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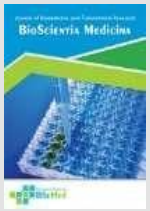

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Functional Limb Salvage Following a 24-Hour-Delayed Fasciotomy for Pediatric Hand Compartment Syndrome: A Case Report

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A B S T R A C T

Background: Acute compartment syndrome (ACS) in the pediatric hand is a formidable surgical emergency where delayed diagnosis can lead to devastating neuromuscular deficits and limb loss. The narrow therapeutic window, often termed the "golden hours," is considered critical for preventing irreversible ischemic necrosis. Presentations delayed beyond this period, especially in pre-verbal children, pose a significant clinical and ethical dilemma regarding the utility and risks of surgical intervention. **Case presentation:** A 2-year-old female presented to our emergency department 24 hours after a severe crush injury to her right hand from a noodle-making machine. The hand was massively swollen, cyanotic, and insensate, with no detectable capillary refill or digital oximetry readings. A clinical diagnosis of advanced, multi-compartmental ACS was made. Emergency surgical decompression was performed via seven incisions, releasing all ten osteofascial compartments. Intraoperative assessment revealed dusky, non-contractile but bleeding muscle. Following staged debridements, the patient demonstrated remarkable recovery. At six months, she achieved full range of motion in all digits except the middle finger and demonstrated age-appropriate grip and pincer grasp, with the primary sequela being a fixed flexion contracture of the middle finger's proximal interphalangeal joint. **Conclusion:** This report highlights a case of unexpected functional recovery following a significantly delayed fasciotomy. The outcome supports a nuanced approach to delayed pediatric ACS, suggesting that in select cases, aggressive surgical decompression should be considered as the potential for a positive outcome may exist. This case serves as a powerful, hypothesis-generating observation that raises critical questions about the absolute temporal limits for surgical intervention and underscores the importance of individualized surgical judgment.

1. Introduction

Acute compartment syndrome (ACS) is a time-sensitive surgical emergency defined by a critical increase in pressure within a fixed osteofascial space, leading to compromised tissue perfusion and profound cellular hypoxia.[1] The ensuing pathophysiological cascade, if not promptly arrested, progresses relentlessly from reversible ischemia to irreversible myoneural necrosis, culminating in devastating outcomes such as Volkmann's ischemic contracture, chronic neuropathic pain, permanent functional loss, and potentially, limb amputation.[2]

The management of ACS is governed by a foundational principle of modern trauma surgery: the time-dependency of ischemic injury.[3] Skeletal muscle and peripheral nerves, the primary occupants of these compartments, can only tolerate a finite period of warm ischemia—classically cited as four to six hours—before the onset of permanent cellular death.[4] This narrow therapeutic window has given rise to the concept of the "golden hours," a critical period during which emergent surgical decompression via fasciotomy is believed to be most effective at averting catastrophic, long-term sequelae.[5] The hand, with its intricate architecture comprising ten distinct, small-volume compartments (four dorsal interossei, four palmar interossei, thenar, and hypothenar), is particularly susceptible to rapid and devastating pressure elevations. Crush injuries, such as the one central to this report, represent a particularly virulent etiology for hand ACS. These injuries inflict a double insult: an initial, direct compressive force causing immediate myocellular damage and microvascular hemorrhage, followed by a post-extrication phase of reactive hyperemia and massive edema formation that rapidly overwhelms the compartment's limited capacity for expansion.[6]

Diagnosing ACS in the pediatric population, especially in pre-verbal toddlers, presents a well-documented and formidable clinical challenge. The classic pentad of signs—the "5 Ps" (Pain out of proportion, Paresthesia, Pallor, Paralysis, and Pulselessness)—has proven to be of limited utility as an early diagnostic tool in this demographic. A young child cannot verbalize the subtle tingling of paresthesia, and their generalized distress makes the critical sign of "pain out of proportion" difficult to reliably discern from the expected pain of the initial injury.[7] Furthermore, the latter signs of pallor, paralysis, and pulselessness are not early warnings but rather ominous harbingers of advanced, and likely irreversible, tissue necrosis. This has led to a clinical shift towards recognizing more subtle behavioral cues—the "3 A's" of increasing Analgesic requirement, escalating Anxiety, and Agitation—as more sensitive indicators of evolving ischemia in non-communicative children.

The intersection of these diagnostic difficulties with the unforgiving timeline of ischemia makes delayed presentations particularly perilous. The decision to perform a fasciotomy in a patient who presents 24 hours after the inciting injury is fraught with clinical controversy, a debate that is not binary but nuanced. Some authorities argue that after such a prolonged ischemic duration, the potential benefits of reperfusion are outweighed by the substantial risks, which include releasing a toxic tide of metabolic byproducts (potassium, myoglobin) from necrotic muscle into systemic circulation, potentially causing life-threatening hyperkalemia and acute renal failure.[8] Furthermore, decompressing compartments filled with non-viable tissue may create a contaminated wound, increasing the risk of deep infection and subsequent amputation. Conversely, other viewpoints maintain that any potential for tissue salvage, however small, justifies the surgical attempt, particularly in a child's hand where function is so critical. This controversy is amplified by a significant paucity of high-quality evidence to guide decision-making.[9] While case reports exist for delayed fasciotomy in the pediatric lower extremity, a comprehensive review of the literature reveals a near-complete absence of reported outcomes for crush-induced ACS of the hand in the pre-school age group when the delay to decompression exceeds 24 hours.[10]

This manuscript presents a detailed account of a case that resides at the very fulcrum of this clinical and ethical debate. It chronicles a successful functional outcome in a scenario that conventional wisdom dictates should have resulted in catastrophic limb loss, thereby serving as a critical, hypothesis-generating data point. The profound novelty of this report is its direct challenge to the perceived absolute limits of ischemic tolerance in pediatric tissues, compelling the surgical community to question whether the established "golden hours" paradigm applies with uniform rigidity across all ages and anatomical locations. The aim of this study, therefore,

is to provide a rigorous, objective, and multi-faceted documentation of this rare case of functional limb salvage. Through a detailed clinical narrative and a balanced, deeply analytical discussion, we seek not to establish a new standard of care from a single observation, but to contribute to a more nuanced understanding of the true therapeutic window for fasciotomy, to champion the primacy of individualized surgical judgment over temporal dogma, and to underscore the urgent need for further research into the unique physiological responses to ischemia in the very young.

