TRANSPEDICULAR DECOMPRESSION FOR PAINFUL AO SPINE TYPE A1 THORACIC AND LUMBAR COMPRESSION FRACTURES: CASE SERIES STUDY

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Objective. To analyze dynamics of vertebrogenic pain syndrome and quality of life after transpedicular decompression in patients with depressed fractures of the thoracic and lumbar spine.

Material and Methods. An observational prospective pilot study included 10 patients with AO Spine type A1 fractures of the thoracic and lumbar spine operated on in 2020—2021. All patients underwent transpedicular decompression. Severity of pain syndrome according to VAS, ODI score, and the magnitude of apical kyphosis were studied in the preoperative period and at 3 day, 1, 3, 6 and 12 month postoperative follow-up.

Results. The age of patients was 35—70 years (median 46). The ratio of men and women was 1 : 4. By localization, the fractures were distributed as follows: T10 — 1 patient (10 %), T11 — 1 patient (10 %), L2 — 2 patients (20 %), T12 — 3 (30 %) and L1 — 3 patients (30 %). Statistically significant regression of pain syndrome according to VAS from 9.5 (7.3; 10.0) to 2 (1.0; 2.0) scores during the year ($\chi^2 = 35.5$, df 4, $p < 0.001$) was observed. Noteworthy was a rapid regression of the pain syndrome 3 days after decompression from 9.5 (7.3; 10.0) to 4.5 (4.0; 6.0) and a decrease of ODI score and improvement in the quality of life of patients from 69.0 (58.5; 82.0) to 9.0 (4.8; 10.8): $\chi^2 = 36.8$, df4, $p < 0.001$. During the follow-up period, an increase in the Cobb segmental angle from 5.3 °(4.1°; 6.7°) to 9.7°(8.4°; 12.5°) ($p = 0.005$) was observed in all patients. However, this did not affect the intensity of back pain or the quality of life of patients. Newly occurring fractures, Kümmel's disease and postoperative complications were not identified. Instrumental diagnostics revealed spontaneous fusion at the fracture level during the first year after transpedicular decompression in all cases.

Conclusion. Transpedicular decompression is an effective, safe and pathogenetically substantiated method of treating vertebrogenic pain syndrome associated with spinal fracture.

Key Words: vertebral fracture, compression fracture, decompression, pain syndrome, back pain.

Please cite this paper as: *Basankin IV, Gyulzatyan AA, Nesterenko PB, Tayursky DA, Tomina MI, Mukhanov ML. Transpedicular decompression for painful AO Spine type A1 thoracic and lumbar compression fractures: case series study. Hir. Pozvonoc. 2022;19(3):49—56. In Russian.*
DOI: http://dx.doi.org/10.14531/ss2022.3.49-56.

Compression fractures or AO Spine type A fractures are traumatic lesions of the anterior structures of the vertebra, in which the posterior support column remains intact. A1 type fractures are characterized by injury to one, as a rule, the upper endplate without involving the posterior vertebral wall and are called impaction fractures [1]. According to Reinhold et al. [2], the frequency of A1 type fractures is 6.8 % of the total number of injuries to the thoracic and lumbar spine. Impaction fractures in acuity are almost always followed by a varying intensity vertebrogenic pain syndrome [3]. Nowadays, the pathogenetic mechanisms causing the occurrence of vertebrogenic pain associated with a fracture have not been fully studied. Meanwhile, elevated intraosseous pressure is one of its main triggers [4–6]. Studies conducted in the early 1970s with the measurement of intraosseous pressure in the spinous processes of intact and broken vertebrae showed a statistically significant elevation of intraosseous pressure in injured vertebrae [5–7].

Treatment of patients with painful A1 type fractures includes conservative treatment, cement augmentation of the vertebra and transpedicular fixation. We have introduced the transpedicular decompression technique into clinical practice [8] that allow reducing and stabilizing intraosseous pressure, which results in a change in blood circulation and a decrease in the concentration of proinflammatory factors in the vertebral body, contributing to a decrease in vertebrogenic pain syndrome.

The objective is to analyze the dynamics of vertebrogenic pain and quality of life after transpedicular decompression in patients with impacted fractures of the thoracic and lumbar spine.

