Chapter

An Anatomical and Pathological Classification of Thoracolumbar Adjacent Segment Disease

David Christopher Kieser and Niels Hammer

Abstract

Structural failure of the spine adjacent to the level of a previous spinal fusion is commonly observed. It may be defined by the radiologic degree of adjacent deformity, often termed junctional level kyphosis, proximal junctional kyphosis or junctional level failure, or the symptomatic failure of the spine above the level of an operation, termed adjacent segment disease (ASD). ASD can be further specified according to its anatomical location of failure, which provides insight into the specific pathological cause of failure and the optimal subsequent management. This chapter describes the anatomical and pathological classification of ASD in order to help clinicians understand the cause of failure and thereby reduce its rate and offer a treatment algorithm if it occurs.

Keywords: adjacent segment disease, thoraco-lumbar fusion, pathological cause, junctional level kyphosis

1. Introduction

Adjacent segment disease (ASD) is the symptomatic structural failure of the spine or sacrum adjacent to an area of previous operative intervention, most notably fusion [1]. Internationally, the numbers of spinal fusions being performed is increasing. Within the USA approximately 457,500 adult spinal fusions and 38,000 paediatric spinal deformity corrections are performed annually, with similar rates per capita reported worldwide [2]. Of concern is that more than 20% of patients undergoing lumbar deformity surgery will develop ASD within 8 years, most of which occurs early with 40% requiring revision within 6 months [3–9]. This has a significant clinical effect on patient outcomes, with pain, neurological, emotional, social and occupational concerns, but also carries a large financial burden, with an estimated cost in the USA for revision being $77,432 USD per patient [10, 11]. This would suggest that over 500 million USD is spent annually on the surgical treatment of ASD. Yet, a complete understanding of the aetiology of this problem has not been compiled.

It is believed that the cause of ASD is multifactorial [12]. These causes can be separated into non-modifiable, potentially modifiable and modifiable risk factors.

Non-modifiable risk factors include patient age and expected baseline motion segment degeneration that cannot be modified with current known treatments [13, 14]. These factors are particularly pertinent in the adult population where their index procedure is often related to degeneration which itself renders patients at higher risk of degeneration at other levels.

Potentially modifiable risk factors include bone density, which may be amenable to medical treatment [15]. Others include fusion without instrumentation and limiting the fusion length, however the pathology often dictates the length of fusion and implant requirement [13, 16].

Modifiable risk factors include intraoperative surgical techniques, notably motion preservation to reduce the adjacent segment load, avoiding circumferential fusion of the most cranial segment that increases the stresses on the adjacent level, ensuring spinal balance, avoiding extensor musculature and ligamentous damage, protecting the adjacent facets, endplates and intervertebral disc (IVD) [13, 14].

While multiple classification systems and definitions have been proposed, none have attempted to group these into anatomical or pathological considerations. Our classification broadly categorises ASD into five groups according to the anatomical region of failure which can then determine the likely pathological cause and offer treatment direction.

2. The Kieser and Hammer classification system

2.1 Type 1: global failure (implant pull-out)

This form of ASD is seen when metalware pulls out of the vertebrae (**Figure 1**). This failure is not seen in anterior interbody constructs, unless supplementary posterior or lateral instrumentation is utilised. This is because it is recognised as persistent spinal malalignment, which requires interconnected rigid implants to remain in one position and the spine to displace from the rigid instrumentation. However, with lateral implants (plates, stapes, etc.) and a failure to restore coronal balance or posterior implants and a failure to restore sagittal balance, the metalware can pull out of the bone producing a type 1 failure.

This failure is therefore almost always due to malalignment, but can be sub-classified into:

1a. Bone failure: osteoporosis.
1b. Implant failure: insufficient fixation.
1c. Combination.

