Pediatric Nonfracture Acute Compartment Syndrome: A Review of 39 Cases

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Background: Compartment syndrome in the absence of fracture is rare and poorly described within the pediatric literature. The purpose of this study was to report the varying etiologies, risk factors, and treatment outcomes associated with pediatric nonfracture acute compartment syndrome (NFACS).

Methods: We conducted a retrospective chart review on 37 children who suffered a NFACS and were treated at a single pediatric trauma center between 1997 and 2013. Demographic, diagnostic, treatment, and outcome characteristics were reviewed. Five causal groups were generated: trauma, exercise related (acute presentation after exercise without trauma), infectious, vascular, and postoperative (in the absence of osteotomy). Univariate and multivariate analyses were performed to identify risk factors of NFACS. *P*-values <0.05 were considered statistically significant.

Results: There were 39 cases of NFRCS in 37 children [6 females, 31 males, mean age of 11.7 y (SD + 7.2 y)]. The leg was the most commonly involved limb (29 cases, 74%). Diagnosis of NFRCS was made either by compartment pressure monitoring [59%, 23/39 cases, mean pressure 66 mm Hg (SD + 28)] or by clinical examination. According to etiology, vascular was most common (11/39, 28%), followed by trauma (10/39, 26%) and postoperative (8/39, 21%), with exertion and infection representing a small proportion (6/39, 15% and 4/39, 10%, respectively). Pain was present in 33 cases (85%), swelling in 28 cases (72%), paresthesias in 13 cases (33%), motor deficit in 12 cases (31%), and poor perfusion in 11 cases (28%). Average time from symptom onset to diagnosis was 48 hours (IQR, 9 to 96 h). At surgery, 21 patients (54%) had evidence of myonecrosis. Children required an average of 3 surgeries for wound closure. The median time to follow-up was 232 days (IQR, 73 to 608 d). A total of 54% made a full recovery, whereas 31% suffered a persistent neurological or functional deficit.

Conclusions: NFACS in children is associated with a delay in diagnosis and a high rate of myonecrosis. Timely assessment with high clinical suspicion is necessary to prevent a delay in diagnosis.

Level of evidence: Level IV.

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Acute compartment syndrome (ACS) is a potentially devastating condition of elevated pressure within a closed fascial space that can lead to muscle necrosis and nerve injury if not treated promptly by fasciotomy.[1,2] The incidence of ACS after pediatric fractures in the forearm and leg is estimated to be 1% and 3%, respectively, and physicians caring for these patients have high awareness of this clinical entity.[3–5] In contrast, there may not be heightened awareness of the risk of ACS in trauma without fracture or in atraumatic presentations, and low suspicion can delay diagnosis and treatment.[6–9] Despite ample literature on ACS in adults, far fewer studies have examined ACS in children,[2,3,10–13] who are at particular risk for delay in diagnosis.[4] Furthermore, there are only scattered reports discussing ACS without fracture in children.[11,13–15] We present a retrospective case series of pediatric patients with nonfracture acute compartment syndrome (NFACS) to describe the varying etiologies, risk factors, symptoms, treatments, and outcomes of this uncommon but potentially catastrophic condition.

METHODS

Study Design

After IRB approval, we conducted a retrospective search of our institutional database of operative reports from 1997 to 2013 to identify patients who underwent fasciotomies for ACS. Chronic exertional compartment syndrome and ACS after fracture or osteotomy were excluded. The remaining cohort's medical records were reviewed to collect demographic, diagnostic, treatment, and outcome data. Demographic data included age, sex, referral source, limb involved, and etiology. Diagnostic data included presenting symptoms (pain, swelling, paresthesias, paralysis, and/or decreased perfusion as defined in the medical record), diagnosis modality (clinical examination or compartment pressure), compartment pressure measurement (mm Hg if measured), and time to diagnosis. Treatment data included number of compartments released, presence of muscle necrosis at index or subsequent surgery, number of surgeries, and need for complex wound closure. Outcome data included presence

of neurological (motor or sensory) deficit or pain at final follow-up.

After initial analysis, 5 etiologic groups were created: traumatic (without fracture), exercise related (acute presentation after exercise without trauma), infectious, vascular, and postoperative (without osteotomy). Time to diagnosis does not represent duration of NFACS before fasciotomy, but rather time from injury in the traumatic group, time from onset of symptoms in the exercise-related and infection groups, time from end of surgery in the postoperative group, and time from vascular access in the vascular group.