2. Case Presentation

A 2-year-old female with an unremarkable past medical history was brought to our tertiary care emergency department by her parents with a chief complaint of a severely injured and discolored right hand. The injury had occurred approximately 24 hours prior, when the child's hand was caught and compressed in a household electric noodle-making machine. The parents extricated the hand immediately but did not seek medical attention until the following day. During the intervening period, they observed a rapidly progressive and massive swelling of the entire hand, a deepening dark discoloration, and noted the child was in severe, unremitting pain, crying inconsolably. The clinical history is summarized in Figure 1. Figure 1 provides a comprehensive overview of the critical events preceding surgical intervention for a 2-year-old female patient diagnosed with acute compartment syndrome of the right hand. The timeline is anchored by the initial traumatic event (T=0 hours), identified as a severe crush injury from an electric noodle-making machine. This high-energy mechanism initiated a cascade of soft tissue damage, setting the stage for the subsequent pathophysiological developments. A crucial and defining element of this case, highlighted in the timeline, is the significant 24-hour ischemic period that elapsed between the injury and the patient's presentation to the emergency department. This delay represents a substantial deviation from the standard golden hours protocol for managing compartment syndrome, a period during which the patient experienced a progressive and alarming constellation of symptoms. These included severe and unremitting pain, indicative of profound tissue distress; rapidly evolving edema, reflecting significant fluid extravasation and venous congestion; and a deep, violaceous discoloration of the hand, a visible marker of severe deoxygenation and impending tissue necrosis. Upon presentation at T=24 hours, the patient's clinical status had deteriorated to a state of advanced limb compromise. The diagnosis of multi-compartmental acute compartment syndrome was made based on definitive clinical findings of profound ischemia, characterized by the absence of capillary refill and detectable pulse oximetry signals in the digits. Furthermore, a complete neurological deficit, manifesting as both anesthesia (lack of sensation) and paralysis (inability to actively move the digits), confirmed the severity of the myoneural ischemia. Figure 1 effectively encapsulates the temporal and clinical context of the case, underscoring the gravity of the 24-hour delay and the critical condition of the limb at the time of initial medical assessment, thereby framing the subsequent successful surgical intervention as a remarkable and noteworthy outcome.

Upon initial evaluation, the patient was afebrile and hemodynamically stable. She was visibly distressed, agitated, and highly protective of the injured extremity. The clinical examination of the right hand was alarming, revealing findings consistent with an advanced and severe multi-compartmental ACS. The orthopedic and surgical teams were immediately consulted for emergent intervention. The detailed physical examination findings are presented in Figure 2. Figure 2 provides a detailed schematic and visual summary of the critical physical examination findings upon the patient's arrival, which collectively formed the basis for the unequivocal diagnosis of advanced multi-compartmental acute compartment syndrome.

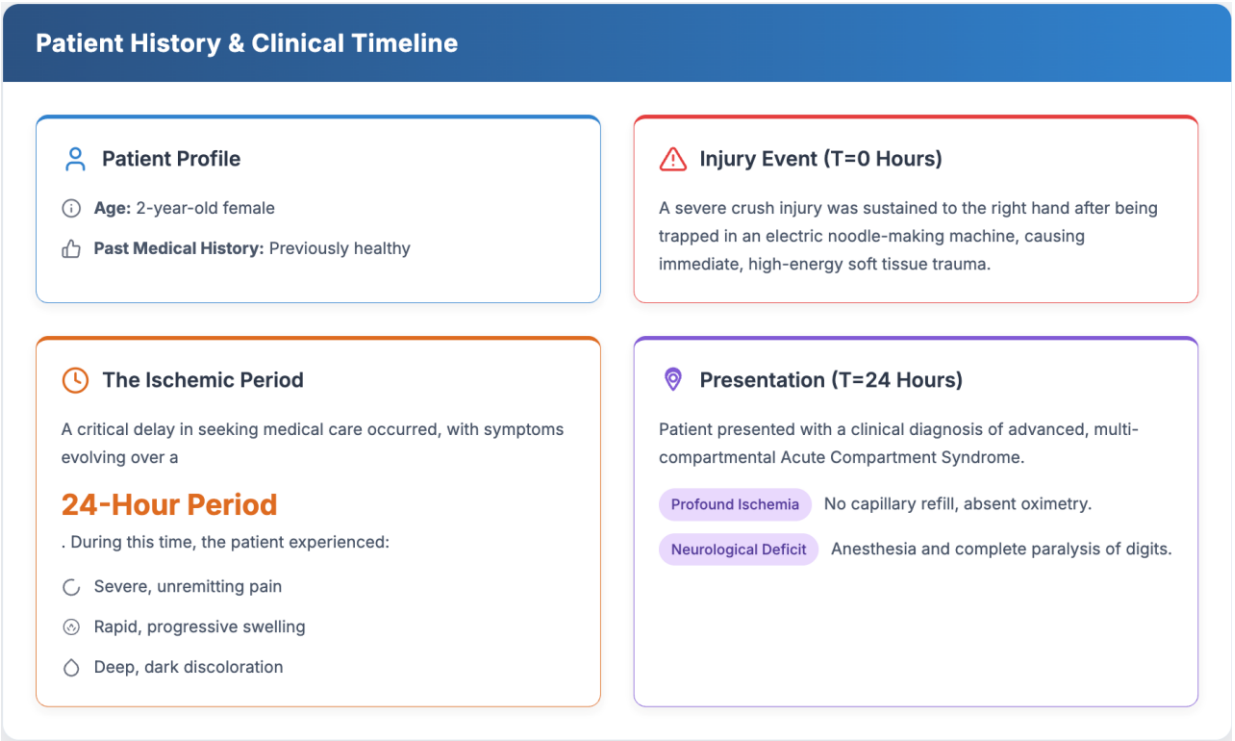

Figure 1. Patient history and clinical timeline.

The clinical photograph at the center of the figure offers a stark visual testament to the severity of the limb's condition after a 24-hour ischemic period, characterized by massive swelling and a deep, cyanotic discoloration indicative of impending necrosis. The assessment was systematically organized into four key domains. Inspection revealed a grossly edematous hand with tense, uniform swelling that obliterated the normal anatomical contours from the wrist to the fingertips. The deep violaceous and ecchymotic discoloration was a pathognomonic sign of profound venous congestion and stagnant, deoxygenated blood flow. Furthermore, the digits were held in a fixed, semi-flexed, and immobile posture, a classic sign of critically elevated pressure within the intrinsic muscle compartments. Palpation confirmed the visual findings with tactile evidence of extreme pathology. The entire hand felt "woody" and unyieldingly tense, a tactile sign of intracompartmental pressures far exceeding the capillary perfusion pressure. Critically, any attempt at passive extension of the digits elicited extreme pain, a cardinal and highly specific indicator of muscle compartment ischemia. The hand was also palpably cooler than the contralateral limb, signifying a severe compromise of arterial inflow. The Vascular and Neurological Assessments documented a complete functional collapse of the distal limb. There was a total absence of capillary refill in all five digits, and no pulsatile signal could be obtained with pulse oximetry, indicating a failure of microvascular perfusion. This was further confirmed by the silence on Doppler ultrasound, signifying a distal vascular standstill. Neurologically, the hand was completely insensate, with no withdrawal response to pinprick stimuli (anesthesia), and exhibited a total lack of active motor function (paralysis). This profound neurological deficit confirmed severe, established myoneural ischemia. In synthesis, these findings represent the ominous, late-stage signs of compartment syndrome, illustrating a limb on the brink of biological non-viability and underscoring the urgency of the surgical intervention that followed.

