007$), vein grafting ($p=0.02$), injury to the brachial artery ($p=0.049$), fasciotomies ($p=0.049$) and revisional surgery ($p=0.049$). The incidences of macrovascular repair (3/4 patients vs 1/9 patients, $p=0.05$) and compartment syndrome (2/4 patients vs 0/9 patients, $p=0.077$) were higher although not significantly. There was no significant association with patient age, number of injured nerves (median: 1 nerve injured in patients with and without necrosis) or primary muscle injury (median: 4 injured muscle bellies with and without necrosis).

Overall functional outcome was good in four cases, intermediate in four cases and poor in five cases (Table 1). A worse outcome was associated with necrosis although this was not statistically significant. Of the five cases with poor results, three had both necrosis and nerve injuries, and two had nerve injuries alone (one median nerve palsy and one median and ulnar nerve palsy, Patients 8 and 13).

Table 1 Patient data

Patient	Sex	Age	Referral	Level	Mechanism	Nerve damage	Muscle bellies damaged	Initial repair	Vein graft	Fasciotomies	Initial repair successful	Muscle necrosis	Function*
1	M	29	Secondary	BA	Glass	MN	2	Macro	Yes	Yes	No	Yes	Poor
2	M	19	Secondary	BA	Glass	RN	6	Macro	Yes	Yes	No	Yes	Intermediate
3	M	29	Secondary	BA	Glass	MN	2	Macro	Yes	Yes	No	Yes	Poor
4	F	30	Primary	BA	Dog bite	MN, UN	9	Micro	Yes	Yes	No	Yes	Poor
5	M	25	Secondary	BA	Knife	MN	0	Macro	No	No	Yes	No	Intermediate
6	F	42	Primary	RA, UA	Knife	No	4	Micro	No	No	Yes	No	Good
7	M	38	Primary	BA	Knife	MN	1	Micro	No	No	Yes	No	Intermediate
8	M	34	Primary	RA, UA	Machete	MN, UN	6	Micro	No	Yes	Yes	No	Poor
9	M	18	Primary	RA, UA	Glass	UN	4	Micro	No	No	Yes	No	Good
10	M	20	Primary	RA, UA	Glass	MN	5	Micro	No	Yes	Yes	No	Intermediate
11	M	24	Primary	RA, UA	Glass	MN	2	Micro	No	No	Yes	No	Good
12	M	28	Primary	BA	Glass	No	1	Micro	Yes	Yes	No	No	Good
13	M	17	Primary	RA, UA	Glass	MN	7	Micro	Yes	No	Yes	No	Poor

BA = brachial artery; RA = radial artery; UA = ulnar artery; MN = median nerve; RN = radial nerve; UN = ulnar nerve; Macro = macrovascular; Micro = microvascular

*Good functional outcome was defined as no documented impairment. Intermediate outcome represents a minor functional impairment but little disability (such that the patient was able to return to work). Poor functional outcome was defined as a significant impairment and disability preventing normal use of the hand.

Figure 1 Patient 1: Initial subcutaneous fasciotomies (A); thrombosis in vein graft (B); revision flexor fasciotomy demonstrating extent of muscle necrosis (C); outcome demonstrating maximal finger and wrist flexion following loss of most of the flexor compartment (D)

In order to further evaluate the occurrence of muscle necrosis, we studied in detail the cases in which it occurred.

Patient 1

This 29-year-old right hand dominant man (Table 1, Patient 1) sustained a glass laceration resulting in complete transection of the brachial artery, common flexor origin and median nerve. Initial macrovascular vein grafting and subcutaneous fasciotomies (Fig 1A) restored a radial pulse but not muscle perfusion and despite severe pain, this was not recognised. On transfer we performed urgent microsurgical revision of the now thrombosed vein graft and extension of the fasciotomies (Figs 1B and 1C). Necrosis of most flexor muscles ensued and four debridements, an unsuccessful free functioning gracilis transfer and tendon transfers were undertaken. Compartment syndrome complicated therapy. Weak pinch and large object grasp were eventually achieved but the patient could not return to work as a driver.

