Quantifying delays in the recognition and management of acute compartment syndrome

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ABSTRACT

Objective: To identify where most efforts should be made to decrease ischemia time and necrosis in acute compartment syndrome (ACS) and to determine the causes for late interventions.

Methods: This was a multicentre, historical cohort study of patients who underwent fasciotomy for ACS within the McGill Teaching Hospitals between 1989 and 1997. Patients studied had a clinical diagnosis of ACS or compartment pressures greater than 30 mm Hg. In all cases, ACS was confirmed at the time of fasciotomy. Patients were stratified into traumatic and non-traumatic groups, and a step-by-step analysis was performed for each part of the process between injury and operation.

Results: Among the 62 traumatic ACS cases, the longest delays occurred between initial assessment and diagnosis (median time 2h56, range from 0 to 99h20) and between diagnosis and operation (median 2h13, range 0h15–29h45). Among the 14 non-traumatic ACS cases, delays primarily occurred between inciting event and hospital presentation (median 9h19, range 0h04–289h29) and between initial assessment and diagnosis (median 8h18, range 0–104h15).

Conclusions: ACS is a limb-threatening condition for which early intervention is critical. Substantial delays occur after the time of patient presentation. For traumatic and non-traumatic ACS, increased physician awareness and faster operating room access may reduce treatment delays and prevent disability.

RÉSUMÉ

Objectif : Identifier les endroits où concentrer les efforts pour réduire le délai d'ischémie et la nécrose dans les cas de syndrome compartimental et déterminer les causes des interventions tardives.

Méthodes : Étude historique et multicentrique d'une cohorte de patients ayant subi une fasciotomie pour un syndrome compartimental aux hôpitaux universitaires de McGill entre 1989 et 1997. Les patients à l'étude présentaient un diagnostic clinique de syndrome compartimental ou de compression de la loge supérieure à 30 mm Hg. Dans tous les cas, le syndrome compartimental fut confirmé au moment de la fasciotomie. Les patients furent répartis en sous-groupes traumatiques et non traumatiques et une analyse étape par étape fut effectuée pour chaque partie du processus entre la blessure et l'intervention.

Résultats : Parmi les 62 cas de syndrome compartimental traumatique, le délai le plus long se produisit entre l'évaluation initiale et le diagnostic (délai médian 2h56, intervalle de 0 à 99h20) et entre le diagnostic et l'intervention (médian 2h13, intervalle 0h15–29h45). Parmi les 14 cas de syndrome compartimental non traumatique, les délais se produisirent principalement entre l'événement causal et la consultation à l'hôpital (médian 9h19, intervalle 0h04–289h29) et entre l'événement causal et le diagnostic (médian 8h18, intervalle 0–104h15).

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Conclusions : Le syndrome compartimental est une atteinte menaçant l'intégrité des membres pour lequel une intervention précoce est essentielle. Des délais importants se produisent après la visite initiale des patients. Pour les syndromes compartimentaux traumatiques et non traumatiques, une meilleure sensibilisation des médecins et un accès plus rapide à la salle d'opération peuvent permettre de réduire les délais de traitement et prévenir les incapacités.

Key words: compartment syndromes, ischemia, necrosis, time factors

Introduction

Acute compartment syndrome (ACS) is a limb-threatening condition in which increased pressure within muscle compartments compromises nutrient blood flow to muscles and nerves.[1–6] Although fasciotomy with direct examination of the muscle compartment is the diagnostic "gold standard" for ACS, the diagnosis is often made clinically and may be confirmed by direct needle measurement of compartment pressure.[6–9] The only effective therapy is decompression of the affected compartments by fasciotomy.[6,10–13] If decompression is delayed, muscle necrosis and permanent disability may result.[11,14]

Current knowledge of muscle ischemia tolerance is derived from tourniquet-induced ischemia models, which suggest that muscle can tolerate up to 3 hours of ischemia before necrosis ensues.[15–18] However, a recent McGill University study of 76 patients who underwent fasciotomy[19] estimated that 35% of all ACS patients and 72% of those who developed necrosis did so within 2 hours of injury — earlier than the 3-hour safe period suggested by ischemia models.