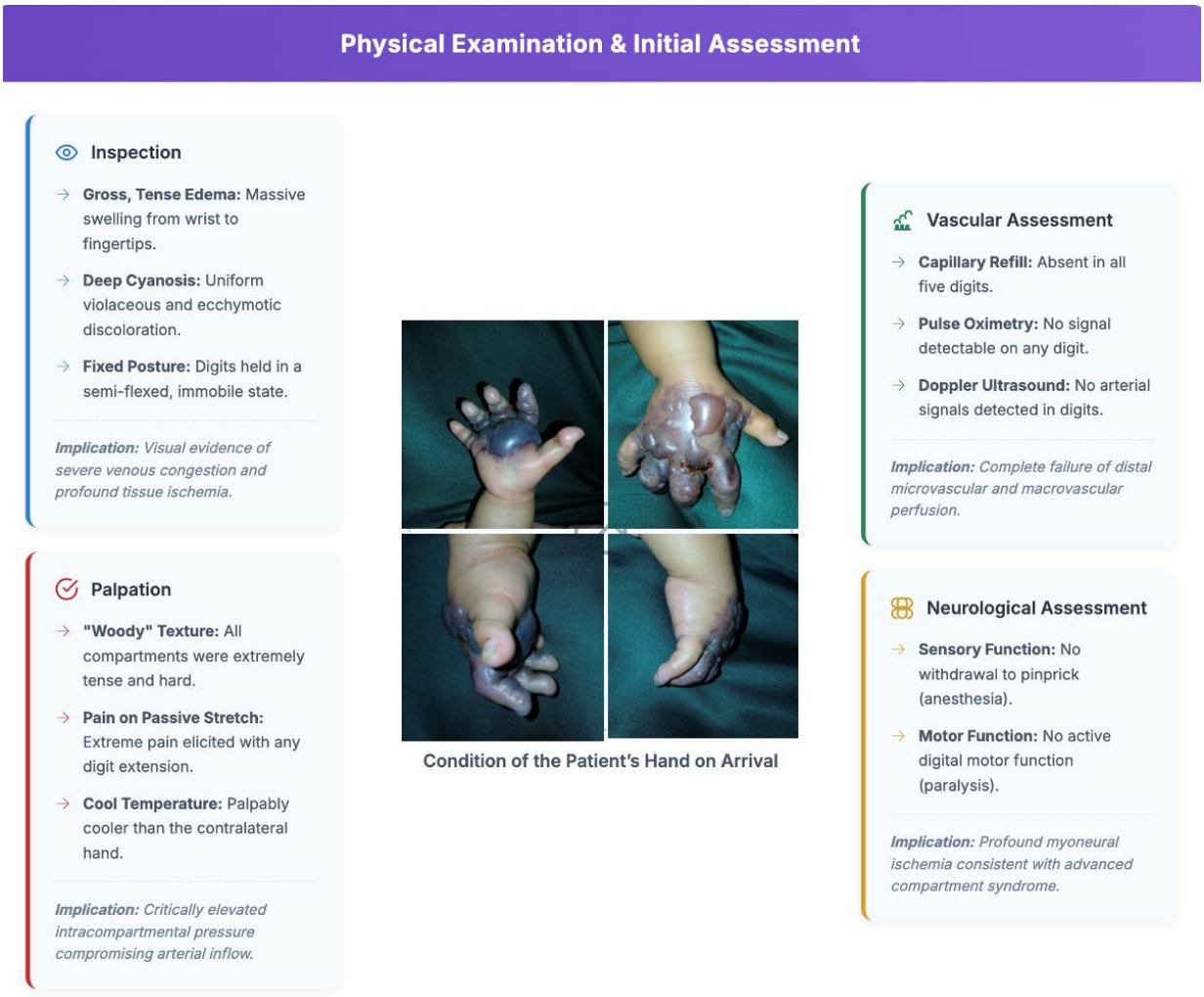

Figure 2. Physical examination & initial assessment.

Anteroposterior and oblique radiographs of the hand were obtained to assess for concomitant bony injury. As detailed in Figure 3, the imaging confirmed the absence of fractures but provided a stark visual correlate for the clinical examination. No compartment pressure measurements were taken, as the unequivocal clinical signs of advanced ischemia necessitated emergent surgical intervention without delay. Figure 3 illustrates the critical, albeit limited, role of radiographic imaging in the diagnostic pathway of this patient's acute compartment syndrome. The anteroposterior and oblique plain radiographs of the right hand, presented in the figure, were a crucial initial step in the trauma workup. The primary finding from this imaging was the conspicuous absence of any bony injury. There were no visible fracture lines, dislocations, or other osseous abnormalities that could have been the primary cause of the severe swelling or acted as a confounding variable in the clinical picture. However, the radiographs provided a powerful secondary confirmation of the clinical assessment. The images clearly demonstrate marked and diffuse soft tissue swelling, which is visually apparent by the expanded intermetacarpal spaces and the general haziness that obscures the normal, crisp fascial planes. This radiographic sign, while non-specific, directly corroborated the extreme edema observed during the physical examination. The

diagnostic conclusion, therefore, was not derived from the radiographs themselves but from their interpretation within the broader clinical context. The absence of a fracture served to isolate the etiology of the condition to a pure soft-tissue, crush-induced vascular, and compartmental catastrophe. This finding streamlined the diagnostic process, eliminating the need to consider fracture reduction or stabilization as part of the immediate surgical plan. Ultimately, the radiographic workup played a vital, supportive role: by ruling out bony injury, it reinforced the overwhelming clinical evidence that pointed towards advanced acute compartment syndrome as the sole diagnosis, confirming that the only appropriate and urgent course of action was immediate surgical decompression without the need for further, time-consuming diagnostic modalities like compartment pressure monitoring or advanced imaging.

