

# Reverse and pseudoreverse cortical sign in thoracolumbar burst fracture: radiologic description and distinction—a propos of three cases

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**Abstract** In thoracolumbar burst fracture the “reverse cortical sign” is a known entity that corresponds to a fragment of the posterior wall that has been flipped 180° with the cancellous surface of the fragment facing posteriorly in the canal and the cortical surface (posterior wall) facing anteriorly. The identification of such reverse cortical fragment is crucial as ligamentotaxis is classically contraindicated as the posterior longitudinal ligament is ruptured. Recognition of such a flipped cortical fragment has relied so far on the axial CT. The advent of CT scans with sagittal reconstruction has allowed us to better describe such entities that have received little attention in the literature. The goal of this report was therefore to describe the appearance of the reverse cortical sign and its likes as they can appear on axial CT scans, sagittal reconstructions and MRI. During 1-year practice at our institution we had to treat three patients with thoracolumbar burst fracture associated with what looked like a reverse cortical sign on the axial CT scans. Further analysis of the sagittal reconstruction CT could differentiate the true reverse cortical sign from a new entity that we coined “the pseudoreverse cortical sign” as observed in two out of the three cases. In the pseudo reverse cortical sign what appears to be a flipped piece of posterior vertebral body is actually part of the superior or inferior endplate that is depressed into the

comminuted vertebral body. In such cases the posterior longitudinal ligament appears to be in continuity and therefore such fracture can theoretically be treated with posterior ligamentotaxis as evidenced in one of our case. Careful analysis of the CT scan and specifically the sagittal reconstruction and MRI can differentiate two separate entities that may correspond to a different severity injury.

**Keywords** Reverse cortical sign · Burst fracture · Spine · Ligamentotaxis

## Introduction

Burst fractures of the thoracolumbar spine typically involve the posterior wall of the vertebral body that is retropulsed to a certain degree in the spine canal. The amount of spine canal compromise has drawn lots of attention as to the need to restore the spine canal anatomy especially in the phase of neurologically compromise patient [7, 9, 10, 12].

To achieve restoration of the canal size two opposite techniques continue to be debated: anterior direct decompression through a corpectomy followed by anterior vertebral body replacement with cage or graft or the posterior approach to the spine and ligamentotaxis using the tension of the posterior longitudinal ligament to push back the retropulsed fragment and restore the size of the canal [1–4, 6, 8, 10–13]. If debate continues between the proponents of anterior surgery versus posterior surgery the existence of a reverse cortical sign has been classically a contraindication of posterior ligamentotaxis [5]. However, no reference and no description of such reverse cortical sign could be found in a pubmed search. Over a 1-year period we had to treat three thoracolumbar burst fractures with what appeared a reverse cortical sign on the axial CT

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scans. Further scrutiny of the CT scans and MRI when available with specific attention to the sagittal reconstruction ruled out two cases out of three that were not a true “reverse cortical sign”. We therefore called these two cases “pseudoreverse cortical sign”. We report on these three cases to describe and explain this new radiographic entities and what they correspond to.

#### Case 1: true reverse cortical sign

A 53-year-old male sustained a fall from a significant height. He was noted to have loss of neurologic function

from thoracic level ten. Radiographs, CT, and MRI demonstrated an L1 flexion distraction burst fracture (B1.2 in the Magerl classification) with significant retropulsion and cord contusion. The posterior wall fragment appears to be totally detached from the vertebral body, its cortex appears to be sitting anteriorly and its cancellous portion is lying posteriorly in the spine canal (Fig. 1a). On the sagittal MRI the posterior longitudinal ligament appears to be disrupted. The posterior protruding wall fragment was interpreted as a “reverse cortical sign”. Because of the presence of this reverse cortical sign and his neurologic deficit he was urgently taken to the operating room and underwent an