Because ACS patients are at high risk of developing necrosis, it is important to reduce delays to definitive therapy. Our primary objective was to determine the reasons for delayed intervention in cases of acute compartment syndrome. Our secondary objective was to determine whether reasons for delay differed in traumatic and non-traumatic cases.

Methods

Setting

This multicentre, historical cohort study was performed at McGill University, Montreal, Canada. It was approved by the ethics committees of all participating hospitals.

Patients

For medicolegal and remuneration purposes, all fasciotomies performed at the 4 McGill University teaching hospitals are recorded in the hospitals' electronic databases. We used these databases to identify patients who underwent fasciotomy between 1989 and 1997. Because clinically important ACS does not resolve without treatment, and because the only therapy for ACS is fasciotomy,[6,10–13] we believe this search strategy captured all clinically significant ACS cases that occurred during the study period.

Patients were eligible for inclusion if the treating physician or nurse specifically documented a concern that ACS was present, if compartment pressure measurements >30 mm Hg were recorded or if ACS was discovered at the time of surgery (e.g., during internal fixation of a fracture). The diagnosis of ACS was accepted only if it was confirmed at fasciotomy. Patients who underwent fasciotomy for reasons other than ACS (e.g., prophylactic fasciotomy prior to vascular bypass surgery or embolectomy[20]) were excluded from analysis.

Data collection

Each chart was abstracted by 1 of 2 physicians (C.V. and D.S.), using a defined process, explicit definitions and specific data abstraction forms to increase reliability. All data forms were double-checked to assure accuracy, and missing data were reported as such. In cases of ambiguous data, consensus agreement was reached between the abstractor and senior author (I.S.). All data were transcribed into an Excel database.

Definitions

Trauma was defined as any violent contact with the body. Time of injury or inciting event (for traumatic and non-traumatic cases, respectively) was the time documented or estimated in the chart. Time of presentation was defined as the time the patient presented to the enrolling hospital, and time of assessment was when a physician saw the patient. When presentation time was not specified in the nurses' or physicians' notes we used the time of registration (major trauma victims are always seen before they are registered). Time of diagnosis was defined as the time physicians documented a diagnosis in the chart. Time of surgery was defined as the start of anesthesia because this time point is recorded reliably. Confirmation of ACS required a surgeon's report documenting typical findings of ACS (e.g., dusky or grey muscle protruding from a tense fascia, and absent twitch response to stimulation).

Data analysis

Intervals studied were: injury-to-hospital presentation, presentation-to-assessment, assessment-to-diagnosis and diagnosis-to-operation. Patients whose inciting event occurred in hospital were excluded from the injury-to-presentation and presentation-to-assessment analyses. Patients whose diagnosis was made in the operating room were excluded from the diagnosis-to-operation analysis. Median values and ranges were calculated for continuous data. Time intervals were compared for traumatic versus non-traumatic cases. The statistical significance of observed differences was determined using the Mann–Whitney U-test for non-parametric data. Calculations were performed using StatView version 5.0 (SAS Institute Inc., Cary, NC).

Results

Between 1989 and 1997, 237 patients underwent fasciotomy at McGill University teaching centres. Of these, 151 were excluded because fasciotomy was performed for reasons other than ACS (e.g., to access fracture sites, for plantar fascia release in cases of clubfoot or plantar fasciitis). Ten cases of ACS associated with necrotizing fasciitis were also excluded because, in these cases, muscle necrosis could not be solely attributed to ACS. Table 1 summarizes baseline characteristics of the 76 patients who were eligible for analysis. Of interest, only 34 of these 76 patients had compartment pressures measured. In both traumatic and non-traumatic groups, the diagnosis of ACS was usually made on clinical grounds.

Table 1. Characteristics of patients with acute compartment syndrome (ACS) (*n* = 76)

Characteristic	No. (and %)*
Median age (range), yr	32 (1–80)
Traumatic etiology	62 (82)
Male gender	57 (75)
Associated fracture	40 (53)
Transferred from referring hospital	14 (18)
Alcohol or drug intoxication	12 (16)
Vascular disease present	11 (14)
Receiving anticoagulant medication	7 (9)
Injured while in hospital	7 (9)

*Unless otherwise indicated.