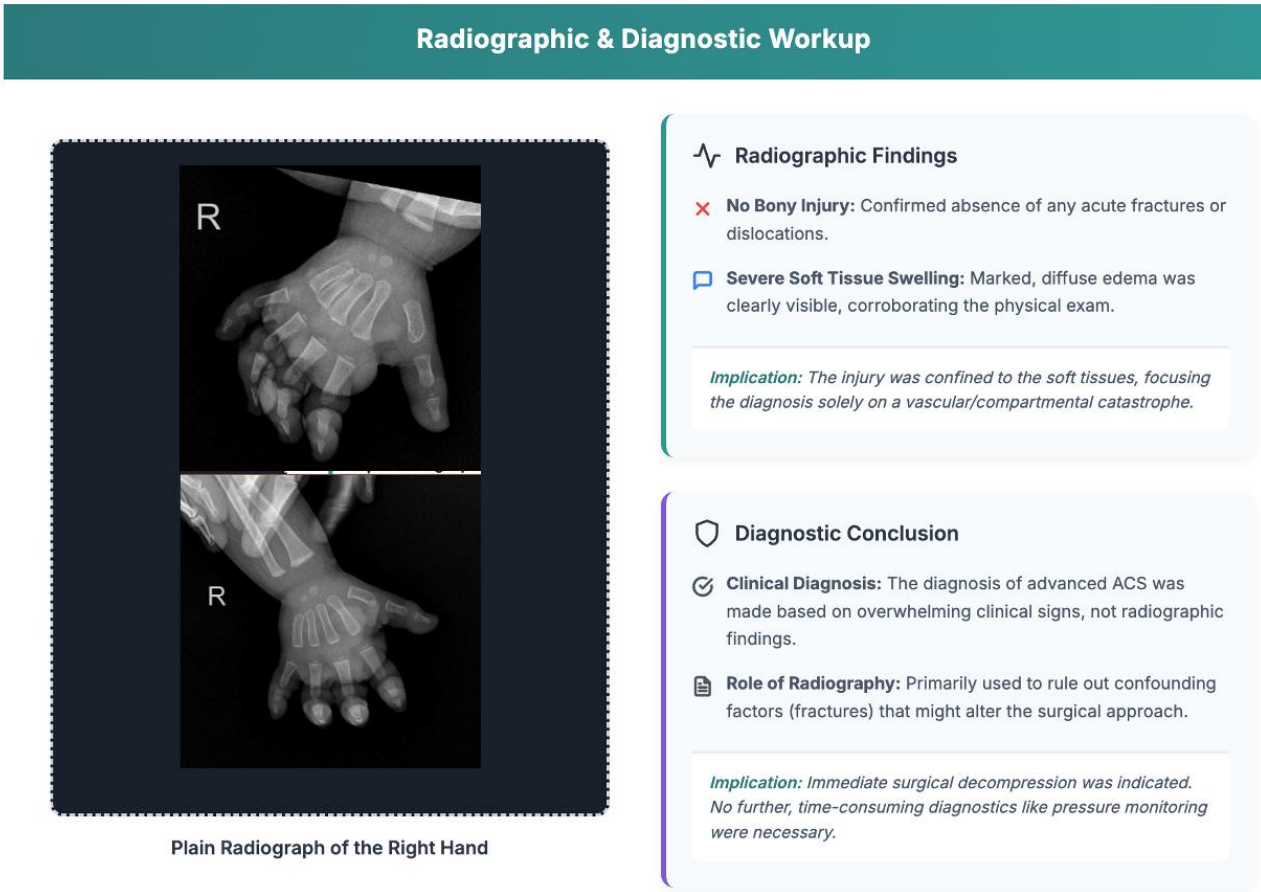

Figure 3. Radiographic & diagnostic workup.

The patient was taken emergently to the operating room for surgical decompression. A comprehensive seven-incision fasciotomy was performed to ensure the complete release of all ten hand compartments. Upon incising the fascia, the intracompartmental muscle was observed to be under extreme pressure, bulging immediately from the wounds. A meticulous intraoperative assessment of tissue viability was performed, the results of which are summarized in Figure 4. Figure 4 provides a detailed summary of the

definitive surgical intervention and the critical intraoperative findings that confirmed the diagnosis of advanced acute compartment syndrome. The central component of the figure is the clinical photograph, which captures the state of the hand immediately following the extensive fasciotomy, revealing the underlying ischemic muscle tissue. The surgical approach was a comprehensive 7-incision fasciotomy, a strategy designed to ensure the complete and immediate decompression of all ten osteofascial compartments within the hand. This included two dorsal incisions to release the eight interossei compartments, dedicated palmar incisions for the thenar and hypothenar eminences, and multiple midlateral incisions along the digits to address the profound distal ischemia. This aggressive and anatomically thorough approach was deemed essential to maximize the potential for tissue salvage. The intraoperative tissue assessment provided a stark, real-time confirmation of the pathophysiology that had been unfolding over the preceding 24 hours. Upon incision of the overlying fascia, the underlying muscle tissue was observed to bulge out explosively, a physical manifestation of the critically elevated intracompartmental pressures being released. The muscle itself exhibited a dusky, grayish, and grossly edematous appearance, lacking the healthy, contractile, reddish-pink hue of viable tissue. Furthermore, there was a complete absence of muscle contractility in response to direct stimulation, a definitive sign of profound and prolonged myoneural ischemia. These direct visual and functional findings were unequivocal. They provided the definitive confirmation that the patient was suffering from a severe, established compartment syndrome, validating the aggressive surgical approach and underscoring the severity of the ischemic insult the limb had endured.