In type 1a, the bone quality is insufficient to hold the implanted device in a given configuration, with the effect that the metalware pulls out. Similarly, in type 1b the implant configuration is insufficient to stabilise and anchor in the bone and

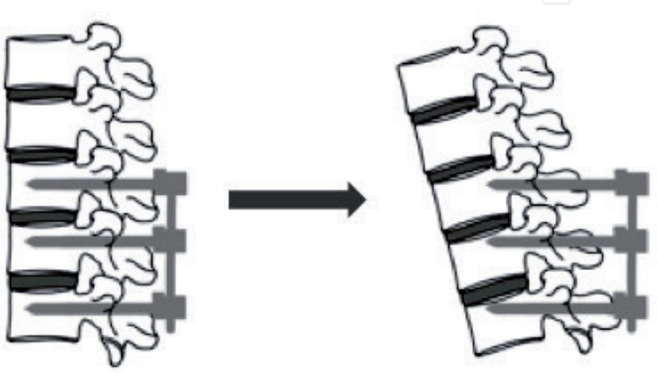

Figure 1.
Type 1 (global failure).

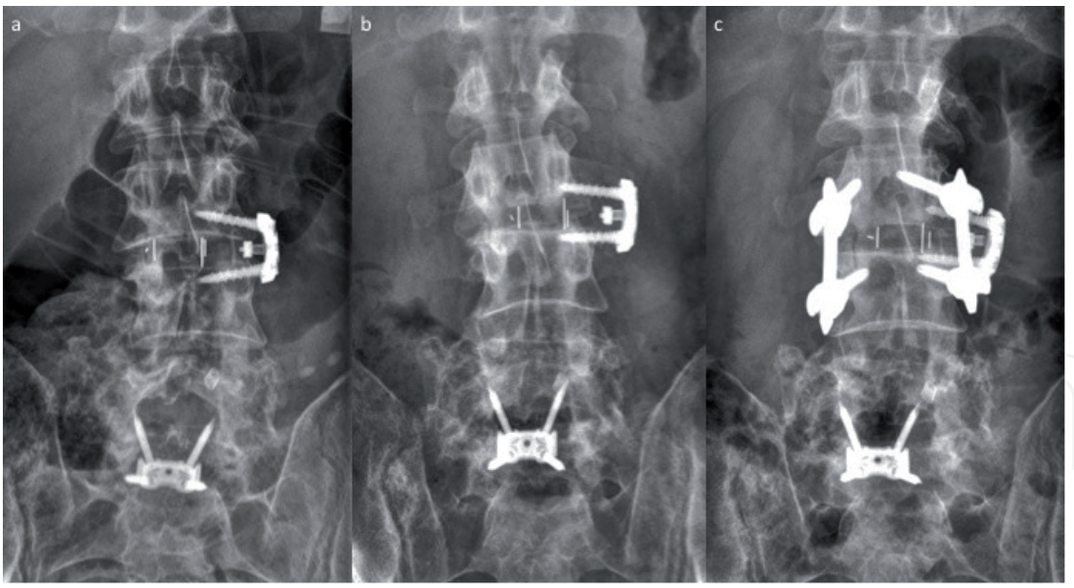

Figure 2.
Antero-posterior X-rays of a patient with a previous L4-S1 fusion who developed type 4d ASD of L3/4 causing foraminal stenosis and therefore underwent a lateral interbody fixation and plate (a. immediate postoperative) but with failure to completely restore coronal balance and insufficient fixation resulting in implant pull-out and progressive coronal imbalance with recurrence of symptomatic foraminal stenosis (b. 6 weeks postoperative). Therefore supplementary percutaneous pedicle screw insertion to augment fixation and bracing was undertaken, which resulted in sufficient fixation to permit definitive fusion (c. 6 months postoperative).

therefore it pulls out (**Figure 2**). In most cases it is a combination of both poor bone stock and insufficient implant fixation.

In an asymptomatic patient without skin compromise, the practitioner or surgeon advises bracing to prevent further progression until the fusion has developed. In contrast, in symptomatic patients or those with progressive failure amendable to operative intervention, the treatment of type 1 failure is revision surgery with restoration of spinal alignment and supplementary bracing until fusion has developed. In addition, for each sub-classification we advocate.

1a. Bone supplementation with medical management of osteoporosis (e.g. calcium, vitamin D, bisphosphonates, etc.) and increased fixation (e.g. cemented screws, HA coated screws, sublaminar bands, etc.).
1b. Increase fixation of the operative levels. May require extension of fusion if adequate fixation of the operative levels is not possible.
1c. Both.

