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CASE REPORT

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Multiple thoracic vertebral compression fractures caused by non-accidental injury: case report with radiological–pathological correlation

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Abstract We report a 21-month-old boy with multiple contiguous thoracic vertebral compression fractures involving eight vertebral bodies, attributable to non-accidental injury. No subluxation was associated, however, there was extensive injury to the upper cervical and lower lumbar regions of the spinal cord. Anterosuperior beaking, thought to represent a previous injury, was evident in a mid-lumbar vertebra. Clinical examination revealed bilateral retinal hemorrhages and retinoschisis. Death occurred as a result of severe brain edema with bilateral subdural and subarachnoid hemorrhages. Radiological-pathological correlation is presented.

Keywords Children · Injuries · Spine · Fractures · Non-accidential trauma

Introduction

Spinal fractures are uncommon in young children; the majority occuring in the cervical spine because of the increased range of movement of this portion of the spine [1]. It is estimated that of all fractures seen in abused children, less than 1% involve the spine [2]. Often, vertebral fractures are not associated with clinical findings and unless other injuries present for medical attention, they may go undiagnosed.

Fortunately, spinal cord injuries, which are usually caused by fractures with retropulsion, are rare [3–5]. The majority of fractures involve the vertebral body without displacement, for example, anterior compression fractures. Fractures of the spinal processes have been documented in the shaken baby syndrome [6]. Involvement of the posterior elements, however, is rarely seen without an associated fracture of the vertebral body [7].

Anterior beaking of vertebral bodies has been described as a normal variant [8]. However, if the appearance is marked, present at multiple levels and associated with intervertebral disc narrowing, the appearance suggests previous injury with disturbance of growth at the endplate physis.

Case report

A 21-month-old boy arrived at the emergency room with a head injury. He had reportedly fallen backward while standing on a chair, hitting the back of his head on a concrete floor. He had been eating lunch and reportedly choked on food after falling. The boy's caregiver, his mother's boyfriend, said he had attempted to sweep food particles out of the boy's mouth and noticed that he was not breathing. He said he picked up the patient and ran

to the nearest emergency room, arriving there within 10 min. The patient was apneic and pulseless on arrival.

Cardiopulmonary resuscitation was started. He was intubated and ventilated, and spontaneous cardiac rhythm was re-established after approximately 10 min. His Glasgow Coma Scale rating was three throughout this time.

The patient had been previously healthy and had attained appropriate developmental milestones. He had been seen in the same emergency department 3 months earlier after a fall down the stairs in his home. The caregiver had also been his mother's boyfriend on that occasion. He had a contusion of his forehead, but a three-view skull radiograph series showed no bone injury and he was discharged home.

Prior to transfer to this institution a CT brain study was performed. It revealed bilateral subdural hematomas overlying the frontal convexities bilaterally, extending over the vertex and tracking along the falx cerebri. There was also a subdural hematoma along the right tentorium cerebelli. Subarachnoid hemorrhage was present within the left sylvian fissure and along the posterior temporal and parietal sulci on the left. There was loss of demarcation of the gray and white matter interface suggestive of cerebral edema. No acute fractures were seen, but there was a partially healed fracture adjacent to the left lambdoid suture.

A second CT scan of the brain obtained 8 h after the first scan showed progression of the cerebral edema. Ophthalmologic evaluation revealed extensive bilateral retinal hemorrhages. A skeletal survey was obtained to assess for other occult injuries. Anterior wedge compression fractures were noted in the thoracic spine extending from T5–12 (Fig. 1). Additionally, there was anterior-inferior beaking of the L2 vertebral body with narrowing of the L1-2 disc space.