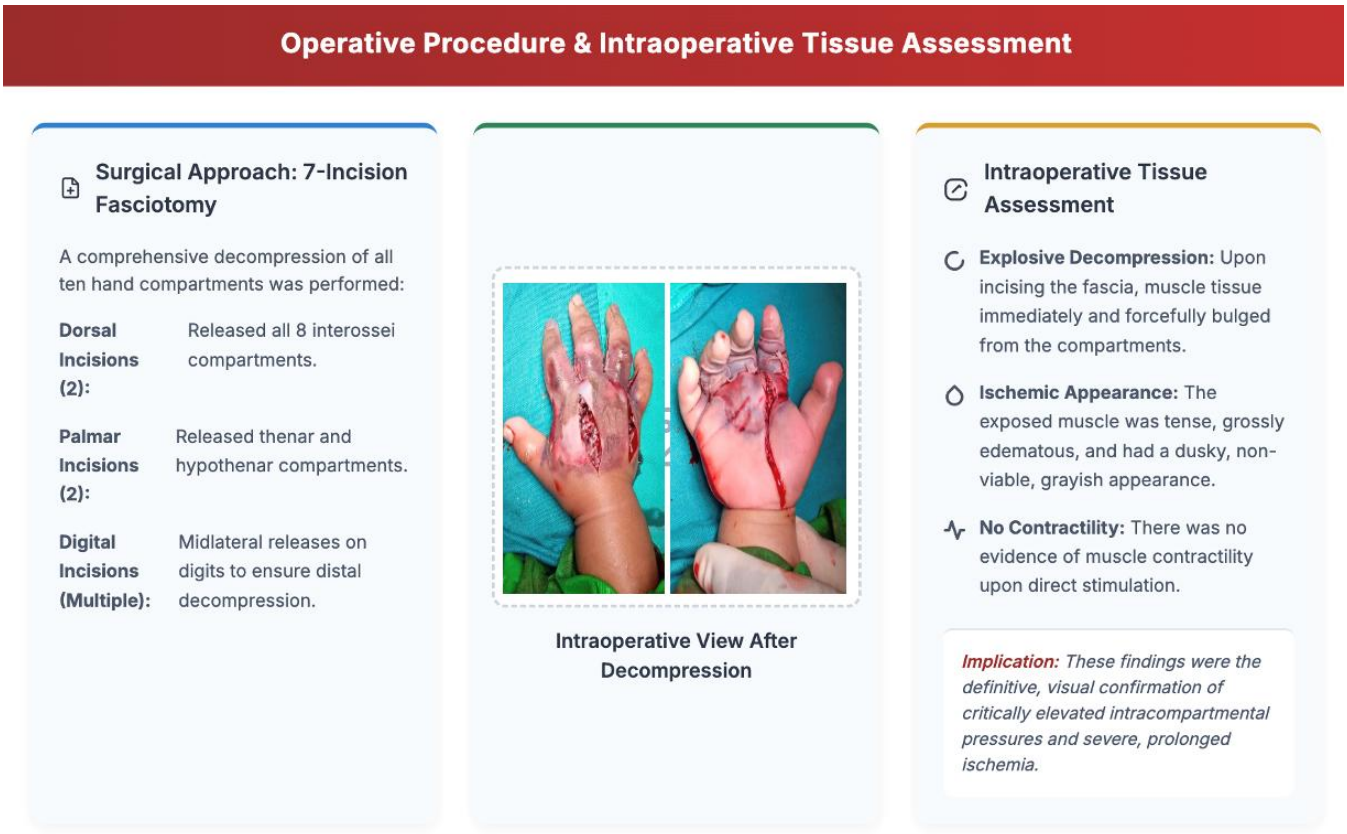

Figure 4. Operative procedure & intraoperative tissue assessment.

Figure 5 provides a comprehensive summary of the patient's clinical journey following the initial surgical decompression, charting the meticulous postoperative management and culminating in the final, highly favorable functional outcome at the six-month follow-up. The schematic is bifurcated to distinctly present the temporal sequence of care and the ultimate clinical result. The left panel illustrates the structured, staged approach to postoperative management. The timeline begins on Day 0 with the critical, limb-saving 7-incision fasciotomy, after which the wounds were strategically left open to accommodate residual swelling and allow for drainage. A pivotal moment in the patient's care occurred on Day 4, during a planned "second look" surgery. This procedure was essential for reassessing tissue viability after a period of reperfusion. While four of the five digits showed promising signs of recovery, the middle finger remained dusky, indicating persistent ischemic stress and raising concerns about its ultimate viability. A third surgical procedure on Day 7 marked a significant positive turning point: all five digits were now deemed viable, and the wounds exhibited healthy granulation tissue, signaling the beginning of a robust healing response. This allowed for a transition to open wound management, with the patient being stable for discharge on Day 8 to continue healing on an outpatient basis. The right panel showcases the remarkable success of this management strategy, detailing the final outcome after six months. The clinical photograph displays a well-healed hand with pliable scars and a near-normal contour. Functionally, the recovery was excellent, with the patient regaining full, pain-free use of the hand for all age-appropriate activities. The range of motion was restored to normal in the wrist and all digits, with the notable exception of the middle finger. The sole sequela was a mild, fixed flexion contracture at the proximal interphalangeal joint of this digit—a localized, residual sign of the severe ischemic insult it uniquely endured. This minor contracture, a form of focal Volkmann's contracture, stands as the only remnant of a catastrophic injury, underscoring an otherwise extraordinary functional salvage.

Figure 5. Postoperative course & final outcome.

3. Discussion

The successful functional salvage of this patient's hand, following a 24-hour period of profound, crush-induced ischemia, represents a profound clinical event that compels a re-examination of the established principles governing the management of acute compartment syndrome, particularly within the pediatric population.[11] This case transcends a simple report of a favorable outcome; it serves as a biological testament that challenges the rigid temporal boundaries often cited as the absolute limit for viable surgical intervention. A comprehensive discussion of this case requires a deep, multilayered analysis, dissecting the intricate pathophysiology of prolonged ischemia, the unique diagnostic hurdles in pre-verbal children, the controversial yet validated role of delayed surgical decompression, and the constellation of factors that converged to produce such an unexpectedly positive result. To truly appreciate the magnitude of the recovery, one must first understand the depth of the physiological devastation that a 24-hour delay imposes upon neuromuscular tissue. The pathophysiology of ACS is a famously vicious cycle, but its protraction over a full day amplifies each destructive component exponentially.[12] The initial crush injury from the noodle-making machine delivered a massive energy transfer, causing instantaneous myocellular rupture, microvascular tearing, and immediate intracompartmental hemorrhage. This event alone initiated the spike in pressure within the hand's ten small, unforgiving osteofascial compartments. As the pressure began to climb, it sequentially surpassed critical thresholds. First, the low pressure of the lymphatic and venous systems was overcome, leading to an immediate cessation of outflow. This created a state of intense venous congestion, trapping deoxygenated blood and metabolic waste products within the compartments. The resulting backpressure further elevated the interstitial fluid pressure, which in turn began to compress the arterial capillaries.