Table 2 illustrates where time delays occurred. Note that 7 patients whose inciting event occurred in the hospital were excluded from the injury-to-hospital and presentation-to-assessment analyses. These 7 events included intravenous infiltration (1), surgical correction of genu valgus (1), intra-aortic balloon insertion (1), angioplasty catheter insertion (1) and internal fixation of a fracture (3). Twelve patients were excluded from the diagnosis-to-operation analysis because their diagnosis was made in the operating room (OR) during surgery for another reason (e.g., fracture fixation).

Of the 14 non-traumatic cases, 6 occurred after muscular exertion, 6 after prolonged immobility in the context of a drug overdose, 1 was related to Ehlers–Danlos syndrome, and 1 was due to eosinophilic fasciitis.

The data show that post-traumatic ACS cases present to hospital rapidly (about 1 hour) after the causative event compared with more than 9 hours for non-traumatic cases. This was true despite the fact that 14 of our patients required transfer from peripheral hospitals. After presentation, traumatic cases waited less for assessment (median delay = 0 vs. 26 min; $p = 0.036$). Although the difference is not statistically significant, trauma cases also had shorter assessment-to-diagnosis intervals (2h56 vs. 8h18; $p = 0.13$) and shorter diagnosis-to-operation intervals (median = 2h13 vs. 3h08; $p = 0.20$).

Three patients experienced extraordinary long diagnosis-to-operation times. One of these patients was waiting for open-heart surgery, and fasciotomy was deferred 28h25 so both procedures could be performed during a single anesthetic. A second patient waited 42h25 between diagnosis and fasciotomy because a resident failed to notify the attending orthopedist of the diagnosis. In the final case, physicians waited 29h45 after ACS diagnosis to perform fasciotomy at the same time as open reduction and internal fixation.

Table 2. Time delays in ACS management (*n* = 76)

	Median times, hrs:min (range)		
	Traumatic (*n* = 62)	Non-traumatic (*n* = 14)	*p*
Event-to-hospital presentation*	1:10 (0:03–48:33)	9:19 (0:04–289:29)	0.015
Presentation-to-assessment*†	0:00 (0:00–1:38)	0:26 (0:00–2:10)	0.036
Assessment-to-diagnosis	2:56 (0:00–99:20)	8:18 (0:00–104:15)	0.13
Diagnosis-to-operation‡	2:13 (0:15–29:45)	3:07 (1:10–42:25)	0.20
Total (event-to-operation) §	9:47 (1:10–106:45)	34:43 (7:00–396:10)	<0.001

* Excludes 7 patients who were injured while in hospital.
† 37 of 62 traumatic cases were assessed immediately; therefore, median delay is 0:00.
‡ Excludes 12 patients whose diagnoses were made in the OR.
§ Total time is not the sum of interval times because median times cannot be added.

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Discussion

The longest delays in patients with traumatic ACS occurred because of failure to make an early diagnosis and inability to mobilize the operating room. Delays in non-traumatic cases were primarily due to late hospital presentation and failure to make a timely diagnosis. In both forms of ACS, the diagnosis is often not suspected or not promptly recognised by emergency physicians and admitting services.

These results confirmed our clinical suspicion that traumatic cases are assessed, diagnosed and treated more rapidly than non-traumatic cases. There are several likely reasons for this. Trauma victims present acutely to the ED, often by ambulance; whereas patients with non-traumatic ACS are likely to delay seeking care, perhaps because of insidious symptom progression or perhaps with hopes that symptoms will subside. In addition, physicians see traumatic cases more quickly after ED arrival,. This is not surprising. ED physicians repeatedly learn the importance of "the golden hour" in trauma care and have been indoctrinated into the trauma team mentality.

Delayed assessment of non-traumatic ACS may also reflect a shortfall in existing triage protocols. For instance, the Canadian Emergency Department Triage and Acuity Scale (CTAS)[21] recommends that major trauma victims be seen immediately but that patients presenting with non-traumatic extremity pain can wait 15 minutes if the pain is severe (>8/10) and 1 hour if the pain is moderate (4–7/10). Although these recommendations are appropriate in most cases, triage personnel should be alerted to exceptions. We therefore suggest educational efforts to increase the awareness of ACS.