It should be recognised that bone supplementation takes a prolonged period of time to achieve clinical benefit and most of these patients require semi-urgent surgical intervention. Thus, in symptomatic patients with deficient bone quality, increased fixation should be provided in addition to the medical management of osteoporosis. Furthermore, bracing should be considered to supplement the spinal stability provided by the surgery until fusion has occurred.

2.2 Type 2: adjacent bone failure (failure of the cranial or caudal uninstrumented vertebrae)

This form of ASD occurs when an adjacent, uninstrumented vertebrae fails, typically with a compression type fracture (**Figures 3** and **4**). This is most commonly caused by poor bone quality and/or malalignment and is therefore subclassified as:

2a. Poor bone stock.
2b. Malalignment.
2c. Combination.

Most patients have a combination of malalignment and osteoporosis but are predominantly affected by their poor bone quality. Unlike type 1 failures, these patients are rarely in need of an urgent surgical intervention. Therefore, a conservative approach can be initially trialled. Bracing as well as vertebroplasty or kyphoplasty should be considered to avoid progressive collapse and deformity. However, the clinician should recognise that vertebral body cementation may affect subsequent extension of fusion if necessitated, particularly if pedicle screws are considered necessary. Therefore, surgeons treating these patients should consider alternative fixation techniques, such as cortical trajectory screws, in the cemented vertebrae if extension is subsequently required. In those that become asymptomatic these fractures should be treated as osteoporotic compression fractures. In those that remain symptomatic and are amenable to operative intervention, the treatment should be:

2a. Bone supplementation ± bracing until fracture union is achieved. In a globally aligned spine with union of the fracture, no operative intervention is required.
2b. Deformity correction with extension of fusion and increased fixation. Increased fixation is necessary because the bone has shown evidence of weakness, even in the absence of global osteoporosis, and therefore increased fixation is necessitated.
2c. a ± b: Bone supplementation and bracing if the global malalignment is acceptable OR bone supplementation and deformity correction with extension of fusion, increased fixation and bracing if the malalignment is unacceptable.

2.3 Type 3: endplate failure

This is one of the most common forms of ASD and it occurs when the most cranial instrumented vertebral body collapses (**Figures 5** and **6**). It can occur with posterior or anterior/lateral implants and its causes can be classified as:

3a. Poor bone stock.
3b. Devascularisation of the endplate.
3c. Excessive endplate load.
3d. Combination.

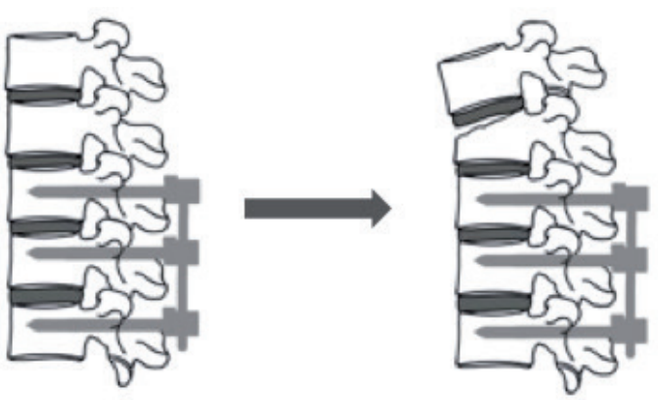

Figure 3.
Type 2 (adjacent bone failure).

