

Influence of blood supply on fracture healing of vertebral bodies

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Introduction: The relevance of blood supply for bone fracture healing has been discussed throughout the literature, using scaphoids as probably the most referred to. But, there is so far nothing known about the relevance of blood supply for the vertebral fracture healing. Indeed, even the AO guidelines are not dealing with this issue.

Materials and Methods: A prospective cohort study of 107 patients was run from January 2016 - December 2016, with 54 male and 53 female patients, who were treated for traumatic vertebral fractures of thoracolumbar spine using posterior stabilization only. The average age was 67 years and the follow-up 12.3 weeks. The total number of damaged vertebrae was 129. We analyzed the fracture morphology, and measured the vertebral bodies in all three dimensions, with five reference planes. The progress of vertebral deformity in time measured before and after the surgery, was correlated with the potential damage of the main vascular canal in the rear of each vertebral body. The bone pattern and morphology were analyzed in detail as well. Pathological fractures were not taken into our consideration.

Results: The overall deformity progression of vertebral bodies in the fractures with morphologically damaged blood supply was in all measured dimensions significantly higher than in the fractures with supposedly maintained perfusion. The osteoporosis played its role as well, but only with medium effect-size compared with strong effect-size of the vessel canal damage (Cohen). The combination of the both factors (damage to the vessel canal together with osteoporosis) showed also a strong correlation with a relevant deformity progression (Evans), but not much different from the vessel canal damage alone. With regards to the relevant changes of the vertebral body dimensions / volume we found relevant changes in 52% of all fractures (SD 0.5017) generally, for the subgroup with the canal damage in 84 % (SD 0.3691), with strong correlation (Evans, 0.7721). In the group of fractures with maintained perfusion we found such changes in only in 5% of fractures (SD 0.2333).

Conclusion: For surgical decision making we should take mechanical fracture analysis and dynamic processes within traumatized tissue a part of whose is the blood supply and oxygenation into surgical consideration. The decisionmaking of anterior vs. posterior surgery for the cases with damaged vessel canal seems to have more attributes. In the elderly a simple vertebroplasty could pose an alternative.

Key words: fracture, vertebral body, vascular canal, deformity, ischemic atrophy

Vplyv cievneho zásobenia na hojenie zlomenín stavcov

Úvod: Vplyv cievneho zásobenia na hojenie zlomenín je v literatúre diskutovanou témou, azda najčastejšie sa publikácie venujú os scaphoideum, prípadne aj proximálnym zlomeninám femuru. Ohľadom vplyvu cievneho zásobenia na hojenie zlomenín stavcov sa však prakticky nič nevie, dokonca ani klinické odporúčania AO sa touto problematikou vôbec nezaobierajú.

Materiál a metodika: Prospektívna kohortová štúdia, od začiatku januára 2016 po koniec decembra 2016. Celkovo 107 pacientov, z toho 54 mužov a 53 žien. Pacienti boli operovaní výhradne dorzálnou stabilizáciou pre úrazové zlomeniny stavcov torakolumbálnej chrbtice. Počet zlomených stavcov bol 129. Priemerný vek pacientov bol 67 rokov a priemerná dĺžka pooperačného sledovania 12,3 týždňa. V čase sme analyzovali vertebrálnu morfológiu vo všetkých troch rozmeroch pomocou piatich referenčných rovín. Progresia deformácie stavca bola meraná pred- a pooperačne a túto dynamiku sme dali do korelácie s poraním hlavného cievneho kanála v dorzálnych časti postihnutého vertebrálneho korpusu. Analyzovali sme aj detailnú štruktúru kosti. Patologické zlomeniny sme v našej kohorte nebrali do úvahy. **Výsledky:** Zistili sme signifikantne väčšiu deformáciu tel stavcov pri morfológickom poškodení cievneho kanála, než ako to bolo v opačnom prípade, kde sa predpokladala zachovaná perfúzia. Rovnako hrala úlohu aj osteoporóza, ale len so stredne silným efektom, v porovnaní s výrazným pre poranenia cievneho kanála (Cohen). Kombinácia oboch uvedených faktorov vykazovala silnú koreláciu s progresiou posttraumatickej deformácie stavca (Evans), avšak bez výrazného rozdielu proti samostatnému poraneniu cievneho kanála. Výrazné zmeny tel stavcov čo do rozmerov a objemu sme našli celkovo pri 52 % zlomeninách (SD 0,5017), pre podskupinu s poraním cievneho kanála u 84 % (SD 0,3691), s významnou koreláciou (Evans, 0,7721). V podskupine so zachovanou perfúziou sme obdobné zmeny našli len u 5 % zlomenín (SD 0,2333).