Patient 2

This 19-year-old right hand dominant male gym worker (Table 1, Patient 2) sustained a glass laceration resulting in transection of the brachial artery, common flexor and extensor origins, biceps, brachialis, triceps, and deep and superficial branches of the radial nerve. Initial macrovascular vein grafting and subcutaneous fasciotomies were performed, restoring hand perfusion. However, re-exploration in our unit 24 hours later revealed a thrombosed vein graft and partly necrotic common extensor origin requiring microsurgical revision and debridement. By 14 months there was good motor and almost full sensory recovery and the patient returned to his previous occupation.

Patient 3

Due to intoxication, this 28-year-old right hand dominant male heavy goods vehicle driver (Table 1, Patient 3) could not recall how he transected his brachial artery or injured

his median nerve and flexor carpi ulnaris and brachioradialis muscles. Initial surgery consisted of macrovascular vein grafting of the brachial artery with a second graft from the first to the radial artery due to poor radial perfusion. Despite subcutaneous fasciotomies, compartment syndrome developed but despite severe pain, this was not diagnosed due to the presence of peripheral pulses and hand perfusion. On transfer to our unit three days later, exploration revealed thrombosed vein grafts. Microsurgical revision was performed as well as fasciotomy extension. Partial necrosis of flexor digitorum superficialis and flexor digitorum profundus developed, and recovery was complicated by contracture. At 7 months the patient had a grip strength of 4/5 and improving sensation. Although he was unable to return to his original occupation, he found alternative employment.

Patient 4

This 30-year-old right hand dominant female drug abuser (Table 1, Patient 4) was attacked by a pit bull terrier and sustained a crush injury to the left brachial artery and crush/avulsion of biceps, brachioradialis, flexor carpi radialis, flexor digitorum superficialis, flexor carpi ulnaris, flexor pollicis longus, extensor carpi radialis longus and brevis, palmaris longus and the median nerve. She was admitted directly to our unit where vasospasm of the brachial artery initially improved in theatre but later thrombosed, requiring re-exploration five hours later with microvascular vein grafting, debridement and fasciotomies.

Pulses were palpable post-operatively and the hand remained perfused but over the course of five further operations it was necessary to debride necrosis of the biceps, brachioradialis, flexor carpi radialis, palmaris longus, flexor digitorum superficialis, most of the flexor carpi ulnaris, 50% of the flexor pollicis longus and most of the extensor carpi radialis longus and brevis. The arm was reconstructed using a pedicled, innervated latissimus dorsi flap and split skin grafts. Functional outcome was poor, with limited elbow flexion, median nerve palsy and loss of long flexor function. The patient remained unemployed.

Discussion

Our patients suffered a high level of damage to nerves and muscles, which were damaged in 11 of 13 (85%) and 12 of 13 cases (92%) respectively. Previous studies found an incidence of nerve injury between 24.6% and 68% in brachial artery injuries and up to 100% in blunt injuries.⁹⁻¹¹ To our knowledge, no other series has documented direct muscle damage although McCroskey *et al* documented tendon injury in 13% of brachial artery injuries.⁹

Muscle necrosis developed in 4 of 13 patients (31%) and appeared to affect outcome, with poor function in 3 of these 4. None of the patients with necrosis made a good recovery. Peripheral nerve injuries have been suggested to be the primary determinants of disability in upper extremity vascular injuries.⁵ However, although they were present in all five of our patients with poor outcomes, they were also present in all four with intermediate and half of those with good outcomes (2 of 4 patients), confirming satisfactory recovery is

possible. As necrosis will degrade the motor targets of peripheral nerves and the causative ischaemia is also likely to affect neural tissue, it is likely to indicate a compound neuromuscular insult. Similarly, nerve injury may reduce pain perception, increasing susceptibility to compartment syndrome and further muscle ischaemia. Nerve and muscle injury therefore seem likely to be synergistic.