Material and Methods

The study design: a small prospective series of case studies. The target of the study: patients with impaction (A1 according to AO Spine classification) fractures of the thoracic and lumbar spine. The scope of research: dynam-

ics of pain syndrome and quality of life of patients after transpedicular decompression.

Patients

The subjects were enrolled from January 2020 to December 2021. The study included 10 patients.

Inclusion criteria:

– high- and low impact solitary fracture of the thoracic and lumbar spine of type A1;

– chronic or increasing pain syndrome with a severity of more than 6 points on the VAS and no time limit;

– Cobb segmental angle is less than 10°;

– reduction of body height by no more than 30 %;

– follow up at least 12 months.

Exclusion criteria:

– Kümmell's disease;

– acute inflammatory diseases, either regional or general;

– hematologic malignancies of the spine.

Techniques

The study used neurological and clinical examinations for an objective assessment of the patient's condition. The pain intensity according to a 10-point VAS and the severity of disorders associated with pain according to the Oswestry low back disability questionnaire (ODI) were evaluated before surgery, in three days, a month, six months, and a year after surgery. MRI and CT examinations were used to visualize fractures before and after surgery (in three days, a month, six months, and a year after surgery). The segmental angle of kyphosis was measured using the Cobb technique (the angle between the upper endplate of the superjacent vertebra and the lower endplate of the subjacent vertebra relative to the injured vertebra) on CT scan before and after surgery; the X-ray technique was not used due to pronounced vertebrogenic pain syndrome in most patients on admission to the hospital.

The analysis of the clinical material was performed in accordance with the principles of the Declaration of Helsinki.

The course of the surgery

The patient's position is prone. Using X-ray control, a fractured vertebra was identified, and marking was carried out. The perforation of the injured vertebra from both sides was performed under local infiltration anesthesia and a single-up intravenous sedation with a Jamshidi needle, perforating the skin and layer-by-layer soft tissues, under X-ray monitoring through the vertebral pedicle (Fig. 1).

Core wires were taken out of the needles and syringes of 20 cm^3 with plungers lowered were attached to them. After that, the plunger of the syringe was gradually pulled, carrying out the evacuation of blood, thereby performing active decompression (Fig. 2). The total volume of evacuated blood is 10–20 ml.

Then the needles were removed; passive drainage was done into the paravertebral soft tissues through the formed canal, and an aseptic dressing was applied to the site of the puncture.

Statistical analysis

The obtained clinical outcomes were processed using the IBM SPSS 16.0 software. Once that the number of patients was 10 for a total, and the distribution of numerical values in part of the sample was considerably different from the normal law of distribution (the hypothesis of the normality of the distribution was done using the Kolmogorov-Smirnov test), nonparametric statistical methods were applied: the Wilcoxon signed-rank test and the Friedman test. The statistical significance level $p < 0.05$ was taken as the lower limit of validity. The data obtained on a sample with a distribution other than normal were recorded in the form of median – Me (25 and 75 percentiles) during the presentation of the study results.

Results

The treatment outcomes of 10 patients were analyzed. The patients were aged from 35 to 70 (Me = 46). The ratio of men to women was 1: 4. Low impact vertebral fractures associated with reduced bone mineral density (T-test 2.1 ± 0.6) were found in seven patients; high impact vertebral fractures (road accident, fall from a height) were noted in three patients. From the moment of injury to admission to the hospital: 6–8 days – in 3 (30 %) patients; 7–14 days – in 7 (70 %) patients. The fractures were divided according to localization as follows: T10 – 1 (10 %) patient; T11 – 1 (10 %) patient; L2 – 2 (20 %) patients; T12 – 3 (30 %) patients; and L1 – 3 (30 %) patients.

For the follow-up of patients during a year, there was an improvement in the form of a statistically significant regression of pain syndrome according to VAS from 9.5 (7.3; 10.0) to 2.0 (1.0; 2.0) points (Table 1, Fig. 3).

Also, a favorable evolution was noted in the form of a decrease in ODI indicators and an improvement in the quality of life of patients from 69.0 (58.5; 82.0) to 9.0 (4.8; 10.8) during a year (Table 2, Fig. 4).