**Fig. 1 a** True reverse cortical sign in a patient with a burst fracture of L1 (B1.2) and complete neurologic deficit. Note that the protruding piece in the spine canal comes from the superior aspect of the vertebral body and has “flipped 180°”, it is totally detached from the vertebral body. The posterior wall cortex is facing anteriorly, the

cancellous portion of the fragment is facing posteriorly. The MRI shows rupture of the posterior longitudinal ligament. Reduction of the fragment with ligamentotaxis is contraindicated. **b** After anterior decompression, removal of the reverse cortical fragment and anterior reconstruction

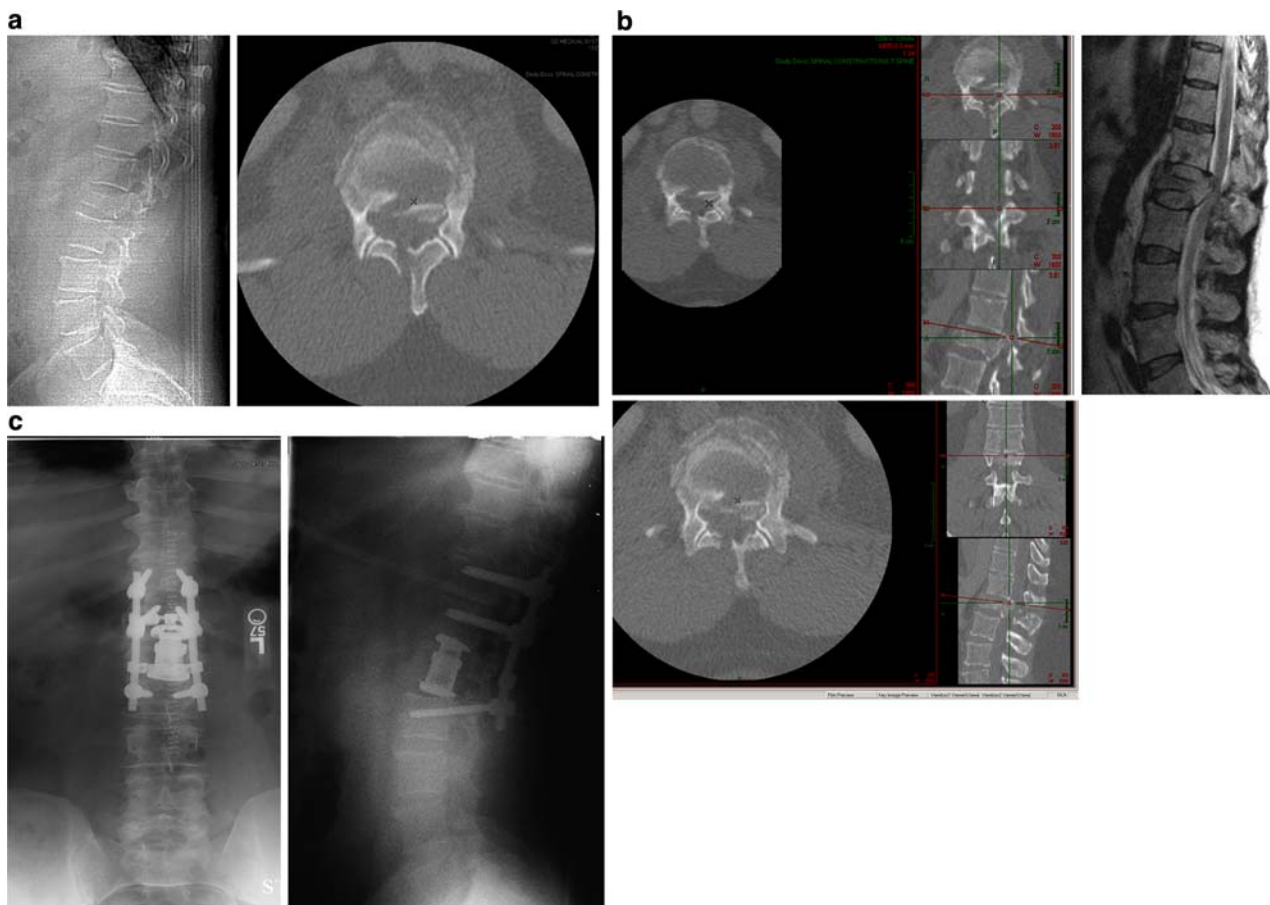
anterior L1 vertebrectomy, placement of an expandable cage (Synex, Synthes, PA, USA) and grafting of the defect was performed. In addition a T12–L2 anterolateral fusion was used with the Antares Plating system (Medtronic, Memphis, TN, USA) (Fig. 1b). Postoperatively he had no return of neurologic function.