We found a significant association between necrosis and failure of the initial arterial repair. This will inevitably result in a period of secondary warm ischaemia. As noted above, muscle may become necrotic after as little as two hours although this may be greater for secondary ischaemia due to a protective preconditioning effect.^{5,4,12–14} The strength and duration of preconditioning in humans is not known but prolonged ischaemia will inevitably result in necrosis.

Technical factors are important for successful revascularisation. In Patients 1, 2 and 3 the initial vein graft repairs thrombosed. All three cases were sharp glass lacerations. The brachial artery is unusual in that on sharp division at the elbow it retracts proximally but on reanastomosis it resumes its original length. A segment may also appear to have been lost should repair be attempted with the elbow extended, risking kinking on flexion during later tendon and nerve repair. However, should there be segmental arterial damage as typified by blunt injuries, then resection and vein grafting is essential. In the crushing injury seen in Patient 4, prompt vein grafting might have prevented secondary ischaemia. Careful consideration is therefore necessary when deciding whether to perform vein grafting. Vein grafts introduce an additional anastomosis and a section of vein that is often of larger diameter with subsequent slower and more turbulent flow. Microvascular technique may help to optimise flow.¹⁵

Post-operative monitoring is crucial to identify secondary ischaemia. The perfusion of the hand and the presence of pulses do not accurately reflect the vascularity of forearm muscle.^{6–8} In Patients 1 and 3 there was a failure to identify secondary ischaemia that delayed re-exploration and allowed compartment syndromes to develop. Pain and resistance on passive extension of the fingers are the earliest signs of muscle ischaemia and precede compartment syndrome. Patients must be monitored by senior clinical staff prepared to take prompt action should these signs develop.

In Patients 1 and 3 secondary ischaemia progressed to compartment syndrome despite the use of subcutaneous fasciotomies. This technique has been criticised in the lower limb as the undivided distal skin may act as a tourniquet.¹⁶ It may also result in incomplete fascial release and failure to relieve post-ischaemic contraction.² We advocate fasciotomies with direct exposure and division of all constricting fascia.

Clearly, combined arterial and nerve injuries are severe, presenting a challenge to treat and monitor, and also risking necrosis and a poor outcome. When isolated, they are also usually treated separately by vascular and hand (orthopaedic or plastic) surgeons respectively. We believe that a team approach to the management of these injuries is required to optimise outcomes.

Limitations of the current study

In interpreting our results some limitations must be borne in mind. First, the sample is unlikely to be representative of these injuries as a whole. Our data suggest the injuries are more severe than normal and this is likely to be due to the selected nature of patients referred to the regional plastic surgical hand unit. It is therefore feasible that the overall incidence of muscle necrosis is lower. However, due to its significant functional impact, we feel it warrants emphasis, particularly given the potential for mitigation with the proposed optimisation of surgical technique and monitoring.

Second, no formal measurements of function or patient-assessed outcomes were obtained. These would quantify and add validity to the gross functional deficits we have described.

Conclusions

We believe that the risk of muscle necrosis should be given a stronger emphasis in the management of these injuries. Together with the available literature, our series points to

several principles that can be applied in order to achieve the optimum outcome:

- Revascularisation should be prompt to reduce primary ischaemia. We would suggest these injuries are treated within the same time constraints as arm replantation as it is difficult to predict how much muscle is devascularised and subjected to warm ischaemia. A team approach between treating vascular and hand surgeons is required and should help avoid delays.
- Initial surgery should strive for the optimal repair and surgeons should have a low threshold for performing full fasciotomies.
- Post-operative monitoring is crucial. The best indicator of muscle perfusion is the presence or absence of muscle pain. Its presence should prompt action. As local anaesthetic blocks and heavy analgesia may mask a deteriorating situation, these should be avoided.

We propose a protocol for the management of these injuries (Fig 2). In the modern management of upper limb arterial injuries, limb salvage is no longer enough. Instead, fully viable muscle is necessary to restore function and livelihoods.

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