Paravertebral calcification as a potential indicator for nonaccidental trauma

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ABSTRACT

We report a case of nonaccidental trauma (NAT) involving a 23-month-old boy who presented with seizures, acute subarachnoid hemorrhage, and acute subdural hemorrhage. Ophthalmologic examination showed bilateral intraretinal hemorrhages. Further evaluation revealed that he had bilateral thoracolumbar paravertebral calcifications. The Children's Protective Services agency was involved in the case. The child was discharged to an inpatient rehabilitation facility. Vertebral fracture associated with paravertebral calcification has been reported as a sign of NAT. This case was unique because our patient had paravertebral calcifications without vertebral fracture. Paravertebral calcification alone could serve as an indicator of NAT.

CASE REPORT

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A 23-months-old previously healthy boy presented to the emergency department with status epilepticus. He had been examined at an emergency department 2 weeks prior, where he was diagnosed with viral gastroenteritis. At that time, it was noted that he also had bruises and a petechial rash of the left upper arm, right arm, and left ear. On the day of admission to the hospital, he had been found unconscious at home. He was brought to the emergency department via ambulance while still actively seizing. Lorazepam was administered to stop the seizure. Head computed tomography (CT) scan revealed acute subdural and subarachnoid hemorrhages (Figure 1). He was admitted to the pediatric intensive care unit for further management.

CT scan of the chest and abdomen with contrast on day 2 of admission showed soft tissue calcifications posterior to and to the right of the spinous processes of the lower thoracic and upper lumbar spine. Alignment of the vertebral bodies was normal, without evidence of acute or healing fractures. There were also linear soft tissue calcifications adjacent to the rib ends at the costovertebral junctions of the lower thoracic and upper lumbar spine (Figure 2). Brain magnetic resonance imaging (MRI) performed on day 3 revealed subdural hemorrhage and cerebral infarction in the left parietal/occipital cortex and in the posterior portions of the basal ganglia bilaterally (Figure 3). He was extubated on day 3 and he experienced another seizure on day 6. Levetiracetam was started. Electroencephalogram showed encephalopathy without epileptiform activities. His ophthalmologic evaluation revealed multiple bilateral intraretinal hemorrhages and left-sided pre-retinal hemorrhage. Skeletal survey did not reveal any fractures. He received physical therapy, occupational therapy, and speech therapy during his hospital stay. Children's Protective Services obtained custody of the child. He was discharged to an inpatient rehabilitation facility on day 21 of admission to continue intensive inpatient rehabilitation.

DISCUSSION

Etiology, Demographics & Imaging Findings:

Nonaccidental trauma (NAT) is associated with high morbidity and mortality rates [1]. NAT that initially presents with minor trauma is referred to as a "sentinel case" [2]. It is important to correctly detect the underlying NAT in sentinel cases to protect children from subsequent, recurrent, and often worse trauma. We examined a complicated, albeit common, case of pediatric NAT with acute subarachnoid and subdural intracranial hemorrhages, seizures, bilateral multilayered retinal hemorrhages, and cerebral infarctions.

The child was noted to have paravertebral calcifications around the lower thoracic and upper lumbar vertebral bodies without evidence of thoracolumbar fracture. Thoracolumbar fracture is a known radiological finding in NAT and often presents with paravertebral calcification [3]. However, paravertebral calcification without thoracolumbar fracture, which was observed in this case, has not been previously reported as a manifestation of NAT.

Because our patient was a victim of child abuse, we speculated that the paravertebral calcifications in our case were secondary to previous NAT. We also speculated that the patient previously sustained minor injuries that caused soft tissue damage and subsequent calcifications, but that the injury was not significant enough to cause vertebral fracture or spinal injury. Irregularity and subtle adjacent calcifications of the posterior ribs noted on computed tomography (CT) scan also suggested previous NAT. It is possible that the child suffered vertebral fractures that completely healed by the time of this admission. However, we believe that this is unlikely given the absence of any signs of previous vertebral fractures on CT. He did not show neurological abnormalities suggestive of spinal injury at presentation or during his emergency department visit 2 weeks prior to presentation. This supports our speculation that he did not sustain a major spinal injury. MRI of the thoracic spine and lumbar spine was not performed.