Statistical Analysis

Demographic, diagnostic, treatment, and outcome characteristics were summarized. Variables were compared across the 5 etiologic groups using a χ^2 test, analysis of variance (ANOVA), or the Kruskal-Wallis test, as appropriate. Outcome variables (pain at follow-up, motor/sensory deficit at follow-up) were compared across groups with a χ^2 test, Fisher exact test, or univariate logistic regression, as appropriate. Both ordinal logistic regression and penalized likelihood logistic regression were used to analyze the data. The 2 methods yielded similar results, but given our small number of events, to reduce bias, the penalized logistic regression values are reported. For significant factors, penalized likelihood odds ratios with corresponding 95% confidence intervals were estimated. *P*-values <0.05 were considered significant.

TABLE 1. Patient and Diagnosis Characteristics (N = 39)

Patient Characteristics	Frequency (%)
Sex (male)	33 (85)
Age (mean ± SD)	11.7 ± 7.24
Clinic	1 (3)
Emergency department	13 (33)
Orthopaedic hospitalization	2 (5)
Other hospitalization	16 (41)
Outside hospital	7 (18)
Clinical examination	16 (41)
Pressure	23 (59)
Side (left)	21 (54)
Upper extremity	10 (26)
Arm	1
Hand	5
Forearm	9
Lower extremity	29 (74)
Foot	1
Leg	27
Thigh	1
Trauma	10 (26)
Exercise related	6 (15)
Infection	4 (10)
Postoperative	8 (21)
Vascular insults	11 (28)
Pain	33 (85)
Swelling	28 (72)
Paresthesias	13 (33)
Motor deficit	12 (31)
Poor perfusion	11 (28)
1	6 (15)
2	15 (38)
3	12 (31)
4	5 (13)
5	1 (3)
Pressure (mean ± SD, N = 21) (mm Hg)	66 ± 27.91
Time to diagnosis [median (IQR)] (h)	48 (9-96)

IQR indicates interquartile range (25th percentile-75th percentile).

RESULTS

Patient Characteristics

There were 39 cases of NFACS in 37 children, of which 33 were males, with a mean age of 11.7 years (range, 0 to 22 y). During the same study period, a total of 84 fracture-related compartment syndrome cases occurred. There were 2 bilateral cases in the postoperative group, both after prolonged lithotomy position, 1 for reconstructive genital surgery and 1 for bowel resection. Most cases were in the lower extremity. Vascular etiology was most common. This group included intravenous infiltration injuries, spontaneous bleeding from anticoagulation, arterial cannulation, intraosseus cannulation, and an occlusive arterial thrombus. The second most common etiology was trauma, which included blunt trauma from a fall or crush injuries to a limb. The postoperative etiology was the next most common, which included patients having undergone prolonged abdominal, pelvic, or urologic surgery. Two of the 8 postoperative cases involved orthopaedic hip surgeries, where the contralateral limb developed NFACS. There were no cases where the NFACS developed on the ipsilateral surgical limb. Six of 8 postoperative cases had a thoracic epidural placed for pain control. Finally, the exercise-related (from soccer, football, and track) and infectious (spontaneous deep infections of leg or forearm and hand) etiologies represented the smallest proportion of injuries related to NFACS (Table 1).

Diagnostic Characteristics

Pain was the most common symptom. Most patients presented with 2 or 3 symptoms at time of diagnosis, and only 1 patient presented with all 5 symptoms of pain, swelling, motor deficit, paresthesias, and pulselessness. The diagnosis of NFACS was made either by compartment pressure monitoring using an intracompartment handheld pressure monitor (Stryker, Kalamazoo, MI) (59%, 23/39 cases, mean pressure 66 mm Hg) or by clinical examination. When monitoring intracompartment pressure, a delta pressure <30 mm Hg between the diastolic blood pressure and intracompartment pressure was our indication for compartment syndrome. Subjects who were diagnosed by pressure monitoring tended to be older, averaging 14.7 years old (SD ± 6.5 y), compared with those diagnosed by clinical examination [averaging 8.2 y old (SD ± 6.6 y) ($P = 0.004$)]. There were no other differences in diagnostic, treatment, or outcome characteristics observed among the cohort. Median time from symptom onset or inciting event to diagnosis was 48 hours (Table 1).