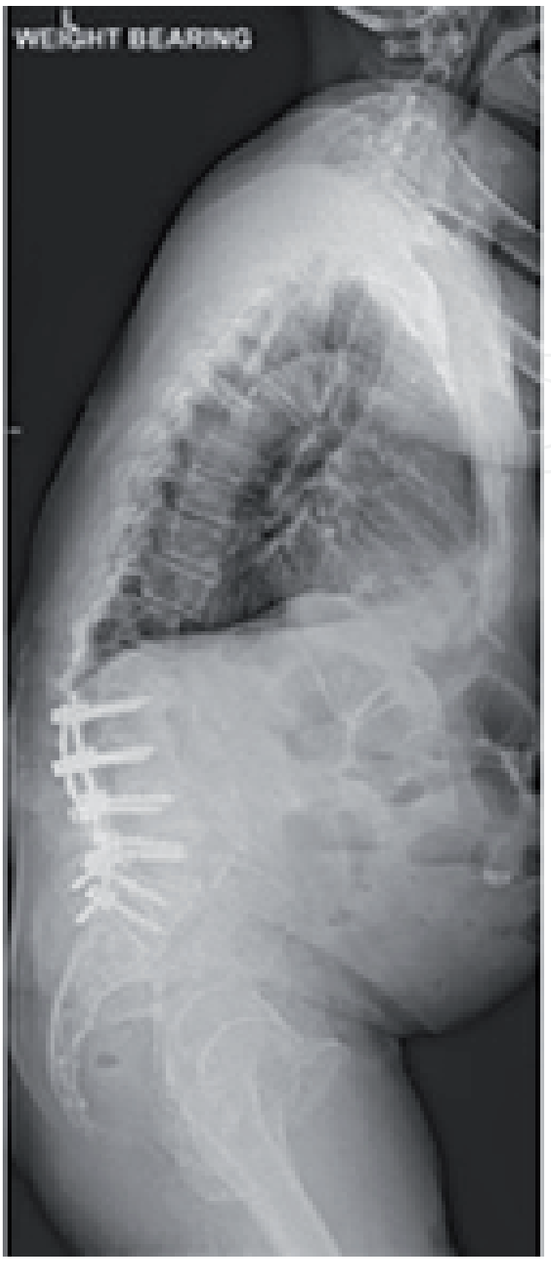

Figure 4.
A lateral standing full spine x-ray of a patient who had undergone an L2-S1 fusion with a failure to correct sagittal balance who developed an adjacent compression fracture of L1 that accentuated the spinal imbalance and over a 6-year period, despite attempted compensation with pelvic retrolisthesis and thoracic hypokyphosis, they developed a progressive anterolisthesis of T11/12 causing thoracic myelopathy.

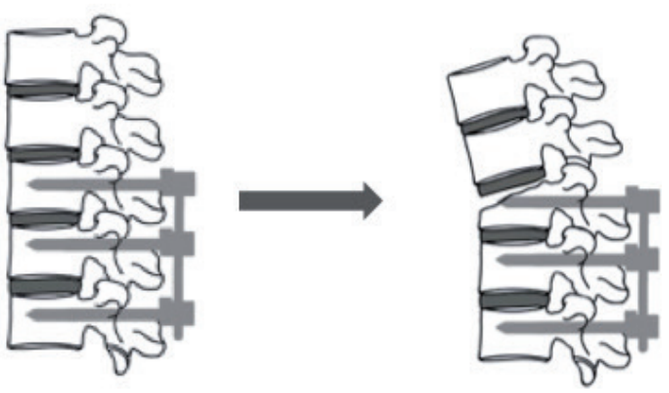

Figure 5.
Type 3 failure (endplate failure).