Záver: Pre indikačné rozhodnutia treba zohľadniť jednak mechanickú analýzu zlomenín, ale aj dynamiku biologických procesov v rámci traumatizovaného tkaniva, súčasťou ktorej je cievná perfúzia, resp. zásobenie tkanív kyslíkom. Rozhodovanie medzi ventrálnou a dozálnou stabilizáciou zohľadňuje viacero atribútov, jedným z nich je predpokladaný vývoj kostnej organizácie v priebehu času. U starších pacientov by mohla byť alternatívou obvyčajná vertebroplastika.

Kľúčové slová: fraktúra, telo stavca, cievný kanál, deformita, ischemická atrofia

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Introduction

The relevance of blood supply for bone fracture healing has been discussed throughout the literature, using scaphoids (1) as perhaps the most referred to. Anyway, there was nothing known about the relevance of blood supply for the vertebral trauma until recently and even the guidelines of AO do not deal with this issue despite the meanwhile known facts.

For the blood supply of vertebral bodies, the biggest importance lies on paired segmental vessels, running on both vertebral sides dorsally from the main artery (i.e. aorta) through intervertebral foramina into the spinal canal (Fig. 1). Being there, they conjoin into the anterior spinal artery, which the particular vertebral branches come off, running ventrally into the adjacent vertebrae, entering the vertebral bodies through a vessel canal (Fig 2).

The anterolateral vessels do not seem to be that important for the vertebral oxygenation, since there's almost no canal showing their course within the vertebral body and they may supply the periosteum and anterior ligament. We found the contrast dye entry into the vertebral body only dorsally (Fig 1 and 2) in our cohorts.

In the literature, the dorsal vessel canal is being usually mentioned just marginally, as the entry point of basivertebral veins not be confused with fracture lines (4).

But indeed a damage to the relevant portion of this virtually only blood supply results in vertebral tissue ischemic atrophy and fracture deformity progression, even despite the stabilizing measures. Biological systems are complex and dynamic, thus difficult to understand. Simplified or mechanistic views could lead to problems afterwards.

Materials and Methods

We designed a prospective cohort study of 107 patients, run from January 1st – December 31st 2016, with 54 male and 53 female patients, with the average age of 67 years, SD 17.3 years and age median 72 years, who were treated for traumatic vertebral fractures of the thoracolumbar spine, with trauma history, operated on