During follow-up, there was an increase in the segmental Cobb angle from 5.3° (4.1°; 6.7°) to 9.7° (8.4°; 12.5°); the value of the T-test = 2.8, $p = 0.005$. Nevertheless, it did not affect the intensity of back pain or the quality of life of patients (Table 3, Fig. 5).

We present the case history of patient T., 52 y.o., with an early period of spinal injury, compression fracture of the T12 vertebra of type A1 according to AO Spine, and vertebrogenic pain syndrome (Fig. 6). The mechanism of injury is a fall from a height of 2 m. VAS (back) indicators: 8 points while taking NSAIDs. According to CT densitometry, the T-test of the T11 vertebra was -2.17; of the L1 vertebra was -2.63, the Cobb angle was 6.8°. The patient underwent a transpedicular decompression of the T12 vertebra. The surgery duration was 20 minutes. Intra-and postoperative complications were not reported.

Three days after the surgery, the VAS (back) indicators were 4 points without taking NSAIDs; the bilateral transpedicular track of Jamshidi needles was visualized on control CT and MRI scans, with an increase in the deformity angle according to the Cobb up to 8.6° (Fig. 7).

The patient was discharged on the third day after the surgery; he was back to work in four weeks. CT and MRI scans in a year after the surgery revealed a spontaneous block at the level of the fracture; according to MRI T2 STIR

there was no hyperintensive signal from the T12 vertebra. The Cobb angle was 12.7°. VAS (back) indicator in a year was 1 point (Fig. 8).

There were no new fractures, K mmell's disease, or postoperative complications. The presence of osteoporosis in some patients did not affect the treatment outcomes and it was not a limiting factor. In all cases, a spontaneous bone block at the fracture level was visualized during imaging diagnostics throughout the first year after transpedicular decompression. Patients were activated in a brace a day after surgery. The duration of the hospital stay was no more than four days. In the postoperative period, NSAIDs were administered in the presence of relevant symptoms.

Discussion

According to the literature data [8-11], there is a direct relationship between vertebrogenic pain syndrome associated with a vertebral fracture and elevated intraosseous pressure. The pain in impacted fractures is chronic and disabling; despite non-surgical treatment, it reduces the quality of life of the patients [12,13]. An impairment of venous circulation plays a big part in the pathogenesis of the elevated intraosseous pressure in compression fractures [11]. Veins have thin walls and low blood pressure. Therefore, they are constricted much more easily in compression fractures than arteries that have a dense wall and high arterial blood pressure [12]. The impairment of venous outflow through the basivertebral vein is followed by reflex narrowing of the

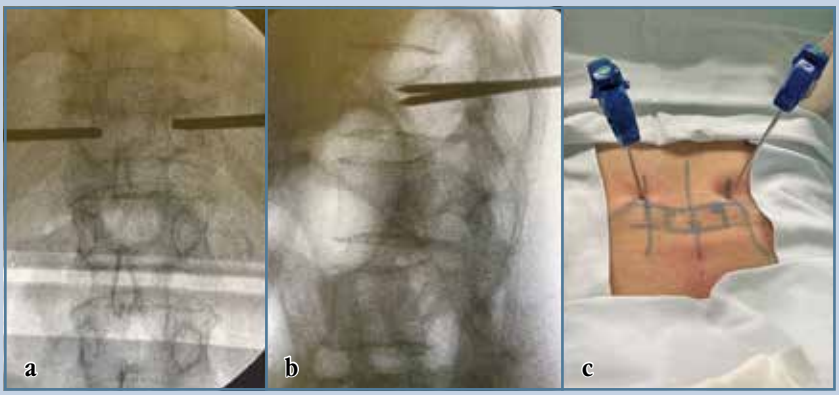

Fig. 1
Intraoperative anteroposterior **(a)** and lateral **(b)** radiographs illustrating the bilateral insertion of Jamshidi needles into the body of the T12 vertebra; lateral x-ray picture; marking on the skin with visualization of the needles **(c)**