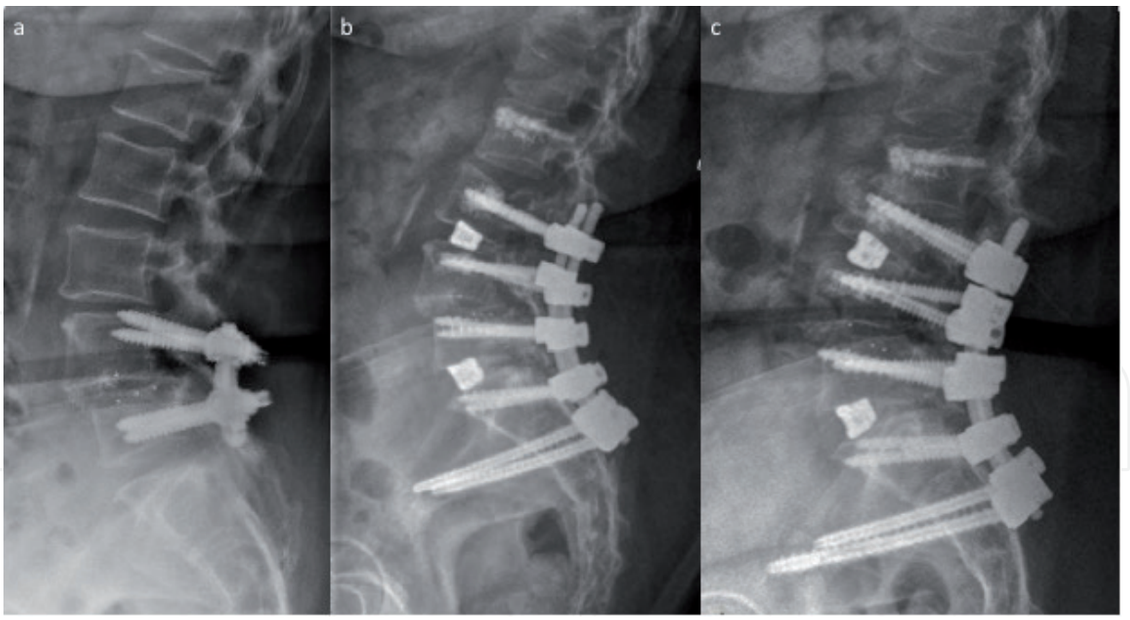

Figure 6.
Lateral standing X-rays of an obese osteoporotic patient who had previously undergone an L4/5 circumferential fusion complicated by global sagittal imbalance, pedicle screw malposition causing right L5 radiculopathy and dysfunction as well as progressive type 4d ASD of L3/4 causing critical central stenosis (a). They underwent revision of their instrumentation with L3-S2 fusion and correction of their sagittal balance, supplemented with cranial vertebroplasty (b). However, the patient developed type 3d ASD (c).

Most are thought to occur because of poor bone stock and therefore mimic osteoporotic compression fractures. However, end-plate devascularisation, either from a direct injury to the end arteries of the endplate by subcortical screws or by damage of the nutrient vessels to the endplate by anterior dissection can occur [17]. Similarly, with interbody devices the excessive load induced by rigid constructs can surpass the endplates' biomechanical tolerance and induce fracture.

Treatment depends on the severity of symptoms and the degree of compression of the vertebrae. In asymptomatic patients bracing to prevent further compression and bone supplementation may need to be considered if there is poor bone stock. In symptomatic patients amendable to operative intervention, treatment should consist of:

3a. Treat as osteoporotic compression fractures. Bone supplementation ± bracing. If metalware protrudes into the adjacent IVD consider deformity correction with increased fixation.
3b. Bracing. If metalware protrudes into adjacent IVD consider deformity correction. While the cause is endplate vascular compromise, there is to date no evidence that changing the surgical technique for the extension of fusion will reduce the risk, however surgeons should consider avoidance of cranial endplate compromise if possible (e.g. cortical trajectory screws, sublaminar bands or hooks).
3c. Deformity correction with extension of fusion and avoidance of a rigid interbody device at the most cranial fusion level.
3d. Treat as osteoporotic compression fractures. Bone supplementation ± bracing. If metalware protrudes into adjacent IVD consider deformity correction with extension of fusion and avoidance of both cranial endplate compromise and a rigid interbody device at the most cranial fusion level.

2.4 Type 4: IVD failure

This is a common form of ASD and is most commonly seen as a late complication of fusion (**Figure 7**). It may be related to inherent disc degeneration that would have occurred whether fusion was performed or not. However, it may also be caused by:

4a. Acute hyper-load which presents as an acute IVD prolapse.
4b. Diffusion insufficiency and/or chronic excessive loading which presents with progressive IVD desiccation.

Hyperload is caused by the rigidity imparted by the fusion. Typically, with circumferential fusions at the most cranial level this causes acute hyperload and acute disc prolapse. In contrast, isolated posterior or isolated interbody devices cause chronic overload as the fusion develops and micromotion of the fusion construct reduces. This causes progressive disc failure of the adjacent level (**Figure 8**). Similarly, implants that induce rigidity of the end-plates, such as subchondral pedicle screws limit the usual motion of the endplate. This motion, which mimics that of a trampoline, aids diffusion of nutrients into, and waste products out of the IVD. Thus, limiting its motion affects disc health by affecting its nutrient supply.