Figure 1. Vessel canal, on the left with contrast dye

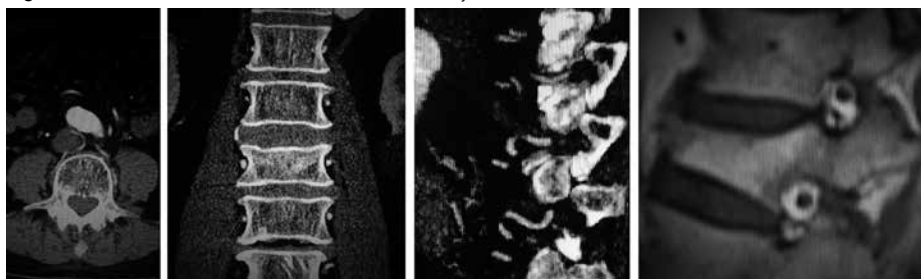


Figure 2. Course of segmental arteries

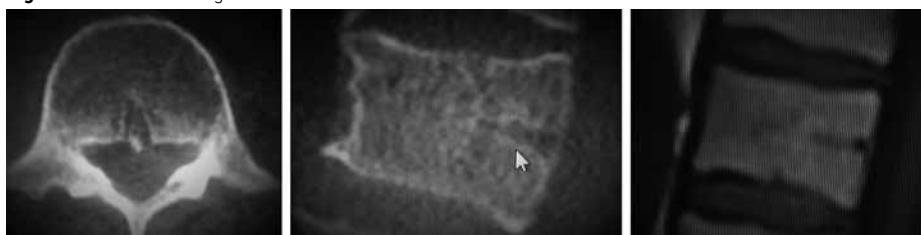
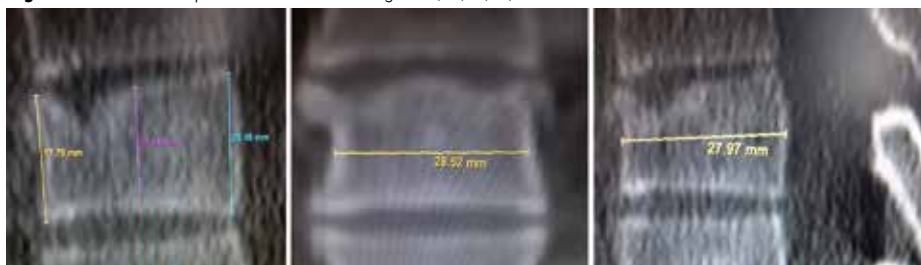


Figure 3. Reference planes from left to right: A, B, C, D, E



The A, B and C – lines shown on the figure above, and depict the three craniocaudal dimensions, the anterior wall, center and the rear wall in sagittal plane, drawn directly in the middle of the vertebral body.

routinely with posterior stabilization (USS2, SynthesDepuy, USA and Matrix, SynthesDepuy, USA).

The average follow-up was 12.3 weeks, with SD 7.67 and median of 9 weeks. The total number of fractured vertebrae was 129. Patients with anterior stabilization, the ones treated conservatively, metastatic or pure osteoporotic fractures without any trauma were not taken into our further considerations.

In all cases a CT-scan was performed (Siemens, 128-slice) for the primary diagnostics, with or without contrast dye, the latest depending on the trauma pattern of the particular patient. We analyzed the fracture morphology and measured the vertebral bodies in all three dimensions (Fig. 3) with five reference lines (A, B, C, D, and E). On purpose we left measuring of the Cobb-angle and soft tissues out, in order to focus on bone tissue solely.

Hereto we focused on the presence of the vessel canal and/or posterior processes for the orientation. Transversal dimension D was measured as the longest possible direct distance

between the concave sides of vertebral bodies. The dimension E reflects the anteroposterior distance in the sagittal plane, lying in the middle of the vertebral body. The progress of vertebral deformity in time measured before and after the surgery during the follow-up, was correlated with the damage to the main vascular canal in the rear of each vertebral body. The vertebral arcs showed in our timescale no dimensional changes at all, regardless the body injury type, and would not be taken into further consideration. For the fracture analysis we always checked the whole column segment and the whole vertebrae with their surrounding soft tissue.

As to the bone pattern and its morphology we performed detailed scrutiny of its density and structure, focusing firstly on fracture lines in all dimensional planes. Areas of comminution, distraction or compression within the bone were examined also. Since the vertebral anatomy shows individual variability, it was necessary to analyze each vascular canal individually; here we

Figure 4. Morphologic changes within vertebral bodies**Diagram 1.** Deformity progression in all measured planes in both patient groups

Planes	Affected perfusion (mm)	SD	Blood supply maintained (mm)	SD
A	2.6133	2.8183	0.0566	0.2333
B	3.6133	3.4480	0.3208	1.0335
C	1.2667	1.9955	0.0377	0.1924
D	3.2400	4.0631	0.1887	0.7610
E	2.0400	2.4242	0.1321	0.5203

took the ones of neighboring vertebrae as a reference.

The imaging programs used was Syngo.Via (Siemens) and MagicWeb (Visage Imaging). For the follow-up measurements, a plain radiogram was used in 19 patients (18%), in 88 of cases (82%) the same CT-scan (Siemens, 128-slice) as before was used. As the scale reference for measurements on plain radiograms we used the implemented hardware, with its precisely defined dimensions, and at the same time the after all constant pedicles.