The mechanism of injury that causes paravertebral calcification is unclear. However, a possible mechanism of thoracolumbar fracture with listhesis is the combination of an axial load, flexion, and rotation [3, 4]. This combination could occur during an aggressive shaking episode. Just as the head moves during a shaking episode, so can the lower body and pelvis. Bleeding in these areas can be caused by the fingers of the abuser pushing deep into the paravertebral soft tissues or from tissue disruption during a shaking episode. Thereafter, calcification, just as is seen in myositis ossificans circumscripta, can result. Paravertebral calcifications are commonly seen with thoracolumbar fractures [3], and suggest an overlap in the mechanisms of injury. Chronic and less intense ligamentous and muscular injuries without vertebral fractures also may have resulted in paravertebral calcifications.

Risk factors for paravertebral calcification without thoracolumbar fracture are unknown. Risk factors for NAT include familial dysfunction, domestic violence, young maternal age, familial disruption and separation, male sex, history of abuse, and a nonbiologiccal parent. Previous case series of paravertebral calcification with thoracolumbar fracture have included children between 6 months and 7 years of age [3]. No treatment is needed for the calcification itself, but coexisting disabilities secondary to NAT must be addressed appropriately. The prognosis depends on the coexisting disabilities and whether a subsequent abusive event occurs.

If NAT is not considered among the differential diagnoses as a cause of paravertebral calcification, then mistaken diagnoses will be made. A previous case of NAT involving thoracolumbar fracture with listhesis was misdiagnosed as neuroblastoma [3]. The consequences of missed NAT can be grave, leading to double or triple mortality rates for subsequent events [5].

We speculated that paravertebral calcification without listhesis was related to NAT in our case. Currently, there are no reports that definitively support this relationship, and we recognize that our speculation remains a hypothesis at this point. However, given the devastating prognosis for missed NAT, clinicians should be aware of any subtle signs of NAT. We believe that paravertebral calcification without listhesis, given the probable mechanisms of injury, should serve as a sign to clinicians that NAT may have occurred.

Differential Diagnosis:

The differential diagnoses for the cause of paravertebral calcification include trauma, infection, rheumatic disease, malignancy, and idiopathic hyperostosis (table 2). In addition to radiologic information, clinical information is helpful for differentiating each condition.

The radiographic characteristics of paravertebral calcification observed in NAT have not been reported in detail. The presence of bony trabecular architecture in paravertebral calcification has been reported as an indicator of traumatic origin of the calcification [6]. X-rays are less sensitive for identifying paravertebral calcification compared to CT. Whether there is a thoracolumbar fracture or any other trauma, if a child has paravertebral calcification, then NAT must be suspected.

In addition to NAT, ankylosing spondylitis and tuberculous spondylitis can be associated with reactive paravertebral calcification. Joint erosions, subchondral sclerosis, and bony ankyloses are characteristics of ankylosing spondylitis. Ossification of the paravertebral connective tissue and posterior interspinous ligament can be seen on CT scan. A Romanus lesion is a radiographic finding characterized by small erosions at the corners of vertebral bodies surrounded by reactive sclerosis. Abscess formation and vertebral changes such as ivory vertebrae, gibbus deformity, and vertebra plana suggest tuberculous spondylitis. Dracunculosis is characterized by linear irregular calcifications of the affected soft tissue. However, this finding is extremely rare outside of South Sudan, Mali, Ethiopia, and Chad [7].