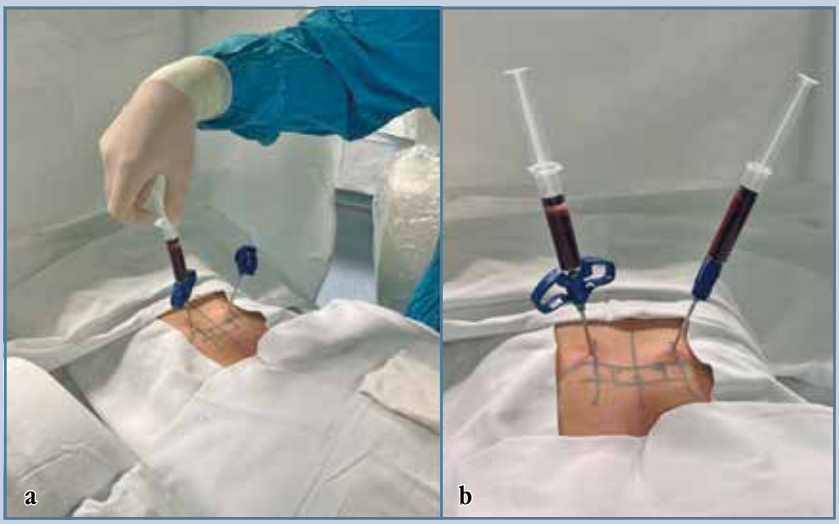

Fig. 2
The process of evacuation of stagnant blood **(a)** and general view after decompression **(b)**

Table 1
VAS scores before and after surgery

Parameters	Before surgery	3 days after surgery	A month after surgery	6 month after surgery	A year after surgery
VAS, points	9.5 (7.3; 10.0)	4.5 (4.0; 6.0)	2.5 (2.0; 3.0)	2 (1.0; 2.0)	2 (1.0; 2.0)
Comparison test: Friedman test	$\chi^2 = 35.5$, df 4, $p < 0.001$				

Me (25; 75).

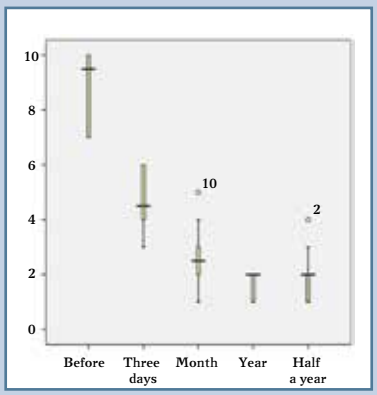

Fig. 3
Dynamics of regression of pain syndrome in the back during the year after surgery

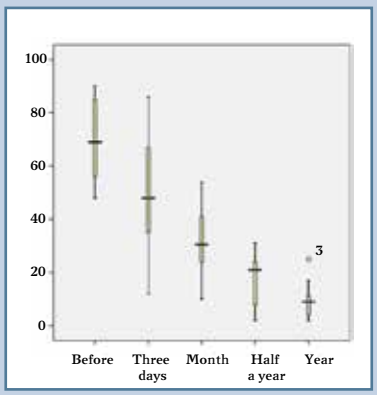

Fig. 4
Dynamics of ODI indicators during the year after surgery

arteries, thereby creating conditions for the onset of hypoxia [13]. This results in the formation of edema, impaired microcirculation in the vertebra, accumulation of lactic acid, algogenic factors, and a decrease in pH. Substances accumulated against the background of venous stasis and hypoxia stimulate mechanonociceptors and also nerve fibers of the basivertebral nerve, which causes the onset of pain syndrome.

Intraosseous decompression has become broadly used in traumatic injuries of the long tubular bones of the lower extremities, as well as in degenerative and inflammatory diseases of the hip and knee joints [12, 14, 15]. Later, this experience was extrapolated and utilized in compression fractures of the vertebrae as well as in degenerative spine diseases [16, 17]. In the course of transpedicular decompression, bilateral perforation of the vertebral body is carried out; and an active evacuation of stagnant venous blood is performed, thereby reducing intraosseous pressure. Additionally, there is a mechanical evacuation of accumulated cytokines, reducing their concentration and decreasing the stimulation of mechanoreceptors and fibers of the basivertebral nerve. During the first few hours, the pain decreases, and the postoperative period, as a rule, proceeds without serious complications. Bone density remains the same. Therefore, the risk of fractures of adjacent vertebrae is much lower since bone cement is not injected, which changes the density and mass of the vertebra. Yokoyama et al. [18] compared the clinical treatment outcomes of 108 patients with painful depressed fractures of the vertebrae who underwent transpedicular perforation (n = 58) or vertebroplasty (n = 50). The authors concluded that transpedicular perforation is an effective treatment for fractures with a moderate decrease in the height of the vertebral body, and the clinical outcomes in the postoperative period are comparable to those with vertebroplasty. Likewise, the frequency of postoperative complications is significantly lower. Remarkably, the frequency of newly occurring vertebral fractures in the group where patients underwent vertebroplasty was considerably higher than in the group with perforation (p = 0.042). Analysis of vertebral height reduction during follow-up in the perforation and vertebroplasty groups did not show statistically significant differences: the perforation group – 6 (22.2 %) cases compared with the vertebroplasty group – 8 (19.0 %) cases; p = 0.38.