In few cases a MRI scan was performed for more precise diagnostics, allowing us to see the extent of the injury to soft tissues (3), and to the cancellous bone.

A direct angiography of a particular vertebral body seems to have some technical difficulties and not the less an invasive aspect, so that we decided to focus on the above mentioned indirect morphologic signs inside of the cancellous bone, indicating the vessel canal trauma (Fig. 4), such as fracture lines or bone compression reaching it.

Considering the irregular and individual anatomy of particular vertebral bodies, varying in their form and volume from one to another, and viewing the per se dynamic and irregular deformity progression in the follow-up, we found virtually no equation for the vertebral bodies' volume. Thus, we took approximating arithmetics for calculation of ellipsoid volume ($V=4/3\pi abc$) and at the same time focused on tissue atrophy signs.

As mentioned, exclusion criteria were all kinds of pathological fractures, fractures treated with anterior surgery and conservatively treated patients.

For statistical analysis we used the program BiAS 11.1, with Pearson-correlation-testing and Welch-test.

Results

In 73 fractures, i.e. 56.5% of all fractures, we found morphological signs of vessel canal damage by the first CT-scan already, in the remaining 54 fractures, i.e. 42% there were no such signs to be found. In two cases (1.5%) with primarily unclear fracture morphology we could find signs of vessel canal trauma in the further diagnostics, as rarefaction or sclerosis in the area of the vessel canal. Thus the total number of the vertebrae with a supposedly damaged canal was 75, making up 58% of the whole number.

The follow-up time for the both subgroups, with and without the canal damage, was about 12.9 weeks, with SD 8 and median 10 weeks for the group with an affected damaged vessel canal and 11.3 weeks, SD 7 and median of 9 weeks in the another one.

Hereto differed the pain scores of the both groups significantly (Cohen, 0.2591) in the first one, with affected damaged canal, we found 4.6 points in average, SD 1.98 and median of 4 points, and on the other hand 5.18 points average, with 2.6 SD and median 5.5 points in the group with an presumably intact perfusion. The average pain score of the whole cohort was 4.8 points, with SD

2.22 and median of 5 points. The both groups were different as to their average age, 71.8 vs. 59.4 years, and also was the osteoporosis prevalence, here overall 41%, further 59 % vs. 15 % respectively.

The gender ratios were different also, with 57% female patients in the first, and 35% in the second group with maintained perfusion. We found a weak correlation between the female gender and damage to the vessel canal (0.2065, Evans). About 56% of all fractures happened around thoracolumbar transition and this number is consistent with the common literature data. As to the fracture type, almost 80 % of the injuries were of types A3 and A4 respectively (56% A3 and 22% A4), hence making up a vast majority.

As seen on the Diagram 1, the overall deformity progression of vertebral bodies with morphologically damaged affected blood supply was in all measured dimensions significantly higher than in the group with supposedly maintained perfusion, with very strong effect size for the vessel canal damage (Cohen, A 1.1809, B 1.2085, C 0.8010, D 0.9681 and finally E 1.0108) in the Welch-Test.

As quite a paradox, the most resilient part to the secondary shape loss of the vertebral body seems to be its disputed dorsal wall (dimension "C"). As to the changes of the vertebral body volume we found a relevant shift (over 4mm³ or over 1mm in any dimension) in 52% (SD 0.5017) generally, for the subgroup with the canal damage in 84% (SD 0.3691), with strong correlation (Evans, 0.7721) of these parameters. In the group with supposedly maintained perfusion, there were such changes in just 5% (SD 0.2333) to be found. Out of 75 cases of the group with supposedly affected perfusion, 24 (32%) of them developed vertebra plana, with excessive bulging to the sides and complete shape loss, represented by "volume gain", with loss and rarefaction of trabecular structure within the bone (Figure 5).