Furthermore, neuroblastoma can present as a heterogeneous soft tissue mass with calcification. If calcification is seen within mass lesions, then neuroblastoma and paraspinal ganglioneuroma should be suspected. Approximately 30% of neuroblastoma cases demonstrate calcification when X-rays are used and 80% demonstrate calcification on CT [8]. Paraspinal ganglioneuroma is a homogenous mass in the posterior mediastinum. Discrete and punctate calcifications are noted in 42-60% of cases when CT scan is used [8].

Diffuse idiopathic skeletal hyperostosis is typically found in elderly individuals and is characterized by idiopathic flowing ossification of the anterior aspect of the vertebral bodies.

TEACHING POINT

Paravertebral calcification without thoracolumbar fracture may be a sign of nonaccidental trauma (NAT). Because of the high morbidity and mortality rates associated with missed NAT, providers must be aware of the possible association between NAT and paravertebral calcification.

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FIGURES

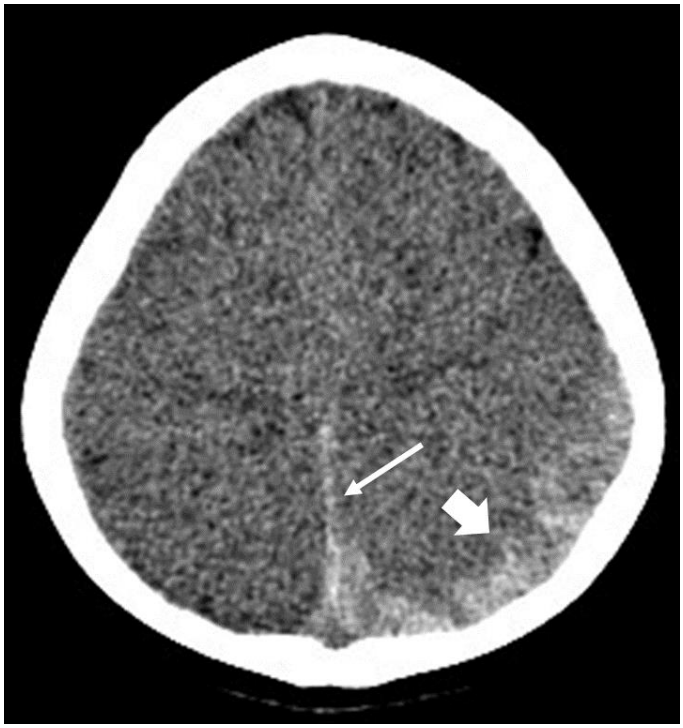

Figure 1: 23-month-old boy with subdural hemorrhage and subarachnoid hemorrhage.
Finding: Computed tomography on day 1 of admission. There is a hyperdensity of the posterior falx (white arrow) as well as sulcal effacement of the left posterior parietal lobe (block arrow).
Technique: Axial CT, 150 mA, 100 kV, 3 mm slice thickness, no contrast.