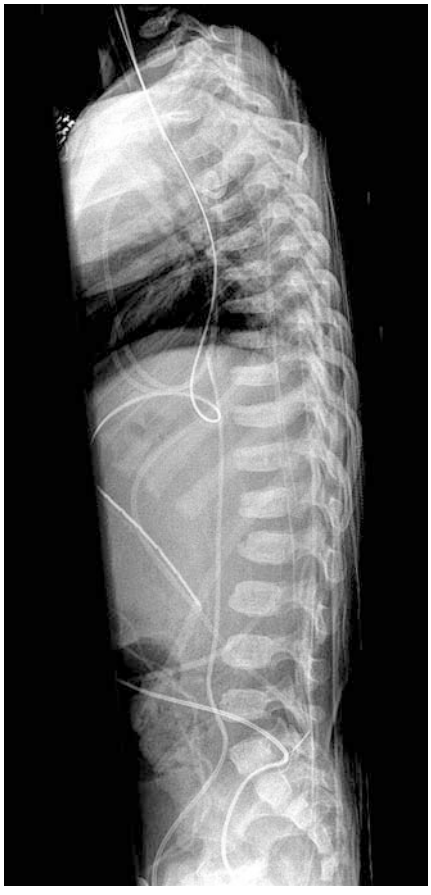

Fig. 1 Lateral radiograph of the thoracolumbar spine. Severe anterior compression fractures are noted of the 5–12 thoracic vertebrae. T7 vertebral body is the most severely affected. Spinal alignment is preserved. There is an anterosuperior defect of the L2 vertebral body with associated narrowing of the L1-2 disc space

The patient remained unconscious and ventilation-dependent. Two days after admission clinical tests revealed no brain activity and he was declared dead.

A post mortem CT scan of the entire spine confirmed the radiographic findings, showing multiple anterior compression fractures of the thoracic spine extending from T5 to T12 (Fig. 2). The most severely affected vertebra was T7, with moderate compression fractures of T6, T8 and T9. Vertebral bodies T5 and T10, T11 and T12 demonstrated only mild anterior wedging. Spinal alignment was normal and no paravertebral soft tissue abnormalities were detected.

Necropsy revealed hemorrhage in the anterior and posterior spinal ligaments in the upper thoracic and lumbar regions. After the spine was decalcified, it was hemisectioned in the saggital plane (Fig. 3). T5–12 showed varying degrees of axial compression and anterior wedging. The process was most evident in the anterosuperior margins of the vertebral bodies, but was also seen in the anteroinferior margins. There was compression of the centrum with an acute anterior angle. Although the wedge-shaped vertebral bodies were thinnest in the region of their anterior lips, the posterior bodies were also compressed.

The upper cervical cord and the lower lumbar cord extending to the conus medullaris showed gross and microscopic injury. There was softening of the cord indicating degeneration of devitalized neural tissue in the interval between the injury and death. Microscopically, extensive multifocal petechial hemorrhages were seen within the cervical cord, almost entirely within the gray matter. The neurons were degenerated or absent. There was architectural distortion and it was difficult to separate the gray from the white matter with a hematoxylin and eosin stain. The thoracic cord was unremarkable. Two central cavities were seen within the lumbar cord, in the dorsal columns bilaterally. The pericavitary cord was composed of rarefied tissue with reactive astrogliosis, marked vascular congestion, and petechial hemorrhages. Rare axonal spheroids were seen

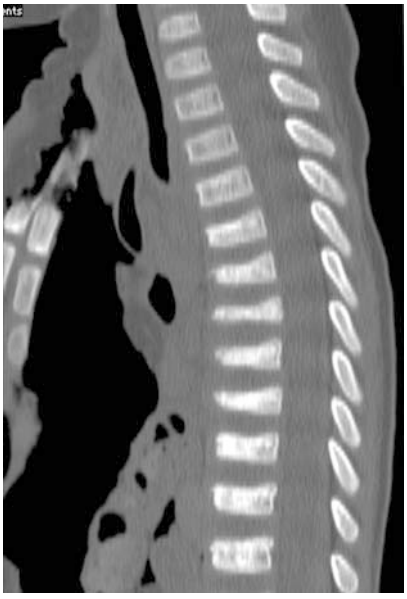

Fig. 2 Sagittal reformat of a CT scan of the thoracic spine from C7 to T12 vertebral bodies. Compression fractures with anterior wedging of the 5–12 vertebral bodies are seen. Loss of height of the posterior aspects of the vertebral bodies is also evident with prominent buckling of the posterior margins of T11 and T12 vertebral bodies. The vertebral alignment is normal with no listhesis

Fig. 3 Post-mortem specimen of the thoracic spine from T2 to L3, cut in the sagittal plane. Anterior wedging and generalized loss of height is evident in the 5–12 thoracic vertebrae. The adjacent intervertebral discs appear normal. Anterosuperior notching is seen of the L2 vertebral body. The L1–2 disc has been disrupted in the post mortem dissection of the spine

in the peripheral white matter tracts. Small volume extradural and subarachnoid hemorrhage was noted in the spinal cord meninges.