In our study, all patients had favorable evolution in the form of a reduction in back pain and an improvement in the quality of life throughout a year of follow-up. During imaging diagnostics in all cases, throughout the first year after transpedicular decompression, a spontaneous spondylodesis was visualized with an adjacent segment above the impacted area. An increase in the segmental Cobb angle was reported during the year from the initial 5.3° (4.1°; 6.7°) to 9.7° (8.4°; 12.5°). Nevertheless, this did not affect the quality of life of the patients or the pain syndrome. According to the literature data [19, 20], the progression of kyphotic deformity also occurs against the background of vertebroplasty of the injured vertebra, and the correction of

Table 2
ODI indicators before and after surgery

Parameters	Before surgery	3 days after surgery	A month after surgery	6 month after surgery	A year after surgery
ODI, points	69.0 (58.5; 82.0)	48.0 (36.8; 64.5)	30.5 (24.5; 39.0)	21.0 (11.0; 23.5)	9.0 (4.8; 10.8)
Comparison test: Friedman test	$\chi^2 = 36.8$, df 4, $p < 0.001$				

Me (25; 75).

Table 3
Comparison test: Friedman test

Parameters	Before surgery	After surgery
Segmental angle, degree	5.3 (4.1; 6.7)	9.7 (8.4; 12.5)
Comparison test: Wilcoxon T-test	T = 2.8°; p = 0.005	

Me (25; 75).

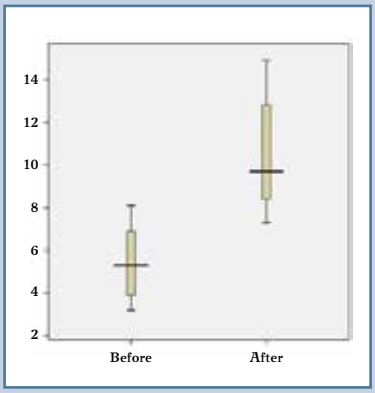

Fig. 5
Progression of segmental Cobb angle one year after surgery

post-traumatic deformity remains one of the topical and studied challenges of modern vertebrology. Nonetheless, there is no reliable correlation between the degree of post-traumatic kyphotic deformity and the intensity of back pain. For example, Gertzbein and Harris [21] in their study found a positive correlation between post-traumatic kyphotic deformity of more than 30° and pain syndrome. On the contrary, Zeng et al. [22] disprove this data. In this regard, the problem of post-traumatic deformities and the appropriateness of their correction remains controversial, and it is the subject of further research.

Study limitations. A small group of patients was analyzed (following a series of cases). Intraosseous pressure was not checked in the patients. The research does not include a control group of non-surgical treatment or those treated with cement augmentation techniques. A comparative analysis will be the topic of further research.

Conclusion

Transpedicular decompression is an effective, safe, and pathogenetically substantiated method of treating vertebrogenic pain syndrome associated with spinal fracture. Nevertheless, the treatment of painful impacted fractures (A1 according to the AO Spine classification) is currently not standardized; it is very variable and often depends on the institutional particularities and personal priorities of the surgeon.

All patients signed an informed consent to participate in the study.
The study had no sponsors. The authors declare that they have no conflict of interest.