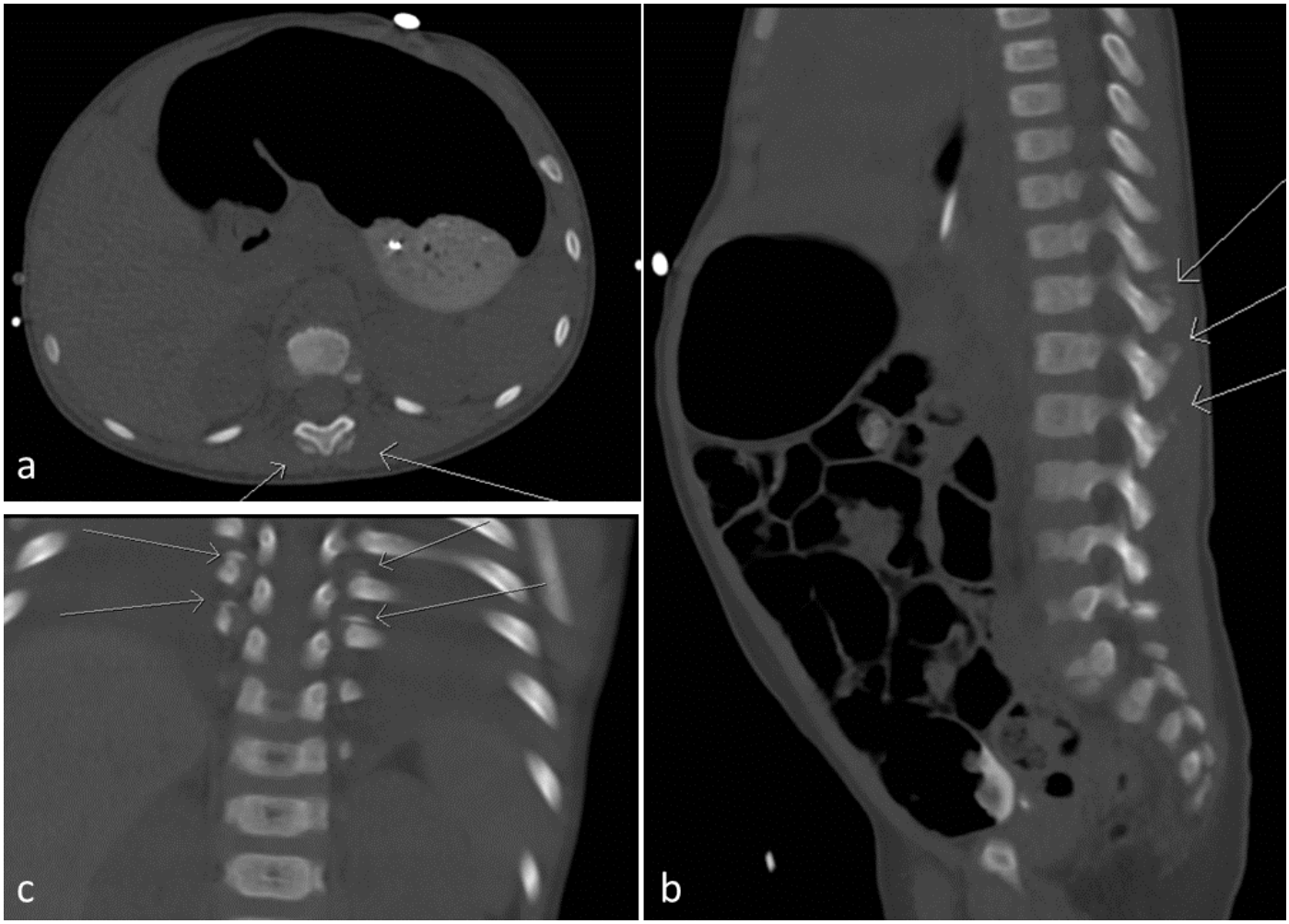

Figure 2: 23-month-old boy with paravertebral calcification.
Finding: Computed tomography (CT) on day 2 of admission. (a) Soft tissue calcification noted posterior to and to the both sides of the spinous processes of the lower thoracic and upper lumbar spine (arrow). (b) Soft tissue calcification noted posterior to the spinous processes at the lower thoracic and upper lumbar spine including T11 through L1 (arrows). Normal alignment of the vertebral bodies without evidence of acute or healing fractures. (c) Linear soft tissue calcifications adjacent to the rib ends at the costovertebral junctions of the lower thoracic and upper lumbar spine (arrow).
Technique: Axial CT (a), 93 mA, 100 kV, 0.5 mm slice thickness, no contrast; sagittal CT (b), 93 mA, 100 kV, 5 mm slice thickness, no contrast; coronal CT (c), 93 mA, 100 kV, 5mm slice thickness, no contrast.

