NEUROLOGICAL SYMPTOMS IN SCHEUERMANN'S DISEASE: REVIEW OF RARE CLINICAL OBSERVATIONS

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Neurological symptoms in Scheuermann's disease are very rare, only a few dozen cases have been described. The main causes of spinal cord compression with the development of neurological symptoms in spinal deformities due to Scheuermann's disease are compression by the anterior wall of the spinal canal, together with the dorsal leaflet of the dura mater, intervertebral hernia, and extradural bone cyst. The review provides a description of 38 clinical observations found in the literature. Compressing factors can also be spinal epidural lipomatosis and a displaced fragment of the annular apophysis. Scheuermann's disease can be combined with syringomyelia. The magnitude of the kyphotic deformity does not correlate with the severity of neurological symptoms. Preoperative examination of a patient with Scheuermann's disease should include methods that allow visualizing the condition of the spinal canal and its contents.

Key Words: Scheuermann's disease, spinal cord compression, surgical treatment.

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Practically any spinal deformity, irrespective of the etiology, may be followed by a conflict between the walls and the contents of the spinal canal. It may arise at a certain stage of pathogeny. Conversely, this conflict is fraught with the neurological symptoms of varying degrees of intensity: from minimal disorders in the sensory and motor areas to paraplegia. The classic paper by Scheuermann [1] has allowed specialists to recognize the presence of a special nosological unit, which is named after the Danish orthopedist and radiologist who characterized it. Scheuermann's disease is detailed clinically and radiologically. Its incidence and course are well known [2]. However, we still do not fully understand the etiology of Scheuermann's disease [3], and its treatment (primarily surgical) strategy has undergone significant changes over the years [4]. The voluminous literature is devoted to this disease. Only in the Scopus database there are more than 800 publications. Few of them are dedicated to the neurological complications accompanying the development of Scheurmann's disease. Moreover, the majority of the publications are descriptions of single (maximum – 6) clinical observations. The aim of our review is to give a comprehensive picture of the solution in terms of the world literature.

The spinal deformity with neurologic impairments is first mentioned in 1888 [5]. Nevertheless, this description does not refer to Scheuermann's disease. The works of Scheuermann [6] and Sorensen [2] do not mention the authors ' own findings of Scheuermann's disease with neurological deficits, though they have treated more than 120 patients. It was Kienbock who in 1936 first described the condition in a 19-year-old shoemaker with Scheuermann's disease and spastic paraplegia [7]. The myelogram clearly showed the block. The diagnosis was confirmed during the surgery. Sorensen emphasised how this complication may develop in patients with moderately severe kyphosis. Sorensen [2] analyzed the few available literature data at that time and distinguished three causes for the neurological symptoms in patients suffering from Scheuermann's disease. They are the following: spinal cord compression at the kyphosis apex associated with deformity of the anterior wall of the spinal canal, intervertebral disc herniation, and spinal epidural cyst. We suggest that each of these pathoses requires detailed consideration.

The pathogenesis of myelopathy associated with spinal cord compression by the wall of the deformed spinal canal is detailed by Lonstein et al. [8]. These authors introduced a group of 43 patients suffering from spinal deformities of various etiologies (Scheurmann's disease – in two) and neurological symptoms of varying severity. We quote: "In cases of gross kyphosis, the spinal cord is stretchable over a bony shelf formed on the anterior wall of the spinal canal, choosing the shortest path through the deformity area. At the same time, the spinal cord is tightly attached to the dorsal surface of the vertebral bodies. Additionally, the spinal cord is constricted from behind by the dura mater (DM). The latter is fixed to the base of the skull and to the sacrum and is characterized by low elasticity. When the spine is flexed, its dorsal portion is stretched. When the flexion increases, particularly if the kyphosis is angular, the tension of the dorsal portion of the DM grows, pressing the spinal cord against the posterior

surface of the bodies of the apical vertebrae. If the dura mater is incised and left in this condition, the pressure on the spinal cord decreases. By this means, the improvement can be noted after laminectomy and dura incision. The combination of spinal cord compression and dural sac traction has an adverse effect on the blood supply to the compromised spinal cord. The blood supply to the spinal cord, the role of which in the compression of the latter is well known, was characterized by Dommisse [9]. He emphasized its segmental nature and the occurrence of anastomoses in the area of the intervertebral foramina. Compression and tension of the spinal cord results in venous obstruction. The more pronounced the obstruction, the lower the blood flow. Hypoxia leads to the disorder of the spinal cord function and the development of the corresponding neurological symptoms" [8].

We managed to find a description of 11 cases of spinal cord compression (Table 1), verified during presurgical examination or during the interference.

It should probably be considered the first report of the American military doctors Gulledge and Brav [10], who in 1950 told about a common soldier of the US Army aged 21 years, who had a week feeling in his legs when performing "about-face" command. The examination showed thoracic gross kyphosis, the X-ray - a typical aspect of Scheuermann's disease (Fig. 1), but the diagnosis (old healed vertebral osteochondrosis) raises eyebrows. Bradford and Garcia [11] also regard this case as a common juvenile kyphosis. From the neurologic point of view, it can be defined as a lower spastic paraplegia. The treatment course included complete bed rest in the position of thoracic spine extension (without any improvement), followed by surgical intervention. Laminectomy was performed throughout T7–T10 vertebrae; the dura mater was dissected longitudinally and transversely on three levels. The spinal cord was released, plastic surgery of the dura defect was performed using polyethylene. After a significant regression of neurological symptoms, a common soldier was discharged from the army after 9 months due to residual weakness and spasticity.