The osteoporosis played a role as well, i.a. with medium effect-size (Cohen) for the A 0.6682, B 0.6654, C 0.5618 and E 0.6086, and even with strong effect size for the dimension D 0.8456 in the Welsch-test. As to the relevant vertebral

Figure 5. Vertebra plana

volume changes, there was just a weak correlation with the osteoporosis found (Evans, 0.3879). A moderate, but significant correlation of osteoporosis with female gender was found also (0.4583, Evans). As to the development of vertebra plana we found weak, but significant correlation with osteoporosis (Evans, 0.2779). However all these cases were with damaged vessel canal and this fact should be noticed. The combination of the both factors mentioned above, i.e. damage to the vessel canal and osteoporosis, showed also strong correlation with a relevant deformity progression (Evans, 0.7742), anyway not much different from the one of the vessel canal damage alone. We found just poor correlation between the relevant changes and fracture type (Evans, 0.086667) and none as to the vertebral etage (Evans, -0.013333). As to the distribution, we found 75% of all fractures near of the thoracolumbar transition from T10 to L3. Between the T11 and L2 vertebrae about 60% of the fractures were found. The Diagram 2 is depicting the particular fracture type prevalence, the type A3 and A4 making up almost 80%.

Discussion

Data above show clearly the influence of blood perfusion for the bone healing, in this case of the vertebral bodies. This attribute of trauma must be emphasized. Despite its simplicity, there's no plausible study to this issue and no guidelines address this explicitly, e.g. in the indications criteria. In our opinion, the damage of the vascular canal or of its relevant portion and thus supposedly compromised blood supply should be taken into surgical consideration. We found a relevant deformity progression

Diagram 2. Fracture type (AO) prevalence within our cohort

Fracture Type	Total Amount
A1	11
A2	5
A3/A4	102
B1	4
B2	3
B3	2
C2	2

in our cohort, despite the posterior stabilization.

For the reposition loss or deformity progression respectively, main changes for both groups were found in the middle of vertebral bodies (reference line B). In the group with damage to the canal it was on the ventral wall 50-fold-, and in the middle of vertebral body about 10-times more than in the other group, even in individuals without osteoporosis. Compared the subgroups of vessel canal damage, with and without osteoporosis, there seems to be a minor influence of the osteoporosis in reference planes A,B,C,E and a moderate in plane D (Cohen), but not more.

Thus we assume that the osteoporosis plays its role just as a cofactor, and the pivotal relevance has the maintained or compromised blood perfusion.

The fracture type or height within the spinal column played just a small or no role altogether. Trauma to posterior longitudinal ligament could also signify damage to the particular vessel segment, thus leading to deformity progression (3), however, we don't believe the simple mechanistic view could give a full answer. Moreover, in some distraction fractures we found no atrophy or deformity progression at all, despite clear mechanic instability of the whole region, as seen on Fig. 6, referred to us from oyster hospital.

The deformity progression in millimeter scale, e.g. in the first group in "B" of about 3.6 mm, must be seen in context of previous, mostly swift dorsal stabilization, and so seems this number to be quite high.

We did not measure the deformity in angles, but decided for the reference planes as a more precise alternative, since the wedging degree would not reflect the vertebral substance loss precisely.

Figure 6. An example of complete mechanic instability, with no atrophy signs at all

Any measurement of kyphosis or lordosis angles would have to count with intervertebral discs also, thus bringing in another factor to view at.

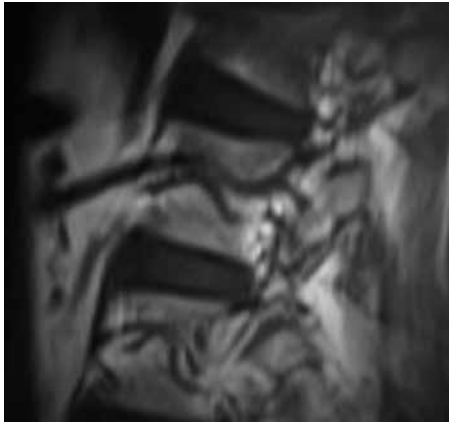
We chose to analyze the patients treated with dorsal stabilization only, in order to pinpoint the changes in the "slow time camera" and get rid of the problem with patient weight or local mechanic instability (6) as possible cofactors. The fractured vertebrae treated with ALIF would be for our purposes of no use and the ones treated conservatively developed changes sometimes much too rapidly for being somehow plausibly interpreted. These both groups were not further considered.