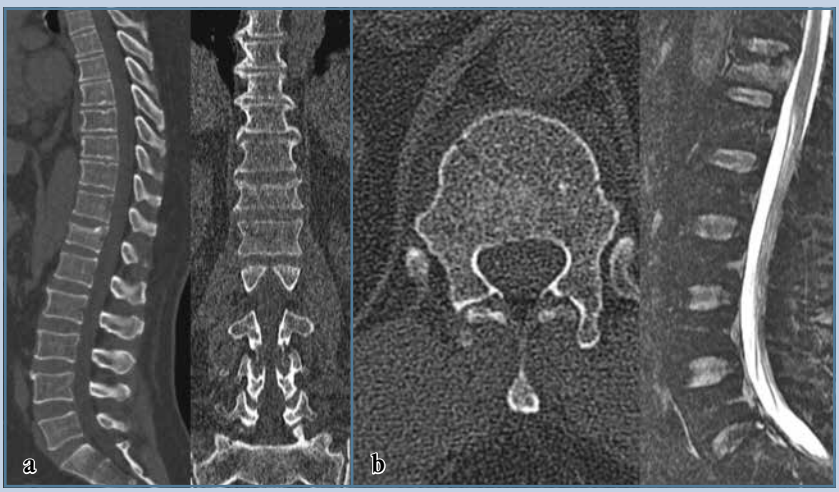

Fig. 6
Preoperative CT scan (a) and MRI in T2 STIR fat suppression mode (b) of patient T., 52 years old: an impacted fracture of the T12 vertebra is visualized

Fig. 7
Postoperative CT scan **(a)** and MRI **(b)** of patient T., 52 years old: the transpedicular course of the Jamshidi needles is marked with arrows

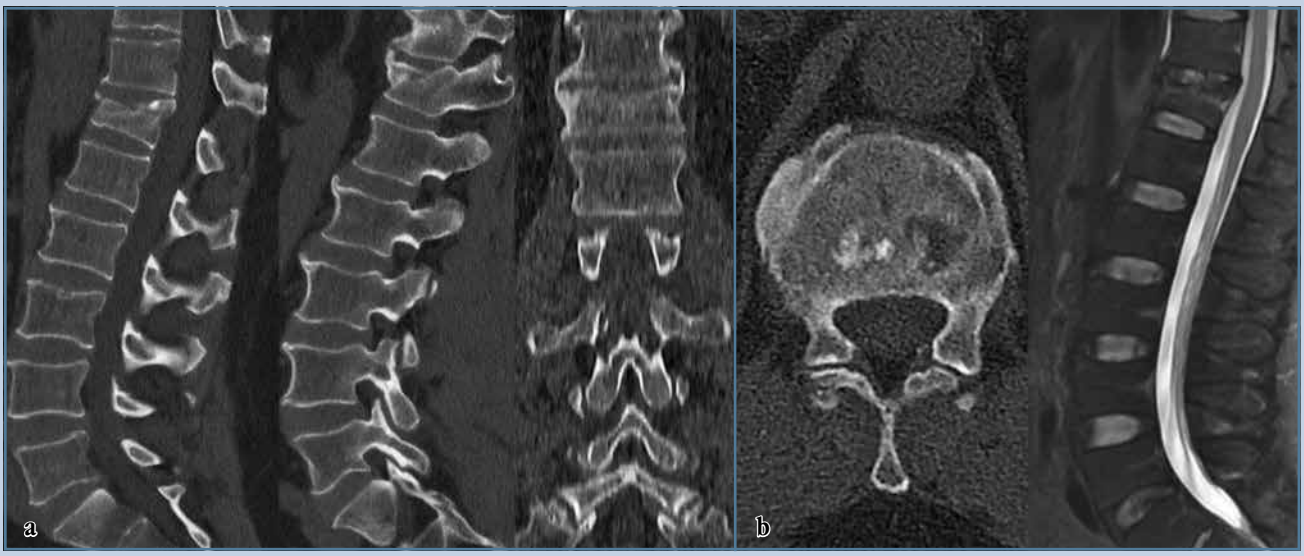

Fig. 8
CT scan **(a)** and MRI T2 STIR **(b)** of patient T., 52 years old, a year after transpedicular decompression: spontaneous bone block at the level of the fracture **(a)** and the absence of a hyperintense signal from the T12 vertebra **(b)**

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Received 21.06.2022
Review completed 22.07.2022
Passed for printing 28.07.2022

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