#### Case 2: pseudo reverse cortical sign of the superior end plate

A 54-year-old male who fell from a 10 m height was evaluated in the emergency room for a burst fracture of the first lumbar vertebrae (Fig. 2a). He was noted to be neurologically intact. Axial CT scans demonstrated a significant retropulsion of the posterior wall. His fracture was

classified as an A3 in the Magerl classification as the posterior ligament complex was intact (despite a split lamina fracture). The cortex of the posterior wall fragment appeared to be sitting anteriorly and the cancellous portion of the piece seems to be lying posteriorly in the spine canal. This was initially interpreted as a “reverse cortical sign”.

Because of surgeon preference and the presence of what was thought to be a reverse cortical sign he was urgently taken to the operating room and underwent an anterior L1 vertebrectomy, and placement of an expandable cage (Synex, Synthes, PA, USA) and grafting of the defect was performed (Fig. 2c). In the same setting a posterior exposure with pedicle screw stabilization was performed from T11 to L2 (and not from T12 to L2 because of the sagittal split that existed at the T12 level). Postoperatively



**Fig. 2 a** Patient with a L1 burst fracture and significant retropulsion of the posterior wall in the spine canal. The patient is neurologically intact. The axial CT cuts (at the pedicle level) show a retropulsed fragment with what appears to be a dense area of cortex sitting anteriorly. This is initially thought to represent a reverse cortical sign with the fragment of the posterior wall flipped 180° with the cortex sitting anteriorly and the cancellous portion posteriorly (A3 type burst fracture). **b** Further scrutiny of contiguous axial views, the sagittal reconstruction and the MRI demonstrate that the posterior wall fragment (that we thought was flipped) correspond as a matter of fact

to the vertical depression of the superior endplate in the comminuted vertebral body. The posterior fragment is only rotated 90° and is not separated from the rest of the vertebral body. The MRI does not show any obvious rupture of the PLL. **c** Because of surgeon preference and what was thought to be a reverse cortical sign the patient underwent anterior decompression, corpectomy and posterior fusion (the posterior reconstruction from T10 to L2 was judged necessary because of the presence of a split sagittal fracture in the vertebral body of T12 not represented here)

patient neurology remained intact and the recovery was uneventful.

During the review of the CT scans and specifically the sagittal reconstructions of the burst fracture (Fig. 2b) at our weekly spine rounds it was thought that he did not have a reverse cortical sign, but in fact, a “pseudo reverse cortical

sign”. What appeared to be the flipped posterior wall of the vertebral body sitting anteriorly (with the cancellous portion of the piece sitting posteriorly in the canal) was as a matter of fact the dense superior end plate that had been depressed vertically in the vertebral body (Fig. 2b). The posterior fragment had rotated 90° in the canal but was not

**Fig. 3** **a** Burst fracture of L1 with 70% canal compromise and what appears to be a reverse cortical sign with the anterior cortex of the posterior wall facing anteriorly (the cut is at the inferior aspect of the pedicle and the foraminal level). **b** The sagittal reconstruction with the corresponding axial cuts show us that we are not dealing with a reverse cortical sign but it is the vertical depression of the inferior end plate into the comminuted vertebral body that mimics the flipped posterior wall cortex. The fragment is not separated from the vertebral body. **c** After posterior ligamentotaxis the retropulsed fragment in the canal has been significantly reduced confirming the integrity of the PLL



flipped as the first case and was not detached from the vertebral body. We thought that in such a case the posterior longitudinal ligament was intact as evidenced on further scrutiny of the preop MRI (Fig. 2a). It was then our opinion that such pseudoreverse cortical sign would have responded to ligamentotaxis.