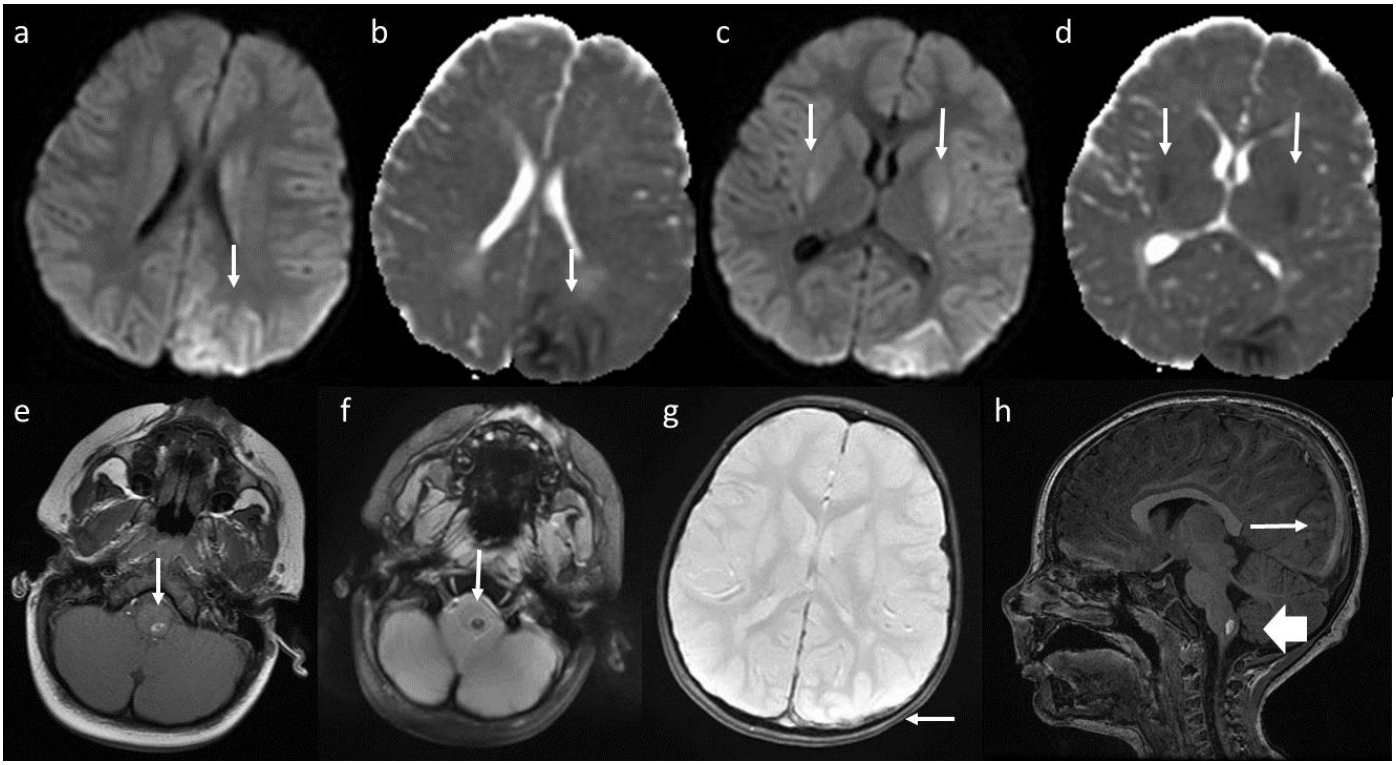

Figure 3: 23-month-old boy with left subdural hemorrhage and cerebral infarction in the left cerebral cortex and bilateral posterior basal ganglia.
Finding: Brain magnetic resonance image (MRI) on day 3 of admission. (a, b) There is an area of restricted diffusion involving mainly the cortex in the left posterior parietal lobe extending into the left occipital lobe (arrow). (c, d) There is an area of restricted diffusion in the posterior basal ganglia bilaterally (arrow). (e, f) Signal loss on gradient echo images noted within the left posterior medulla (arrow). Given the clinical scenario, this probably reflects deoxyhemoglobin from recent injury. (g) Thin left-sided subdural hematoma overlying the left parietal occipital region (arrow). (i) Subdural hematoma extends medially to the falx and tentorium (arrow). Hyperintensity is noted in left medulla (block arrow).
Technique: Brain MRI. 3 Tesla magnet, no contrast used. a. DWI, TR 8000, TE 100.8, no contrast; b. ADC, TR 8000, TE 100.8; c. DWI, TR 8000, TE 100.8; d. ADC, TR 8000, TE 100.8; e. axial T1, TR 616.7, TE 20; f. axial T2 MERGE, TR 600, TE 12.21; g. axial T2 MERGE, TR 600, TE 12.21; h. sagittal T1 BRAVO, TR 11.85, TE 4.8.

Etiology	Nonaccidental trauma (NAT). Exact mechanism is unknown, but proposed hypotheses include occult trauma causing soft tissue damage and subsequent calcification.
Incidence	Unknown. Paravertebral calcification with thoracolumbar fracture was previously reported. However, paravertebral calcification without thoracolumbar fracture previously has not been reported.
Gender ratio	Unknown. No gender difference expected.
Age predilection	Previous case series of paravertebral calcification with thoracolumbar fracture includes children between 6 months and 7 years of age.
Risk factors	Unknown. Risk factors of NAT should be considered, such as familial dysfunction, domestic violence, young maternal age, familial disruption and separation, and prior history of abuse.
Treatment	Treatment is not required for calcification itself, but coexisting disabilities secondary to NAT need to be addressed appropriately.