Treatment and Outcome Characteristics

On average, children underwent 3 surgeries with release of 4 compartments. Myonecrosis was present in 54% of cases at index or subsequent surgery. One child required amputation. Twenty-one percent required complex wound closure, either skin graft or tissue expander. Median time to follow-up was 232 days. Of the 36 cases with complete follow-up data, most experienced a complete recovery (20/36, 56%), but 4 (11%) had pain and 12 (33%) had a motor, sensory, or other functional deficit at final follow-up. Subjects who had more compartments released had higher odds of experiencing pain at follow-up ($P = 0.03$). With each additional compartment released, the odds of pain at final follow-up increased by 30% [OR = 1.3 (95% CI, 1.03-1.64)]. Patients with myonecrosis in the OR had 8 times the odds of neurological or functional deficit at follow-up [OR = 8.3 (95% CI, 1.5-46.9), $P = 0.02$]. Time to diagnosis and compartment pressure had no significant effect on presence of neurological or functional deficit ($P = 0.71$, 0.73, respectively), presence of pain ($P = 0.21$), or need for skin graft ($P = 0.71$, 0.93) (Table 2).

TABLE 2. Treatment and Outcome Characteristics

Treatment Characteristics (N = 39)	Frequency (%)
No. surgeries (mean ± SD)	3.1 ± 2.05
No. compartments (mean ± SD, N = 38)	4.1 ± 3.71
Necrosis in OR	21 (54)
Complex wound closure	8 (21)
Skin graft	6
Tissue expander	2
Pain	4 (11)
Neurological or functional deficit	12 (33)
Length of follow-up [median (IQR)] (d)	232 (73-608)

Data are presented as frequency (%) unless stated otherwise.
IQR indicates interquartile range (25th percentile-75th percentile).

Comparison of Etiologic Groups

As the etiologic groups represented distinctly different patient populations, the demographic, diagnostic, treatment, and outcome measures were examined for each group individually and then compared across groups. Age differed significantly across groups. Infection and vascular cases were the youngest, averaging 4.2 and 4.8 years old respectively, whereas exercise-related and postoperative cases were the oldest, averaging 17.5 and 16.9 years old, respectively. There was a significant difference in the proportion of patients with paresthesias at presentation. The infectious group experienced no cases of paresthesias, whereas the exercise-related and postoperative groups had the highest proportions (67% and 63%, respectively). There was a significant difference in perfusion deficits across etiologic groups, with vascular and postoperative cases showing diminished perfusion in 64% and 38% of cases, respectively, whereas no patients in the exercise-related and infectious cases demonstrated changes in perfusion. There was a trend toward the vascular, postoperative, and exercise-related cases having a higher likelihood of neurological or functional deficit at final follow-up compared with traumatic or infectious cases. Across etiologic groups, there were no significant differences in number of symptoms, compartment pressure, time to diagnosis, number of compartments released, number of surgeries, presence of myonecrosis in the operating room, need for complex wound closure, pain at final follow-up, or length of follow-up (Table 3).

DISCUSSION

ACS is one of the most feared clinical scenarios for orthopaedic surgeons and can occur as a result of systemic disorders or local limb trauma. The presence of ACS without fracture is rare, and previously poorly described in the pediatric orthopaedic literature. The purpose of this study was to report our experiences with 39 cases of NFACS in children and adolescents, to identify the varying etiologies (vascular, traumatic, postoperative, exercise related, and infectious), risk factors, and outcomes.

Prompt, accurate diagnosis of ACS depends on the detection of clinical signs of increasing compartment pressure. Diverging views exist on the relative importance of clinical findings associated with ACS. Ulmer[16] questioned the efficacy of clinical diagnosis and found low sensitivity (13% to 19%), but high specificity (97% to 98%) for accurate ACS diagnosis. At our institution, we rely on clinical examination as the diagnostic cornerstone for ACS. Previous studies of fracture-related ACS at our institution demonstrated the use of compartment pressure monitoring in about 48% of cases.[17] In this cohort of NFACS with atypical presentations, a higher rate of compartment pressure monitoring was observed (23/39, 59%). Looking closely at this cohort, younger children were more commonly diagnosed by clinical examination compared with older children who tended to be diagnosed by compartment pressure measurement. This observation can be explained by the challenges of measuring compartment pressures in an awake child.[12] Furthermore, younger patients in our cohort were more likely to have an infectious etiology for their NFACS, which made surgical intervention unavoidable and pressure measurements less necessary for treatment decision. The observed higher rate of pressure monitoring within our cohort is likely explained by atypical clinical presentations compounded by a delay in diagnosis, and indicates that relying on clinical examination alone to diagnose NFACS may be unsatisfactory in many cases.