The critical turning point in ACS occurs when the intracompartmental pressure exceeds the diastolic pressure, effectively collapsing the arterial-venous pressure gradient required for perfusion.[13] At this stage, blood flow to the capillary beds ceases, and the true ischemic phase begins. For the patient in this report, this ischemic state was not a transient event but a prolonged, 24-hour siege. At the cellular level, this siege wrought havoc. The lack of oxygen forced the myocytes and neurons to switch from efficient aerobic metabolism to desperate, inefficient anaerobic glycolysis. This produced a pittance of ATP while generating large quantities of lactic acid, causing a rapid drop in intracellular pH. The lack of ATP is catastrophic for the cell. It leads to the failure of crucial ion pumps, most notably the Na+/K+-ATPase pump.[14] Without this pump, sodium floods into the cell, bringing water with it via osmosis, leading to massive cellular swelling (hydropic change). Simultaneously, calcium pumps fail, allowing a massive influx of extracellular calcium, which acts as a potent second messenger for cellular death pathways. This intracellular calcium activates a host of destructive enzymes, including proteases that dismantle the cytoskeleton, phospholipases that degrade cell membranes, and endonucleases that fragment DNA.

Over 24 hours, this process moves beyond reversible injury. Mitochondria, the cellular powerhouses, swell and develop dense inclusions, their membranes lose integrity, and they release cytochrome c, a potent trigger for apoptosis, or programmed cell death. Cell membranes, attacked by phospholipases and reactive oxygen species generated in the low-oxygen environment, undergo lipid peroxidation, losing their structural integrity and leading to cell lysis and necrosis. The skeletal muscle fibers, which are highly metabolically active, are the first to succumb. It is widely taught that irreversible necrosis begins after just 4 to 6 hours. After 24 hours, one would theoretically expect to find a compartment filled with a necrotic, liquefied mass of dead muscle.[15] Peripheral nerves, the other primary residents of these compartments, are similarly vulnerable. The ischemia first affects their conductive properties, leading to the

early sign of paresthesia, followed by anesthesia. Prolonged ischemia targets the Schwann cells and the axon itself, leading to Wallerian degeneration, a process of nerve fiber disintegration that can be permanent. It is this well-established, brutal timeline of cellular death that makes the outcome in this case so astonishing. The clinical presentation of a cold, cyanotic, anesthetic, and paralyzed hand was the macroscopic manifestation of this microscopic apocalypse.[16] The prevailing expectation would be that the tissues had fallen too far into the pathophysiological abyss to be rescued.

The decision to perform a fasciotomy 24 hours after the onset of ischemia is one of the most contentious in trauma surgery. The conventional wisdom argues that after 6-8 hours, the ischemic damage is irreversible, and opening the compartments will only expose necrotic, non-viable muscle to the environment, creating a high risk of deep infection, which could lead to systemic sepsis and ultimately necessitate an amputation at a higher level. Furthermore, there is the significant and legitimate fear of inducing a severe systemic reperfusion injury. When blood flow is restored to a large mass of necrotic tissue, a toxic cocktail of cellular breakdown products—including potassium, myoglobin, and acidic compounds—is washed into the systemic circulation.[17] This can cause life-threatening hyperkalemia leading to cardiac arrhythmias, and myoglobin can precipitate in the renal tubules, causing acute kidney injury and renal failure. Given these substantial risks, the decision to proceed with surgery in this 2-year-old was a calculated surgical audacity, predicated on a single, crucial belief: that within the devastated landscape of the child's hand, there might still exist islands of salvageable tissue. It was a decision that prioritized the chance of limb salvage over the conservative approach of expectant management or primary amputation. This decision finds support in a growing but still limited body of literature that suggests delayed fasciotomy can be beneficial. A systematic review by Zhao et al. (2020) acknowledged the higher complication rates but confirmed that limb salvage was still achieved in a significant number of patients who underwent delayed decompression. The key determinant of success appears to be the presence of some residual viable tissue, a factor that is impossible to determine with certainty preoperatively.[18]

The surgical strategy itself was as critical as the decision to operate. A timid or incomplete fasciotomy would have been doomed to failure. The hand is an anatomical puzzle box of ten compartments, and pressure in any one of them can compromise the entire structure. The surgeon's use of a seven-incision technique was a testament to a thorough understanding of this complex anatomy. The two dorsal incisions are the workhorses, allowing for the release of all four dorsal interossei and, by dissecting deeper, all four palmar interossei. This single approach effectively decompresses eight of the ten compartments. However, the thenar and hypothenar compartments must be addressed by separate, dedicated incisions over their respective eminences. Critically, in this case, the surgeon also recognized the profound distal ischemia and proceeded with midlateral incisions on the digits. This final step was likely essential in salvaging the fingers, as it released the pressure within the tight digital sheaths. The intraoperative finding of "tense, edematous, and dusky-looking muscle" that bulged from the incisions is the classic visual confirmation of ACS. The dusky color reflects the severe deoxygenation and venous stasis. The immediate bulging is a physical demonstration of the immense pressure being released.[19] The subsequent staged management, with a planned "second look" operation, was equally vital. This approach allows the surgeon to reassess the tissue after a period of reperfusion. It is in this period that the true extent of necrosis declares itself. The initial postoperative assessment was a mixed but hopeful signal: four digits showed signs of life, while the middle finger remained precarious. The decision to wait and perform another debridement three days later, rather than proceeding with an immediate amputation of the middle finger, was another crucial judgment call. It allowed the tissues the maximal

chance to recover, and this patience was rewarded with the eventual return of perfusion to all five digits.

The most parsimonious explanation may be that the ischemic insult was not as absolute or uniform as the dramatic clinical picture suggested. ACS is defined by pressure exceeding capillary perfusion pressure. It is conceivable that while the intracompartmental pressure was critically high, it did not remain above the patient's diastolic blood pressure in all ten compartments for the entire 24-hour duration. There may have been periods of intermittent, minimal, but biologically significant perfusion. The preservation of faint wrist pulses supports this notion of incomplete macrovascular occlusion. The crush mechanism itself may have been heterogeneous, with some compartments compressed to necrotizing pressures while others were subjected to severe but ultimately reversible ischemia. Therefore, the outcome may not represent a defiance of established ischemic time limits, but rather a successful intervention in a case of severe, but sub-critical and spatially variable, ischemia. Borrowing from stroke neurology, the concept of an ischemic penumbra is highly applicable here. It is biologically improbable that all muscle fibers within the hand progressed to necrosis simultaneously. It is more likely that the injury created a central core of infarcted, non-salvageable tissue, surrounded by a much larger penumbra of severely ischemic but still viable tissue. The surgical intervention was therefore not an attempt to resurrect dead tissue, but a time-critical mission to reperfuse and salvage this penumbra. The final sequela—a contracture isolated to the middle finger—may represent the clinical footprint of this necrotic core, while the excellent function of the remaining digits represents the successful salvage of the penumbra. Children possess a remarkable capacity for regeneration and healing that is lost in adulthood. Their tissues are rich in stem and progenitor cells, including satellite cells in muscle, which are capable of regenerating damaged muscle fibers. It is plausible that a higher density of these cells, governed by more robust signaling pathways like the Notch pathway, allowed for more effective repair following the reperfusion injury. The pediatric vascular system is also more robust and adaptable. Children have a greater capacity for angiogenesis—the formation of new blood vessels. Following the decompression, their endothelial cells may have been able to mount a more vigorous angiogenic response, re-establishing microvascular networks more effectively than an adult's would. This is likely mediated by a more vigorous upregulation of key signaling molecules such as Hypoxia-Inducible Factor 1-alpha (HIF-1α) and its downstream target, Vascular Endothelial Growth Factor (VEGF). Their metabolic rate is also different. While typically higher overall, it is possible that under ischemic stress, pediatric myocytes can enter a state of metabolic hibernation more effectively, preserving cellular integrity for longer periods.

