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GRAND ROUNDS

Expert's comment concerning Grand Rounds case entitled "Multiple revisions in a L2 burst fracture in a suicide jumper: a retrospective analysis of what went wrong" (by P. Gahr, S. K. Tschöke, D. Haschtmann, Christoph-E. Heyde)

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This case description is a complex trauma case in a 35-year-old female patient. This patient had her index surgery performed at an outside facility and a first revision surgery 12 months later in the author's department. The first time a purely posterior instrumentation from T12 to L3 was carried out in this multilevel fracture including T12, L1 and L2. While the L2 fracture was considered a complete burst fracture with 80% of canal compromise and consecutive paraplegia, the fractures of T12 and L1 were considered as stable fractures. When 12 months later the construct failed into kyphosis with new onset neurologic findings, a combined anterior and posterior surgery was done between L1 and L3 to reconstruct the anterior column with an expandable cage replacing L2, augmented with an anterior rod system. Simultaneously the posterior rod system was replaced by a fracture system. Seven months after the second operation, there was again a revision done with again a combined approach with posterior stabilization from T11 to L4 and an anterior support with a partial corpectomy of L1 and complete corpectomy of L2 replacing these defects by a long Mesh cage. Again 7 months later due to infection an additional surgery was necessary. More than 6 weeks later a final revision with a partial corpectomy of T12 was done. Therefore, this patient had five surgeries for a fracture, which seems in the CT reconstruction to be a fracture of T12, L1 as well as L2. L2 is a complete burst fracture with a significant posterior fragment, however, with a disruption of the posterior elements, which speaks rather for a B-injury than a simple A-injury. The vertebral body of L1 is not so sure a stable fracture, since at least in the CT reconstruction the vertebral body of L1 has a wedge configuration. Unfortunately there are no other X-rays available to understand this fracture, however, looking at the Fig. 2, where a posterior stabilization and reduction has been done it seems that the L1 vertebra is reduced, which speaks for the fact that there has also been a disruption between T12 and L1 for rendering this level unstable.

The crucial question is to understand why this injury has taken this complex course and has not been treated sufficiently with the primary posterior surgery. Obviously between the postoperative X-rays and the 12-month follow-up X-rays there is increased kyphosis, the rod has rotated, leading to a different angle between the vertical bar and the screws. This naturally led to the collapse of the disk space between L1 and L2 with a secondary retroprotrusion of the

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posterior wall of L2. I think one of the relevant reasons why this posterior instrumentation was not sufficiently stable is the fact that this instrumentation is not a trauma instrumentation but an instrumentation, which is usually used for degenerative spine and or deformities. This instrumentation has a lack of angular stability between the pedicle screw heads and the rod. That means that overtime movement between the rod and the screw head will occur, leading to loss of correction and maintenance of instability in the system. It was obviously clear at 12 months follow-up that an additional surgery was necessary due to the significant compression of the dural sac by the secondary retropulsed posterior wall of L2. At the time the surgeons assumed that the fractures in T12 and L1 had consolidated in the meantime, and therefore they thought they could do a short stabilization from L1 to L3 with a combined anterior/posterior procedure. They removed the whole posterior instrumentation, reapplied an internal fixator with a stable angle screw situation and used an expandable cage to replace the deficient anterior column at the level of L2. In addition, they used even an anterior instrumentation for additional stability, which is difficult to understand, since the additional posterior internal fixator (USS fracture module) allows a sufficient compression of the anteriorly placed cage by applying the tension-banding concept. It can be assumed that this fixation made a completely rigid construct of L1–L3. To some extent the T12/L1 segment (posterior elements?) has not been sufficiently stable to withstand this rigid block of fixation. We can observe an angulation between T12 and L1 in Fig. 5, and an impression of the superior endplate of L1 with a loss of height of the vertebral body of L1. This speaks for a progressive deformity, most probably due to some weakness of this vertebral body (posttraumatic osteoporosis or necrosis?) and an insufficient discoligamentous complex at the T12/L1 segment. Seven months later a third surgery is performed and 3 h after the surgery is finished the patient deteriorated her neurology and became incomplete paraplegic with a T11 sensory level. The explanation of the authors was a vascular damage of the cord at this level, and the patient was treated with pharmaceutical agents and transferred to a paraplegic center. Why this patient developed a spinalis anterior syndrome as the authors suspected is not clear. This third surgery was an extensive anterior surgery by removing the expandable cage at the level of L1 and doing a vertebrectomy of L1 inserting a very long Harms cage between T12 and L3 and an anterior rod system as well as a posterior stabilization from T11 to L4. The question arises as to whether the artery of Adamkiewicz (which varies anatomically in its origin) has been injured by the surgery or the cord has been manipulated during the surgery. Otherwise it becomes difficult to explain this neurological damage. In Fig. 6 after this major third surgery, we can see that the vertebral body of T12 seems to have a split, respectively a fracture of the superior endplate just above the screw. This supports the fact that T12 was obviously also injured in a more extensive way as primarily assumed from the images. Correctly the authors brought their fixation up to T11.

The authors mention it is unlikely that the onset of the paraplegia has anything to do with a direct surgical damage, since the late onset of the paraplegia can almost exclude this. The postoperative imaging did not demonstrate an external compression. Therefore a vascular damage has been assumed. The authors do not describe the exact neurological picture, although they conclude that the patient had a spinalis anterior syndrome. The spinalis anterior syndrome has a quite distinct neurological finding; therefore it is open for discussion whether this was really the case.

Later the patient developed an infection of her surgical site, and a debridement that was done in an outside facility was unsuccessful. Finally the fifth operation was performed, including a new debridement and stabilization now with a new anterior cage. It seems also that the anterior instrumentation has been removed, however, the posterior instrumentation remained.

In conclusion, the relevant question is, whether all this surgery could have been avoided with a primary surgery addressing the total character of this injury. From what is presented here it is not clear, what exactly the primary injury was. The sagittal reconstruction in Fig. 1 of the CT is insufficient to really make a judgment about the real character of this multisegmental injury. I have just one horizontal section out of L2, which is also not representing the true character of this injury. I believe that there was a distraction injury at the level of L1/L2 maybe even with rotation since the vertebral body L1 has not the same dimension as T12, respectively L3, which leads to the assumption that there is some rotation in-between. In addition, there must be an injury at the level of T12/L1, since the vertebral body of L1 has a wedge configuration, and it seems to me that the endplate superiorly of T12 also has a lesion, which later on in CT reconstructions has been confirmed. This injury should have been evaluated more precisely to be sure about the real extent of these different injuries involving several segments. I believe, however, that assuming the different injury levels a posterior primary surgery as it has been intended would have been sufficient; however, the posterior surgery should have been more differentiated that it has been. It cannot be just a distraction of the anterior column as we can see creating a flat area from T12 to L3. In fact, the construct should have used a fracture system from T11 to L3 with compression between the segment T11/T12, and T12/L1 and with lordosing forces between L1 and L3. One should have avoided any

distraction between T12 and L1. With a stable angle system like the internal fixator this multilevel fixation properly applied could have been by far sufficient to stabilize this complex injury, and would have probably never necessitated to end up with this extensive surgery anteriorly. It needs to be said that after all these pitfalls and complications it is much easier to say what should be done than at the beginning when we do not know what the future history could be. The take home message is that in my view such surgery should only be done when we really understand the extent and the character of the injury at each different level of the spine. This probably should have been done right away in a center specialized in spine surgery and not in a local peripheric hospital.