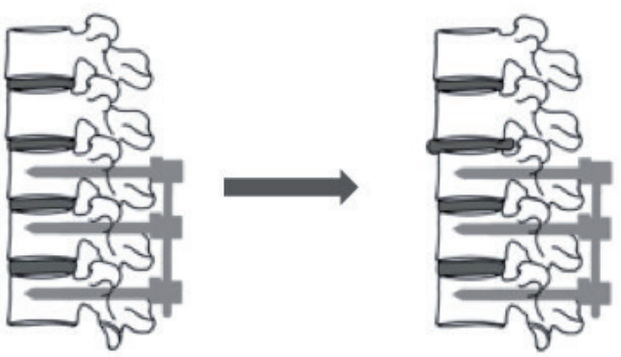

Figure 7.
Type 4 failure (IVD failure).

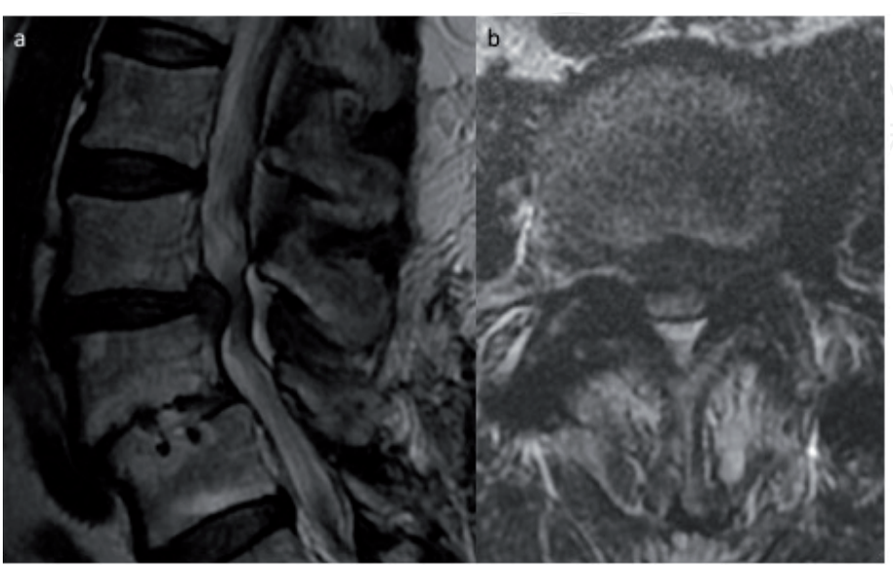

Figure 8.
Sagittal (a) and axial (b) MRI sequences of a type 4b failure above a L4/5 circumferential fusion. The IVD prolapse caused central stenosis and neurogenic claudication.

The treatment of this form of ASD depends on the symptoms and is the same for both 4a and 4b:

a. If neural compromise without instability: decompression alone.

b. If neural compromise with instability: decompression and single level fusion.

c. If discogenic pain or instability: single level fusion.

In patients with malalignment, deformity correction should be considered.

2.5 Type 5: facet failure

This form of ASD occurs when the facets joints fail, usually through hypermobility in the early stages and degeneration in the later stages (**Figures 9** and **10**). Hypermobility may occur in anterior procedures due to excessive stretch from oversized interbody devices, but this affects the motion segment that is fused and is therefore rarely a symptomatic problem once fusion occurs, but may cause long-term symptoms in disc arthroplasty.

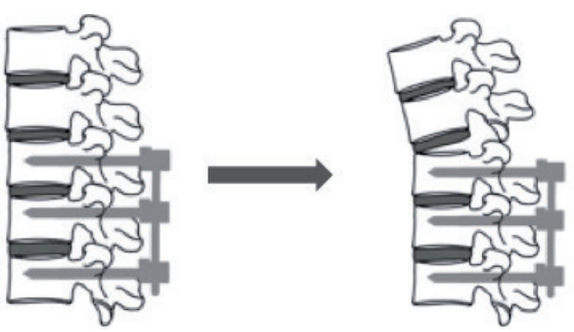

Figure 9.
Type 5 failure (facet failure).