Figure 6 provides a schematic and conceptual framework for understanding the relentless and self-amplifying pathophysiological cascade of acute compartment syndrome (ACS), a process central to the clinical drama that unfolded in the patient presented. This diagram is not merely a sequence of events but a depiction of a devastating biological feedback loop, often termed the "vicious cycle," which, once initiated, proceeds with catastrophic momentum toward tissue death. Each step sequentially triggers and exacerbates the next, creating an urgent, time-dependent surgical emergency where the final outcome is directly proportional to the duration of the cycle's uninterrupted progression. The schematic serves to visually deconstruct this complex process, beginning with the inciting traumatic event and culminating in the final common pathway of irreversible myoneural necrosis. The cascade begins with the Initial Trauma, the unequivocal starting point of the pathology. In this case, a high-energy crush injury from an electric noodle-making machine delivered a massive, instantaneous force to the intricate structures of the hand. This event is far more complex than a simple contusion; it initiates a triad of primary insults. First, there is direct myocellular rupture and damage to the delicate microvasculature.

Figure 6. The pathophysiological cascade of acute compartment syndrome.

Second, this vascular disruption leads to immediate hemorrhage within the confined anatomical spaces of the hand. Third, the body's innate inflammatory response is triggered, flooding the area with mediators that increase capillary permeability, leading to the rapid extravasation of protein-rich plasma into the interstitial space. This combination of blood and edema fluid represents the initial volume increase that serves as the catalyst for the entire ensuing cascade. This initial volume expansion directly leads to the second stage: Increased Pressure. The hand is a complex anatomical unit containing ten separate osteofascial compartments, each with a very small and relatively

fixed volume. Unlike the soft tissues of the abdomen, these compartments, defined by bone and inelastic fascia, cannot expand to accommodate significant swelling. Consequently, the influx of fluid from the initial trauma causes a rapid and dramatic rise in intracompartmental pressure. This is the central physical event of the syndrome; all subsequent pathology is a direct consequence of this escalating pressure within a closed, unyielding space. As the pressure climbs, it sequentially overcomes the closing pressures of the different vascular structures within the compartment, initiating the third step, Venous Obstruction. The low-pressure venous and lymphatic systems are the first to be compromised. As the intracompartmental pressure exceeds venous pressure, the thin-walled veins collapse, effectively damming the outflow of deoxygenated blood and metabolic waste products from the tissue. This creates a state of profound venous congestion, which has two devastating effects. First, it causes a "back-pressure" that further elevates the hydrostatic pressure within the capillaries, forcing even more fluid into the interstitial space and thereby exacerbating the edema. This represents the first turn of the vicious cycle. Second, the stagnation of blood prevents the clearance of cellular waste, leading to a toxic local environment. The unrelenting rise in pressure, now amplified by venous obstruction, leads to the fourth and most critical stage: Arterial Compression. The fundamental principle of tissue perfusion is the maintenance of an arteriovenous pressure gradient, which drives blood flow through the capillary beds. As the intracompartmental pressure continues to rise, it eventually approaches and then exceeds the diastolic, and finally the systolic, pressure within the arterial capillaries. At this catastrophic turning point, the pressure gradient is obliterated. The capillaries are physically squeezed shut, and arterial inflow into the muscle and nerve tissue ceases. The lifeline of oxygenated blood has been severed, and the tissue is now plunged into a state of profound ischemia. This vascular compromise immediately translates into the fifth stage, Tissue Ischemia. The highly metabolic myoneural tissues are exquisitely sensitive to oxygen deprivation. Within minutes, cellular metabolism is forced to switch from efficient aerobic pathways to desperate, inefficient anaerobic glycolysis. This generates a pittance of the required ATP while producing large quantities of lactic acid, causing a rapid fall in intracellular pH. The lack of ATP is the death knell for the cell; it leads to the failure of critical ion pumps, most notably the Na+/K+-ATPase. Without this pump, sodium floods into the cell, drawing water with it via osmosis and causing massive cellular swelling, or cytotoxic edema. Furthermore, the ischemic cell membranes become increasingly permeable, releasing inflammatory mediators and leaking fluid. This is the second, cruel turn of the vicious cycle: the very process of ischemic injury causes more swelling, which in turn raises the compartment pressure even higher, thus intensifying the ischemia. If this cycle is not surgically interrupted, it inevitably progresses to the sixth and final stage: Irreversible Necrosis. After a period of approximately four to six hours of warm ischemia, the cellular damage transitions from reversible to permanent. The toxic influx of calcium, a result of failed membrane pumps, activates a host of destructive intracellular enzymes that dismantle the cell from within. Mitochondria swell and rupture, releasing factors that trigger apoptosis (programmed cell death). Cell membranes, attacked by phospholipases and reactive oxygen species, undergo lipid peroxidation and lose their integrity, leading to cell lysis (necrosis). In the context of our patient, a 24-hour delay meant that these tissues were subjected to this ischemic siege for four times the duration typically associated with irreversible damage. The macroscopic clinical findings of a cold, cyanotic, anesthetic, and paralyzed hand were the grim, visible manifestations of this widespread microscopic death. This final stage represents the culmination of the pathophysiological cascade—a limb that is no longer viable and is destined for either permanent, severe functional loss or amputation. The only intervention capable of breaking this relentless cycle is an emergency

fasciotomy, which physically releases the pressure, restores perfusion, and offers the only hope of salvaging the ischemic tissue before it succumbs to irreversible necrosis.