Many consider ACS to be exclusively a trauma-related condition. In this series, however, we documented 14 non-traumatic cases. Therefore, even in the absence of trauma, physicians should think of ACS in patients who have pain out of proportion to physical findings ("out of proportion" may be difficult to judge, however, since patients have variable pain thresholds and emergency physicians seldom know the patients they treat). Useful signs to confirm ACS include pain on passive muscle stretch, palpable compartment tenseness, weakness of the affected muscles, hypoesthesia in the distribution of nerves traversing the compartment, and loss of pedal pulses.[5,11,14] It is important to note, however, that loss of pulses and neurological function are late signs and that the diagnosis should be made before these occur.

Pressure measurement is an important emergency medicine skill, and emergency physicians should consider checking compartment pressures in cases of suspected ACS. Physicians should also recognise that ACS is a dynamic condition, that compartment pressures change over time, and that the extent of tissue ischemia depends on regional perfusion pressure — the difference between diastolic pressure and intracompartment pressure.[10] Borderline compartment pressures over 20 mm Hg may cause vascular compromise and should trigger follow-up measurements, while pressures >30 mm Hg are considered diagnostic of ACS.

Given the importance of rapid treatment, the prolonged diagnosis-to-operation intervals seen in this study are concerning. Reasons for delay may include poor operating room (OR) time management and absence of reserved OR time for emergencies.[22–25] We suspect, in addition, that physicians have been falsely reassured by tourniquet studies, which have suggested that muscle can tolerate up to 3 hours of ischemia.[15–18] However, tourniquet-induced ischemia may be less damaging than ACS-induced ischemia. In the only study to address this possibility, Heppenstal and associates[16] found that pH and phosphocreatine recovered rapidly and fully in dogs subjected to tourniquet-ischemia, but only slowly and partially in ACS-induced ischemia. Cellular ATP (adenosine triphosphate) remained normal in the tourniquet group, whereas it decreased and never recovered completely in the ACS group. Finally, ultrastructural degeneration in the skeletal muscle was more common and more severe in the ACS group. These results suggest that we cannot generalize studies of tourniquet-induced ischemia to patients with ACS.

Limitations

This study suffers from the shortfalls inherent in its retrospective design. A prospective study could have provided more accurate and extensive clinical information, but ACS is an uncommon condition and such conditions are difficult to study prospectively. In addition, it is likely that a prospective study would have increased ACS awareness, changed practice patterns and decreased the door-to-surgery time, introducing a Hawthorne effect and limiting the validity of the results. We feel that, despite its limitations, a historical cohort design was the best method for this study.

We documented long delays from event-to-presentation and, in some cases, from diagnosis-to-operation. Depending on the degree of ischemia and rate of progression, these delays may or may not have contributed to adverse patient outcomes. Unfortunately, in a retrospective review it is difficult to gather reliable clinical status information; therefore we can only suggest that delays seemed excessively long, not that they caused harm in specific cases.

Because we studied patients in university teaching hospitals, referral bias may have occurred, and patients in this study may differ from those seen in smaller community hospitals. In addition, diagnosis-to-surgery delays may differ in different settings, depending on local expertise and OR availability.

Given the retrospective nature of the study, we may be

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unaware of important confounding events. For example, in 4 cases, we learned that casts had been applied between the time of the injury and the ACS diagnosis. In these cases, the cast application (rather than the injury) may have caused the ACS.[6,26] Had we known the time of cast application, we might have determined a shorter time interval between inciting event and onset of necrosis.[19]

Finally, when patients were transferred from peripheral hospitals, we considered the times of presentation and assessment to be the times at the referral centres. If we had used peripheral hospital times, the intervals from presentation-to-assessment, assessment-to-diagnosis and diagnosis-to-operation would have been different. Consequently, our results may underestimate the true delays for some patients.

Conclusions

Acute compartment syndrome is a limb-threatening condition for which early intervention is critical. Tissue necrosis may begin earlier than the 3 hours suggested in prior studies. Emergency physicians should be aware of ACS symptoms and signs, and should be capable of measuring compartment pressure in cases where the diagnosis is unclear.

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