#### Case 3: pseudo reverse cortical sign of the inferior end plate

A 38-year-old female was involved in single car accident with possible loss of consciousness, escaped a burning vehicle and was transferred to our facility. Her primary complaint was of lower back pain. She was neurologically intact. Plain radiographs and axial CT demonstrated an L1 burst fracture type A3 with approximately 60–70% retro-pulsion (Fig. 3a). Because of the presence of what appeared to be a reverse cortical sign the patient was thought to benefit from anterior decompression reconstruction. However, with further scrutiny and review of the CT scans and specifically the sagittal reconstructions, it appeared that the reverse cortical sign that we had identified corresponded as a matter of fact to a portion of the inferior endplate that had been depressed vertically into the vertebral body of the burst fracture (Fig. 3b). This corresponded to a “pseudo-reverse cortical sign”. We therefore changed our mind being convinced that this burst fracture would respond to ligamentotaxis as it was not a reverse cortical sign and the posterior longitudinal ligament had to be intact.

She was urgently taken to the operating room on the day of admission. She underwent posterior spinal instrumentation, ligamentotaxis and fusion from T12 to L2 (AO USS Paoli, PA, USA). Post operatively she had no neurologic deficits, was discharged on the fourth day and had uneventful recovery. To assess the restoration of the vertebral body height and the clearance of the canal we ordered a postoperative CT scan that showed an excellent reduction of the posterior wall confirming that the fragment initially thought to be a reverse cortical sign was a pseudoreverse cortical sign that had been reduced by ligamentotaxis (Fig. 3c).

#### Discussion

For a long time the presence of a “reverse cortical sign” has been a contraindication to posterior ligamentotaxis in the spine surgeon community. Yet this entity does not appear in the peer review literature, (except in textbooks) this is a well recognized appellation. Despite multiple medline search with different key words such as “reverse cortical sign”, “flipped cortical fragment” “burst fracture

canal compromise” we could not find any article mentioning such entity. For the spine surgeon posterior ligamentotaxis in the presence of a reverse cortical sign is contraindicated as it could theoretically lead to further displacement of the posterior wall fragment in the spine canal and possible worsening neurologic deficit. Posterior surgery may still be favored as an emergency procedure even in the presence of a reverse cortical sign as an initial stabilization procedure together with a direct decompression. The anterior column needs to be addressed secondarily.

Both the reverse and pseudoreverse cortical signs look alike on the axial CT cuts of the vertebral body. The astute physician may differentiate the true reverse cortical sign from the pseudoreverse cortical sign on multiple axial CT cuts but it is the sagittal reconstruction that most easily makes out the difference. The reverse and pseudoreverse cortical sign may correspond to a very different anatomic entity, or to a different stage in the gravity of the thoracolumbar burst fractures. We have shown that for the reverse cortical sign the posterior fragment is flipped 180° or more with a ruptured of the posterior longitudinal ligament and is detached from the vertebral body, for the pseudoreverse cortical sign the posterior fragment is only rotated 90° or less and the superior or inferior endplate attached to the fragment is depressed in the vertebral body mimicking the reverse cortical sign, the posterior longitudinal ligament is very likely in continuity. Naturally our study is limited to three cases only and a larger series will be necessary to confirm our findings that were, however, based on a very thorough analytic description of three cases.

#### Conclusion

Such distinction between a true reverse and pseudoreverse cortical sign is in our opinion essential as they may represent a different severity in the mechanism of thoracolumbar spine fracture, with the reverse cortical sign burst fractures more likely to have a neurologic deficit, as opposed to the pseudoreverse group where the PLL is likely intact. To be able to differentiate the two entities we recommend analyzing very thoroughly the sagittal reconstruction of the axial CT scans and the sagittal MRI sequences. By doing this we shall probably be able to come with a better descriptive anatomy of this posterior wall fragment that may be different according to the mechanism or the severity of injury, with possible different prognosis.

**Conflict of interest statement** In preparation of this manuscript the authors have no competing interests or financial interest to disclose.

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