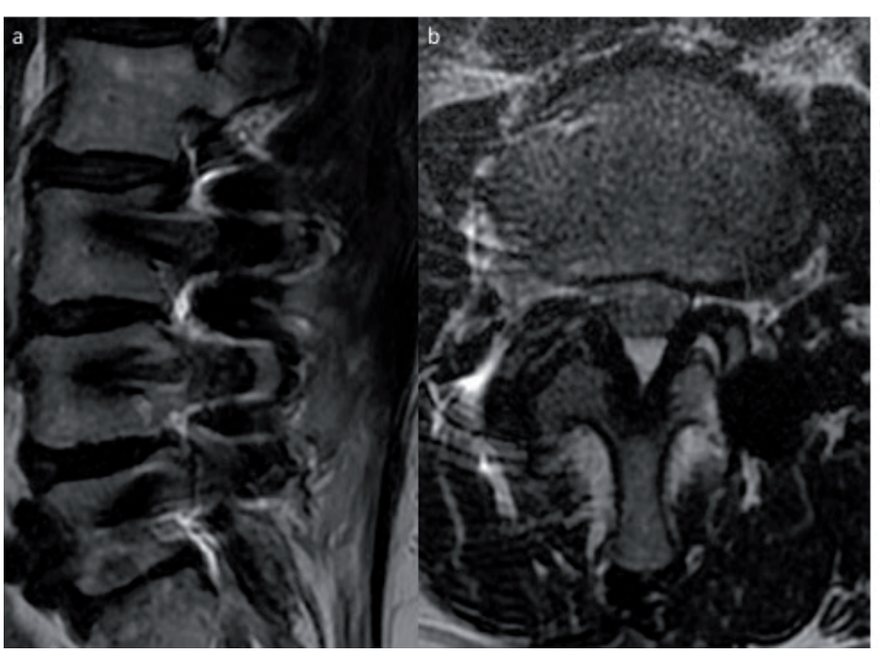

Figure 10.
Sagittal (a) and axial (b) MRI of a patient with a previous L3–5 postero-lateral fusion with left facet dysfunction causing a dynamic rotational deformity and unilateral retrolisthesis of L2/3 causing foraminal stenosis.

Type 5 failure is therefore predominantly seen with posterior instrumentation. The cause of type 5 failure is usually multifactorial and is classified as follows:

5a. Extensor mechanism dysfunction.
5b. Malalignment.
5c. Devascularisation and denervation of the adjacent facet joint.
5d. Metalware impingement on the adjacent facet joint.
5e. Combination.

To understand type 5 failure, one needs to appreciate the anatomy of the posterior spine. The extensor musculature acts to lordose the spine and is a dynamic control of spinal posture. It is innervated by posterior branches of the dorsal ramus, which run with the posterior vascular supply of these muscles, adjacent to the pars and is therefore at risk with the lateral dissection necessary for posterolateral fusion and standard pedicle screw insertion.

The extensor ligaments, namely the interspinous and supraspinous ligaments, and ligamentum flavum act as static restraints to kyphosis. In contrast, the intertransverse ligaments predominantly restrain lateral flexion. The facet capsule restrains excessive motion of the facet joint, particularly kyphosis. The facet joint itself is innervated and supplied by nerves and vessels that run with the dorsal muscular supply and are therefore at risk during posterolateral fusion and the dissection necessary for the insertion of standard pedicle screws.

We believe extensor mechanism dysfunction is caused by dysfunction of the dynamic or static restraints to segmental kyphosis. Dynamic restraint damage is caused by direct posterior musculature trauma and/or denervation and devascularisation of the paraspinal musculature most commonly induced by multi segment posterior dissection. This causes extension weakness, which results in adjacent segment kyphosis with load. Static restraint dysfunction is caused by transection of the cranial inter- and supraspinous ligaments or most cranial spinous process during the index procedure. The adjacent segment then relies on the ligamentum flavum and facet joint capsules as static restraints. Thus, adjacent flavectomy or direct capsular injury further disables the static restraints. Damage to the adjacent intertransverse ligaments is rarer, because the ligaments at risk are usually incorporated into the fusion, and the lateral IVD capsule offers significant restraint to lateral flexion. However, if a cranial transverse process fracture occurs the adjacent intertransverse ligament is affected and that increases the load on the lateral IVD capsule and may predispose to coronal failure.

Malalignment, particularly sagittal imbalance, puts excessive strain on both the dynamic and static restrains. This is particularly important if there is already dysfunction of the extensor mechanism, as the additional load induced by malalignment needs to be compensated for by the extensor mechanism.