Post-mortem findings confirmed the neuroimaging findings of multifocal subdural hematomas, bilateral subarachnoid hemorrhage, and diffuse brain edema. There were no scalp lacerations or other evidence of an impact head injury. Multifocal bilateral retinal hemorrhages and traumatic retinoschisis were also confirmed.

Discussion

There was no objective evidence in this case of an impact injury, although blunt impact could have been inflicted without leaving residual markings such as contusions or lacerations. The partially healed skull fracture detected on CT is not consistent with an acute injury at the time of presentation and is likely to have been inflicted earlier. The patterns of intracranial and retinal abnormalities are most consistent with injuries seen in shaken baby syndrome. Interhemispheric subdural hemorrhage and bilateral retinal hemorrhage are typical of this kind of abuse [9, 10]. Children injured by shaking are usually infants, but toddlers may be abused in this way with or without associated impact injury. Despite the relative large size of this 21-month-old boy, the most likely mechanism of injury causing the intracranial lesions is violent shaking with the assailant's hands on the torso and repeated to-and-fro motion of the head. In contrast, anterior compression fractures of vertebral bodies are most consistent with axial-loading and/or hyperflexion injury to the spine. It is unclear why these injuries were sustained in the relatively protected thoracic spine rather than the cervical or lumbar spine, where a greater range of movement is possible. As is often the case, no adequate history was provided to serve as a clue of the mechanism involved. Hemorrhage was seen in both the anterior and posterior spinal ligaments, but not within the interspinous ligamentous attachments, as is sometimes seen in hyperflexion injuries.

Of interest is the anterosuperior notching of the L2 vertebral body. Although caution is necessary in attributing this finding to an occult remote injury, the associated narrowing of the L1–2 disc space leads credence to this possibility [11]. A previous Salter fracture of the anterosuperior endplate physis, with subsequent restricted growth, would give rise to this appearance. The normal variant described by Swischuk [8] was a subtle notch in the upper anterior corner of the vertebra without associated findings. This radiological finding is also reported in children with mucopolysaccharidosis; however, this entity is easily ruled out given the absence of other characteristic bone findings seen in this disease. The patient's documented fall down 13 stairs 3 months prior to this presentation may not account for this finding, given the short time span involved. The partially healed skull fracture seen on several CT head scans may have occurred at that time. This was not detected on a three-view skull series at the time of the injury and was not evident on skull radiographs performed in our institution as part of a bone survey. This fracture was not confirmed at post mortem, most likely because it was not specifically searched for during the necropsy.

This case emphasizes the importance of a complete skeletal assessment of children suspected of having an

inflicted injury. The radiological workup should require two views of the entire spine, even in the absence of symptoms or clinical signs referable to the spine. Although CT is excellent for evaluation of bone injuries, MRI is indicated as a complementary study for more sensitive assessment of the paravertebral soft tissues, spinal meninges and cord. In this case the advanced intracranial injuries precluded this workup and, indeed, the CT scan of the spine was undertaken as a postmortem study. Sagittal and coronal reformats did not add additional information to that already known from the lateral and frontal radiographs, as the spinal alignment was intact. Presumably, if there had been disruption of the spinal alignment or retropulsion of fracture fragments, CT would have been valuable. In cases where CT of the chest, abdomen, and pelvis is obtained for assessment of severely abused moribund children, reformated images of the spine from raw data acquired is possible with multidetector scanners. This is an advisable exercise to perform routinely in an attempt to detect occult fractures. It may be particularly useful to evaluate the upper thoracic spine or posterior spinal elements—areas relatively poorly assessed by plain radiographs.

We can only speculate whether there was paresis of the upper or lower extremities as a result of the spinal cord injuries documented on post mortem histopathology. The child remained deeply comatose during his entire hospital course, making a complete neurological assessment impossible.

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