As to the implemented hardware we use routinely polyaxial systems, because of their simplicity and low invasivity. In few cases we used the monoaxial systems, but we found no difference between the two (5). The loss of vertebral substance with consecutive reposition loss followed in our group the pattern of the damaged vessel canal.

The vertebral deformity develops mostly in the sagittal plane, known as wedged vertebra and progressive wedging was despite many times irregular fracture morphologies virtually never seen in the coronal plane in our cohort, but indeed, this kind of deformity can be found occasionally. In the case of vertebrae with osteoporosis we found vertebral widening as well, anyway, almost always to both sides symmetric one.

Regardless multiple fracture lines showed the rear vertebral wall out of all measured planes the smallest deformity progression (1.5mm in average). The

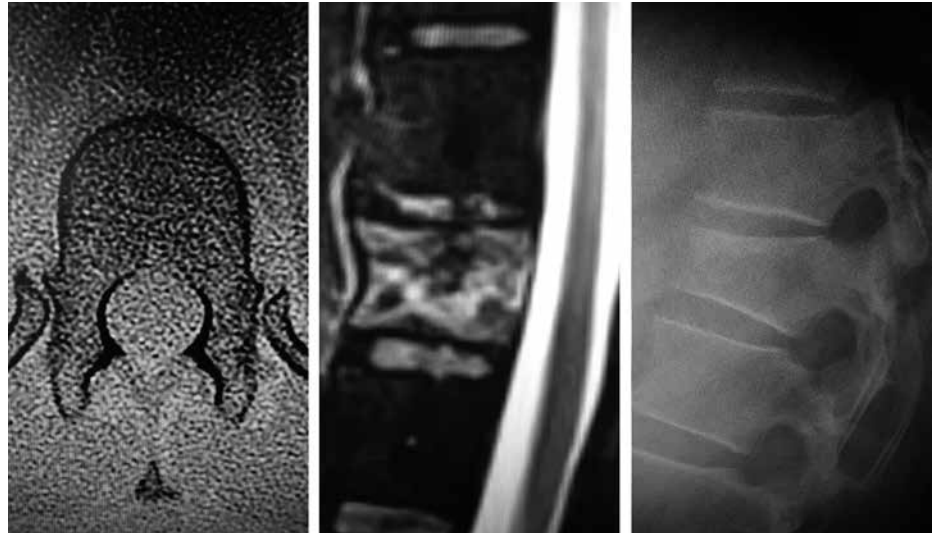
Figure 7. Course and branches of segmental artery



dorsal wall bears the main axial pressure and should give way the most. It even carries remarkable loads in the case of dorsal stabilization, since mostly a reposition is performed during the surgery, shifting the main compression vectors inside the vertebral body dorsally. The explanation could lie in the tissue oxygenation and not in the pure mechanics, although the recent work of the group from Leipzig seems interesting (5). The in time progressive sagittal wedging could relate to the decreasing oxygen blood saturation in context of the postero-anterior direction of blood flow within the vertebral body, and not only to pure mechanics of the fracture. Interestingly, main changes developed within first three weeks after trauma.

McCormack classification (2) based on morphological criteria, corresponds grossly with our findings, since the more the vertebral body is being damaged, the higher the probability of vessel canal lesion would be. Anyway, solely mechanistic view is in our opinion not the right explanation, though seen reasonably it must play its role also. For proper decision making one should rather keep in mind dynamic processes within traumatized tissue, an aspect of whose is the oxygenation. The fractured vertebral bodies may pertain most of their mechanical stability in vitro or even in vivo shortly afterwards, yet this does not have to be the case during the whole process of fracture healing. As already mentioned, the adjacent ventrolateral vertebral branches do not seem to be that important for the oxygenation, since there's almost no canal showing their course within the vertebral body. They

Figure 8. First two pictures show initial status, the last an X-ray study two weeks after



may supply the periosteum and anterior ligament or just smaller front portions of vertebral bodies. Anyway, they immanently must suffer some injury as well, because almost all A-fracture types go with some kind of trauma to the ventral wall. Thus their damage could be virtually counted on, making the rear vessel canal even more important (Fig. 7).