In children with NFACS, the diagnosis can be elusive, resulting in a long duration of time from symptom onset to treatment. Ragland et al[13] reported on 24 cases of neonatal ACS with only 1 case diagnosed within the first 24 hours of presentation. In our study, the average time from symptom onset or inciting event to diagnosis of NFACS was over 48 hours, which is almost 3 times longer than time to fasciotomy in children with ACS after tibia fractures.[2,17] This illustrates the importance of awareness of NFACS among clinicians to avoid delays in diagnosis, which can lead to severe complications, permanent deficit, and contractures.[15] In our study we believe that the delay

TABLE 3. Patient, Diagnosis, and Treatment Characteristics by Event Type

Variables	Trauma (n = 10)	Exercise Related (n = 6)	Infection (n = 4)	Postoperative (n = 8)	Vascular (n = 11)	*P*
Patient characteristics						
Age (mean ± SD) (y)	**12.8 ± 5.7**	**17.5 ± 2.3**	**9 ± 4.2**	**16.9 ± 5.4**	**4.8 ± 6.6**	**0.02**
Sex (male)	9 (90)	6 (100)	2 (50)	8 (100)	8 (73)	0.11
Diagnosis characteristics						
Diagnosed by						
Clinical examination	4 (40)	1 (17)	3 (75)	2 (25)	6 (55)	0.28
Pressure	6 (60)	5 (83)	1 (25)	6 (75)	5 (46)	
Side (left)	5 (50)	2 (33)	2 (50)	5 (63)	7 (64)	0.78
Limb						
Upper extremity	3 (30)	0 (0)	2 (50)	1 (13)	4 (36)	0.31
Lower extremity	7 (70)	6 (100)	2 (50)	7 (87)	7 (64)	
Symptoms						
Pain	10 (100)	6 (100)	4 (100)	7 (88)	6 (55)	0.02
Swelling	8 (80)	5 (83)	2 (50)	7 (88)	6 (55)	0.37
Paresthesias	**3 (30)**	**4 (67)**	**0 (0)**	**5 (63)**	**1 (9)**	**0.03**
Motor deficit	1 (10)	3 (50)	0 (0)	4 (50)	4 (36)	0.17
Perfusion	**1 (10)**	**0 (0)**	**0 (0)**	**3 (38)**	**7 (64)**	**0.01**
No. symptoms						
1	2 (20)	0 (0)	2 (50)	0 (0)	2 (18)	0.71
2	4 (40)	1 (17)	2 (50)	1 (13)	7 (64)	
3	3 (30)	4 (67)	0 (0)	4 (50)	1 (9)	
4	1 (10)	1 (17)	0 (0)	3 (38)	0 (0)	
5	0 (0)	0 (0)	0 (0)	0 (0)	1 (9)	
Pressure (mean ± SD) (mm Hg)	53 ± 17.20	87.4 ± 41.90	47 (—)	59.6 ± 18.00	67.8 ± 24.30	0.46
Time to diagnosis [median (IQR)] (h)	27 (4-90)	48 (24-108)	108 (36.5-210)	36 (24-60)	24 (10-60)	0.75
Treatment characteristics						
No. compartments (mean ± SD)	4.3 ± 3.90	2.3 ± 0.80	6 ± 6.10	2.8 ± 1.40	5.4 ± 4.50	0.55
No. surgeries (mean ± SD)	3 ± 1.60	3.8 ± 2.60	4 ± 2.20	2.2 ± 1.00	3.1 ± 2.60	0.65
Necrosis in OR	4 (40)	3 (50)	2 (50)	3 (38)	9 (82)	0.27
Outcome characteristics						
Pain (N = 36)	1 (10)	0 (0)	1 (25)	2 (25)	0 (0)	0.36
Neurological or functional deficit (N = 36)	0 (0)	2 (33)	0 (0)	4 (50)	6 (55)	0.06
Skin graft/tissue expander	3 (30)	0 (0)	3 (75)	1 (13)	1 (9)	0.24
Length of follow-up [median (IQR)]	166 (87-294.8)	95 (80.5-239.2)	445 (173.5-779.8)	413 (45.5-706.8)	606.5 (120.2-878.5)	0.53

Data are presented as frequency (%) unless stated otherwise.
Bold entries indicate significance at the 5% level.
IQR indicates interquartile range (25th percentile-75th percentile).

in diagnosis was likely multifactorial and dependent on etiology. The majority of the patients in this study were not under the orthopaedic service and in general awareness of the nonfracture-related compartment syndrome is poor. Furthermore, the presence of postoperative thoracic epidurals was another potential risk factor in the postoperative group. Although thoracic epidurals are effective for postoperative pain control, the block should allow for some voluntary ankle and foot motion. If complete paralysis is achieved, then the block should be turned down or decreased until a more normal motor function is achieved. Other strategies to prevent a missed ACS diagnosis in high-risk patients (eg, tibia, forearm fractures) include enhanced education, standardized order sets, and mandatory q2-hour documentation of neurovascular status.[18] While this strategy may help in high-risk fracture patients, it would be difficult to identify target groups at risk for NFACS.