The final outcome is a beautiful illustration of these principles. The near-perfect recovery of four of the five digits demonstrates that the bulk of the intrinsic muscles and nerves supplying them survived the ischemic siege. The isolated flexion contracture of the middle finger is not a failure of the surgery but rather the indelible scar of the injury's severity.[20] This digit was clearly the most affected, as noted in the second-look operation. It likely sustained a degree of irreversible necrosis of its intrinsic muscles. In the healing phase, this necrotic muscle would have been replaced not by contractile muscle tissue, but by fibrous scar tissue. As this scar tissue matured, it would have contracted, pulling the finger into the classic posture of a Volkmann's ischemic contracture. The fact that this significant pathology was confined to a single digit, leaving the rest of the hand with excellent, pain-free, and functional capacity, is nothing short of a spectacular success. It represents the salvage of a functional limb from a situation that could have easily ended in a devastating amputation.

4. Conclusion

This case report chronicles a remarkable instance of successful functional recovery in a toddler following a 24-hour-delayed fasciotomy for a severe, crush-induced hand compartment syndrome. The outcome achieved stands in stark opposition to the grim prognosis typically associated with such a prolonged ischemic insult, offering a powerful message of hope and challenging the rigid temporal dogmas of emergency limb surgery. This case serves as a powerful, hypothesis-generating observation that the unique physiological environment of a young child may confer an enhanced resilience to ischemia, potentially widening the window for viable surgical intervention. While a single case cannot redefine a standard of care, it unequivocally demonstrates that a favorable outcome is possible well beyond the "golden hours." It champions a clinical philosophy where individualized surgical judgment, informed by a deep understanding of pathophysiology and a willingness to challenge conventional wisdom, takes precedence over a nihilistic adherence to the clock. This report should therefore be seen not as an encouragement for complacency in the urgent diagnosis of this condition, but as a compelling argument for an aggressive, optimistic, and unwavering surgical commitment to limb salvage, even in the most daunting of delayed presentations.

5. References

1. Hester AG, Omidi N, Casella DP, Oetgen ME. Activation of a central immunosuppressive cascade prevents ischemia reperfusion injury after acute compartment syndrome in a Murine model. Journal of the Pediatric Orthopaedic Society of North America (JPOSNA). 2021; 3(3): 300.
2. Nakagawa Y, Makita S, Uchida H, Hinoki A, Shirota C, Sumida W, et al. Case report: A giant bilateral inguinal hernia requiring artificial mesh and multi-stage surgery in infancy; hernioplasty with silo placement to prevent acute compartment syndrome. Front Pediatr. 2022; 10: 1030934.
3. Finn D, Ashraf J, Tahir N, Devall M, Van Doorn C, Kumar R. Abdominal compartment syndrome as a complication of percutaneous nephrolithotomy requiring extra-corporeal life support—a case report. Pediatr Med. 2022; 5: 32.
4. Peek K, Schmale G, Lindberg A, Ho C, Miller M, Steinman S. Poor outcomes of pediatric acute compartment syndrome in the setting of extracorporeal membrane oxygenation: a multicenter case series. J Pediatr Orthop. 2022; 42(7): 382–6.
5. Tecos ME, Ballweg M, Hanna A, Thomas P, Zarroug A. Unique presentation of rectal prolapse as alarm symptom for pediatric

abdominal compartment syndrome. J Pediatr Surg Case Rep. 2022; 84(102394): 102394.
6. Wiegandt P, Jack T, von Gise A, Seidemann K, Boehne M, Koeditz H, et al. Awareness and diagnosis for intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) in neonatal (NICU) and pediatric intensive care units (PICU) - a follow-up multicenter survey. BMC Pediatr. 2023; 23(1): 82.
7. Pinto GCC, Gaiga L de C, de Moura MP, Troster EJ. Incidence and risk factors of abdominal compartment syndrome in pediatric oncology patients: a prospective cohort study. Eur J Pediatr. 2023; 182(8): 3611–7.
8. Tonge R, Lalwani S, Vageriya N, Shrivastava B. Acute compartment syndrome in a neonate: May be the first manifestation of hemophilia A. Indian Pediatr Case Rep. 2024; 4(1): 41–4.
9. Taniguchi M, Inata Y, Shimizu Y, Tamura D, Takeuchi M. Compartment syndrome due to group A streptococcal infection associated with intramuscular venous malformation. Pediatr Int. 2024; 66(1): e15756.
10. Bridges CS, Taylor TN, Bini T, Ontaneda AM, Coleman RD, Hill JF, et al. Acute compartment syndrome in pediatric patients on extracorporeal membrane oxygenation support. J Pediatr Orthop. 2024; 44(3): e285–91.
11. Shahrestani J, Smart EL, Mesa L, Martin C. Compartment syndrome in the pediatric patient. Pediatr Rev. 2024; 45(4): 247–9.
12. Manini N, Unno H. Delayed onset of neonatal compartment syndrome associated with compound fetal presentation. BMC Pediatr. 2024; 24(1): 224.
13. Sassoè-Pognetto M, Cavalcante R, Paonessa M. Acute compartment syndrome and fasciotomy after a viper bite in Italy: a case report. Ital J Pediatr. 2024; 50(1): 70.
14. Fanelli D, Barbieri C, Grap S, Adhikary S, Parekh UR. Abdominal compartment syndrome in children: Perioperative concerns and considerations ACS in children. Acad J Pediatr Neonatol. 2024; 14(1).
15. Sajankila N, Tang B, Montelione KC, Robertson JO, Guelfand M. Abdominal compartment syndrome requiring urgent decompression in infants with severe respiratory syncytial virus infection: a case series. J Pediatr Surg Case Rep. 2024; 105(102813): 102813.
16. Wilson KD, Bomar JD, Edmonds EW. Intermediate duration outcomes of surgical management for chronic exertional compartment syndrome in adolescents and young adults: The Mubarak fasciotome. J Pediatr Orthop. 2025; 45(5): 287–93.
17. Otludil B, Aksoy GK, Koyun M, Çomak E, Avanaz A, Akkaya B, et al. Delayed-onset renal allograft compartment syndrome in a pediatric kidney transplant recipient: The role of surgical re-evaluation. Pediatr Transplant. 2025; 29(3): e70070.
18. Zhang H, Lu W. Successful treatment with decompressive laparotomy for abdominal compartment syndrome induced by asparaginase-associated pancreatitis in a pediatric patient with acute lymphoblastic leukemia: a case report. Transl Pediatr. 2025; 14(5): 1050–8.
19. Velaoras K, Pantalos G, Plataras C, Alexandrou I, Mene J, Filos K, et al. Emergency laparotomy for abdominal compartment syndrome in a child due to chronic functional constipation. Case Rep Pediatr. 2025; 2025: 5289632.
20. Haridas V, Jose D, Noble C, Joji P. Management of compartmental syndrome in a child with hemophilia. Indian J Pediatr. 2025; 92(8): 883.