Accidentally we could see some damage to the segmental artery itself. Indeed, this happens mostly unilaterally, thus making no concerns as to the vertebral perfusion at all. An injury to both of those vessels together was not seen in our patient group, its real clinical effect remains questionable considering anastomotic commissures further on.

The rear vertebral vessel, on the contrary, can be viewed at as an end-vessel with no compensation from any proxy around. The vertebral arcs showed no changes at all, neither in the group of conservatively treated patients, nor in those operated on. An explanation of another undamaged arterial branch with sustained perfusion comes here near.

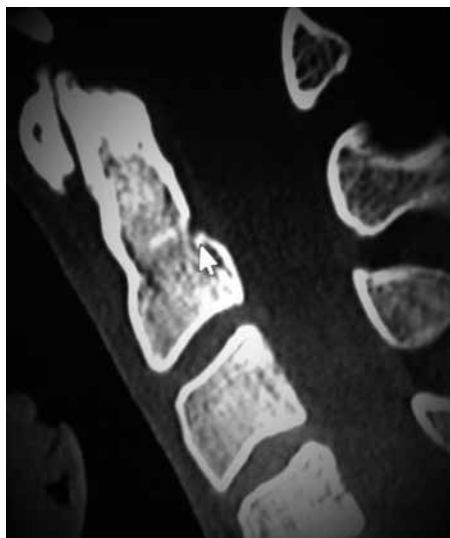
The differences in gender, age and osteoporosis seem have one mutual explanation. The otherwise thick and strong rear vertebral wall declines in lifetime, hence getting more vulnerable, and vessel canal included. Anyway, we believe, the mechanical fracture stability or instability must play some role in the clinical praxis also.

The different pain scores seem to be on the first look rather in support of ischemia antithesis. Anyway, solely to

the periost bound pain receptors are in case of ischaemia within the vertebral body virtually out of stimulus. The outer periost gets its vessel branches from the outside running segmental arteries (Fig. 7), far before they enter the vertebral bodies. We consider the higher pain scores in the group of maintained perfusion to be the result of a higher pressure within the compartment of the vertebral body. This otherwise common phenomenon of posttraumatic hyperemia would be in the case of missing blood supply diminished.

As already mentioned, in two cases (1.5%) primarily unclear fracture morphology was found. The signs of vessel canal trauma were found in the further diagnostics only, in form of bone resorption or sclerosis around and within the area of the vessel canal. In another case, not enrolled into our study, there were no fracture lines or any indirect signs of bone trauma or vessel canal damage found at the CT-scan, with as a bone bruise described diffuse edema of the whole vertebral body at MRI. Interestingly, this otherwise conservatively treated patient, developed progressive wedging within 2 weeks (Fig. 8). Whether we deal with a compartment syndrome within the vertebral body remains unclear. Anyway, we would not assume any mechanical instability. This particular case pinpoints the potential diagnostic trap of our method, that the indirect signs at the CT scan must not ultimately reflect the whole real damage to the vertebral body.

Figure 9. Entry point of adjacent vessel at the basis of dens axis



Further on, we found a vessel canal on the rear side of dens axis basis (Fig. 9), which may explain poor results of conservative therapy of type II fractures, in contrary to the fractures of type I and III. To keep our message short and concise, we chose to focus on the thoracic and lumbar vertebral bodies only. To put it honestly, our findings bring-in more questions than answers.

Maintained blood supply is essential for fracture healing and surrounding soft tissues and its relevance seems to be somehow underestimated. In case of fractures on extremities the eventual previously compensated ischemia manifests itself usually as a critical one, leading to poor bone healing and many times soft tissue necrosis.

As to the spinal column we advocate anterior rather than posterior stabilization for the cases with damaged vessel canal, the vertebroplasty could pose an alternative in the elderly. Anyway the decision making is not always a question of simple solutions. Hence primary posterior surgery and tight follow-up is often being practised, with anterior approach in case of deformity progression.

Conclusion

For decision making we should take mechanical fracture analysis and dynamic processes within traumatized tissue a part of whose is the blood supply and oxygenation into surgical consideration. We recommend anterior rather than posterior stabilization for the cases with damaged vessel canal, the verteb-

roplasty could pose an alternative in the elderly.

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