Prognosis	Prognosis depends on the coexisting disabilities secondary to NAT.
Findings on Imaging	Scattered calcification in paravertebral area without thoracolumbar fracture. Other finding suggestive of NAT may be identified.

Table 1: Summary table of paravertebral calcification without thoracolumbar fracture.

	X-ray	CT
Nonaccidental trauma	- Scattered paravertebral calcification may be observed. Less sensitive compared to CT.	- Paravertebral calcification with or without signs of thoracolumbar fracture. - Bony trabecular architecture indicates traumatic origin.
Neuroblastoma	- Non-specific soft-tissue mass - Calcification may be noted in 30% of cases.	- Heterogeneous mass with calcification observed in 80% of cases.
Ankylosing spondylitis	- Small erosions at the corners of vertebral bodies surrounded by reactive sclerosis (Romanus lesion). - Ossification of the paravertebral connective tissue and posterior interspinous ligament. - Bamboo spine.	- Joint erosions, subchondral sclerosis, and bony ankyloses. - Ossification of the paravertebral connective tissue and posterior interspinous ligament.
Tuberculous spondylitis	- Irregularity of the anterior vertebral margin. - Ivory vertebrae, gibbus deformity, and vertebra plana. - Calcification can be seen.	- Presence of pre- and paravertebral or intra-osseous abscess, and ivory vertebra. - Calcified paravertebral soft tissue.
Dracunculosis	- Linear irregular calcifications of the affected soft tissue	- Linear irregular calcifications of the affected soft tissue
Diffuse idiopathic skeletal hyperostosis	- Flowing syndesmophytes along, but separated from, the anterior aspect of the vertebral bodies.	- Ossification along the anterior aspect of the vertebral bodies.
Paraspinal ganglioneuroma	- Posterior mediastinal mass - Up to 30% may show calcification	- Homogeneous mass - Discrete and punctate calcification in 42-60% of cases.

Table 2: Differential diagnosis table for paravertebral calcification.

ABBREVIATIONS

CT = Computed tomography
NAT = Nonaccidental trauma

KEYWORDS

child abuse; seizure; subdural hemorrhage; subarachnoid hemorrhage; paravertebral calcification

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