The facet joints themselves are also commonly injured with posterior instrumentation. This is caused by the dissection necessary to insert posterior instrumentation, particularly standard pedicle screws through a midline approach, which involves far lateral dissection with stripping of the soft tissue from the posterior facet capsule and exposure of the pedicle entry point, which damages the neurovascular supply of the facet joints and extensor musculature.

Percutaneous insertion of the most cranial posterior implants is therefore preferable if possible, because this limits the degree of dissection necessary for insertion of the metalware, reducing the risk of neurovascular injury to the extensor mechanism and facet joints.

Metalware impingement on the adjacent facets is also common with pedicle screws, with estimates of up to 60% of pedicle screws breaching the facet [18–20].

Furthermore, even without facet joint breach, impingement can occur of the adjacent inferior articular process on the pedicle screw or rod with spinal extension, driving the adjacent level into kyphosis.

The treatment of asymptomatic patients or those not amenable to operative intervention remains non-operative. However, the treatment of symptomatic patients amendable to operative intervention is as follows:

5a. Single level extension of fusion, with protection of the extensor mechanism. This may involve the use of interbody fusion from anterior or lateral approaches, or a posterior approach with cortical trajectory screws or percutaneous insertion of cranial pedicle screws, with protection of the ligamentous restraints to kyphosis.
5b. Deformity correction and extension of fusion.
5c. Single level extension of fusion, with protection of the adjacent facet, often with anterior or lateral approaches, or a posterior approach with cortical trajectory screws or percutaneous insertion of pedicle screws.
5d. Dependent on facet function

- Facet non-functional: single level adjacent fusion.

- Facet functional: remove or reposition metalware depending on fusion of instrumented levels.

5e. Deformity correction with extension of fusion and avoidance of extensor mechanism and adjacent facet injury.

This classification broadly classifies ASD into anatomical and pathological groups, in order to further our understanding of its aetiology and treatment. However, as with any classification, it has limitations. Some patients with ASD fit more than one category and others fail in an atypical way. In such cases, the causes may be multifactorial and therefore the treatment may differ from those proposed in this classification system. Clinicians should be aware of these nuances and treat patients accordingly.

Understanding this anatomical and pathological based classification system allows treating clinicians to limit the modifiable risk factors for ASD after thoracolumbar fusion. By optimising bone quality preoperatively, the surgeon reduces the risk of bone failure, such as type 1a, 1c, 2a, 2c, 3a and 3d. In addition, ensuring spinal alignment and balance, reduces the risks of all failure mechanisms. Furthermore, ensuring adequate fixation at the time of operative intervention surgeons will reduce the risk of type 1b and 1c failure. However, this must be achieved without complete rigidity, which imparts excessive load through the cranial endplate and adjacent IVD motion to type 3c and type 4 failure. In addition, posterior dissection should be limited to avoid type 5a, 5c and 5e failure and the most cranial implants should avoid damage of the cranial endplate causing type 3b failure and the adjacent facet causing type 5d failure. Abiding by these principals reduces the risk of ASD, but does not prevent ASD because there remain non-modifiable risk factors for the condition.

Similarly, if ASD does develop, the clinician should critically appraise the causes of the failure, to ensure that optimal treatment is provided. In the setting of bone failure, bone supplementation should be provided, however, these alone fail to resolve the problem and therefore bracing or further surgical intervention is necessary. In all revision procedures, spinal alignment and balance should be considered. Abiding by the same principals as discussed above, clinicians will reduce the risk of recurrence.

Lastly, this classification is the first to describe the pathophysiology of ASD and therefore provides a framework on which further work can expand the prevention and treatment of this increasingly common condition.

In conclusion, this anatomical and pathological based classification system allows treating clinicians to limit the modifiable risk factors for ASD, understand the causes of ASD and offer a treatment algorithm for ASD. Furthermore, understanding this classification and the causes of failure allows clinicians to not only diagnose and treat ASD, but also offers a clearer understanding of what modifiable factors should be addressed during the index procedure. In addition, it illustrates that new technologies to eliminate modifiable risk factors are necessary, which should stimulate research and industry to find solutions to this common problem.

Acknowledgements

The authors are grateful to Glynny Kieser for her editorial input, and Amelie Poilliot for her illustrative input.

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