The clinical presentation of ACS has often been described as beginning with increasing pain, pain at rest, or pain with passive stretch. In our cohort, although pain was typically present, the presence of neurological findings occurred at a higher rate than patients with typical fracture-related ACS. Previous studies of pediatric ACS have recorded paresthesias in 26%[2] and 20%[17] compared with 33% of our cohort, with higher proportions in the postoperative (63%) and exercise-related etiologic groups (67%). Furthermore, 31% of our patients demonstrated motor weakness at presentation, with an alarming 50% in the postoperative and exercise-related groups and 36% in the vascular group. It is likely that the neurological deficits can be attributed to the delay in diagnosis and irreversible nerve injury.

Untreated ACS leads to irreversible muscle damage beginning approximately 4 hours from the onset of elevated pressure.[19–21] The incidence of myonecrosis in our cohort of patients is much higher (54%) than reported in children with fracture-related ACS (4%).[17] Similarly in adults, patients without fractures have been shown to have a higher incidence of myonecrosis than those with fractures (20% vs. 8%).[22] In addition, previous literature has demonstrated a high rate (92% to 95%) of return to normal activities after pediatric ACS from lower extremity fractures.[2,17] Keeping in mind our observed high rates of neurovascular deficits on presentation and myonecrosis in the OR, it is not surprising that our patients had a lower rate of full recovery (56%).

The development of NFACS is likely a multifactorial event, with many potential areas for improvement in diagnosis and management. Important lessons from each of the 5 etiologies were identified from this analysis. In patients who developed postoperative NFACS, operative positioning may have caused 2 cases, in which bilateral leg NFACS developed after prolonged lithotomy position in muscular adolescent males; careful attention should be given to this age and body habitus group undergoing prolonged lithotomy positioning. Furthermore, 4 of 6 postoperative patients had thoracic epidurals, which provide excellent pain relief but have the potential to mask the development of NFACS. We recommend that when an epidural is in place patients should be able to demonstrate some lower extremity movement and the presence of complete paralysis should signal to the treating physician that the block should be decreased.

Patients with exercise-related NFACS were all late-adolescent males involved in competitive athletics. Perhaps, growing muscle mass and a noncompliant fascial envelope place these adolescents at risk for NFACS.[23] In our series, exercise-related cases experienced high rates of neurological symptoms on presentation with elevated compartment pressures (mean 87 mm Hg). Diagnostic delay was common within this group, with an average delay from symptom onset to diagnosis of 48 hours, and almost all patients required pressure measurement to make the diagnosis. This highlights the potential misdiagnosis of exercise-related NFACS as shin splints, chronic exertional compartment syndrome, and stress fractures.

Our study showed that most cases of compartment syndrome happened in the leg, and more specifically in the anterior and/or lateral compartments of the leg, and this finding is likely related to muscle fiber composition. Muscle groups with higher percentages of fast-twitch fibers, such as tibialis anterior and peroneus longus, are at relatively higher risk for ischemic injury. Comparatively, the higher concentration of slow-twitch fibers in posterior compartment muscles is relatively protective against ischemia.[24] This correlates with our findings of more frequent anterior and lateral releases than posterior releases for NFACS of the leg. In our cohort, the vascular group experienced the highest rate of myonecrosis, neurological and functional deficit, yet the shortest time to diagnosis. These findings underscore the importance of early reperfusion after ischemia.

There are several limitations to our study. Selection bias could have occurred as this study was retrospective and took place at a tertiary referral center. We feel, however, that this hospital represents the majority of all pediatric care seen within our state and likely is an accurate representation without significant bias. The data obtained from the medical record were recorded in a retrospective manner and is only as good as what was originally recorded, and incomplete data entry occurred. Unfortunately, due to limited prevalence of NFACS, a prospective study is impractical. Another limitation of our study is the vast heterogeneity of patients, who ranged from a newborn with a dysvascular arm after an arterial line complication to a teenager with an exercise-related compartment syndrome after playing soccer. We tried to address this issue by looking at data separately in etiologic groups; however, the etiologic groups have small numbers. Finally, it is unclear how many false positives occurred when 40% of the cohort had their diagnosis made clinically. Despite these limitations, however, this study provides important information about the prevention and management of NFACS.

In summary, there are many lessons to learn from the management of NFACS in children and adolescents. Although this event is uncommon, delay in diagnosis and

unfamiliarity with the presentation can lead to permanent long-term deficits for children and adolescents. Physicians caring for children and adolescents must be aware of this clinical entity and refer to orthopaedic surgeons promptly to diminish the serious morbidity associated with the syndrome.

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