In the above-mentioned review, Lonstein et al. [8] described the treatment of two patients suffering from Scheuermann's disease. Forty two out of 43 patients had kyphotic deformity with an average Cobb angle of 95°. Yet the authors did not indicate the magnitude of kyphosis in particular patients. Both individuals with Scheuermann's disease have inflammatory complications, which were cut short without any consequences. Generally, the best results were found in patients undergoing deformity and spondylitis correction surgery without opening the lumen of the spinal canal (in the presence of mobile deformity), and the worst - after laminectomy. The best results in rigid kyphosis are achieved by anterior decompression of the spinal canal contents.

Ryan and Taylor [12] published three findings on the development of acute spinal cord compression. The patients were young (14, 18, and 20 years old), and the kyphotic deformities were small (56°, 47°, and 58°). The compression of the spinal cord, according to myelography, occurred at the deformity height, which resulted in the development of spastic paraparesis. Anterior decompression of the dural sac was performed in all cases, resulting in a significant regression of neurological symptoms. Risk factors were Cobb angle, number of segments involved, degree of increase in deformity (progression), local anatomic conditions, injury, and secondary impairment of spinal cord vascularization.

Normelli et al. [13] reviewed the literature data (16 cases describing spinal cord compression in Scheuermann's disease) and updated them with their own observations. Main frameworks: there are more men, they grow longer, kyphosis develops faster; there is no clear correlation between the severity of neurological symptoms and the magnitude of kyphosis; unlike other types of deformities, the apex of the deformity is on average in the area of the T9 vertebra, dorsal decompression is ineffective.

Kapetanios et al. [14] showed a case of complete recovery, with very fast recovery (2 months), in a 14-year-old patient who had developed lower spastic paraplegia a week before admission. Anterior decompression and spinal fusion were applied, followed by posterior instrumental fixation. There was no correlation between the severity of neurological disorders and the magnitude of kyphosis. It is more likely to develop complications with gibbus than with arcular kyphosis. Risk factors: reduced blood circulation, narrow thoracic spinal canal, rather massive discs in young patients, the absence or small number of Schmorl's nodules (decompression of the disc space).

Putz et al. [15] operated a 15-year-old patient with progressive paraplegia who had hypoplasia of the spinous processes and paraspinal muscles in the upper thoracic spine. The authors assumed that the underdevelopment of the posterior spinal and paraspinal structures in the presence of an injury in the anamnesis and a small length of kyphotic deformity may be the foundation for the rapid development of neurological symptoms. Surgical treatment was effective.

Seriali et al. [16] reported a case of juvenile kyphosis in a 14-year-old patient with symptoms of pyramidal insufficiency. Immobilization was done using a plaster jacket in the position of spinal deformity correction. The pain disappeared immediately, and the neurological symptoms resolved after three weeks. Yet, after 4 months, the pain and neurologic deficit returned. Due to the presence of compression signs of the dural sac, an interbody fusion surgery of T4–T10 was performed. After 18 months, a pseudoarthrosis of the bone block and an increase in kyphotic deformity with compression evidence of the spinal cord were diagnosed. Posterior fixation with CDI and correction of kyphosis were carried out. After 8 years, there is no pain and the neurological status is stable.

M.V. Mikhailovsky et al. [17] analyzed the treatment outcomes of a 17-year-old patient with typical juvenile kyphosis. Neurologically: the brisk of tendon reflexes from the legs; there are foot clonus in the vertical position during the traction test, grosser on the left. There were no negative changes after the sur-

Table 1
Spinal cord compression by the wall of the deformed spinal canal in patients with Scheuermann's disease (literature data)

Authors	Year	Age of the patient	Sex	Cobb angle, deg.	Kyphosis apex	Vertebral arch	Time of symptoms development	Neurological disorders (particular level)	Pain	Surgical intervention	Effect	Follow-up period	Myelography
Gulledge, Brav [10]	1950	21	M	–	–	–	5 недель	Spastic paraparesis	–	Laminectomy, dissection of DM longitudinally and transversely	Significant recovery	9 months	–
Lonstein et al. [8]	1980	17	–	–	–	–	–	Spastic paraplegia	–	Anterior decompression, spondylodesis, posterior spinal fusion	Full recovery	–	Partially
	1980	19	–	–	–	–	–	Spastic paraparesis	–	Posterior spinal fusion, Harrington contractors	Significant recovery	–	Partially
Ryan, Taylor [12]	1982	18	M	–	–	–	1 month	Spastic paraparesis (T8–T10)	Yes	Laminectomy of T8–T10, vertical dissection of dura, anterolateral decompression, interbody fusion	Impairment, significant recovery after the second surgery	3 years	–
	1982	20	M	47	T7–T8	T7–T10	–	Spastic paraparesis (T7–T8)	Yes	Thoracotomy, interbody fusion	Full recovery	1 year	Obstruction at the level T7–T8
	1982	14	M	–	–	–	4 weeks	Spastic paraparesis	–	Thoracotomy, interbody fusion of T6–T9, after 2 weeks-fixation by Harrington contractors	Full recovery	1 year	–
Normelli et al. [13]	1991	20	M	60	T5–T7	T5–T11	6 months	Paraparesis (T9)	No	Posterolateral approach, anterolateral decompression, posterior spinal fusion	Almost full recovery	5 years	–
Bruns, Heise [48]	1994	17,5	M	64	T6–T7	–	–	Spastic paraparesis	–	Anterior release, interbody fusion, posterior fusion	Full recovery	3 months	Content on 2 levels
Kapetanos et al. [14]	2006	14	M	66	T8–T9	T7–T10	5 months	Spastic paraplegia	–	Anterior release, interbody fusion, posterior fusion	Full recovery	2 years	–
Putz et al. [15]	2009	15	M	65	T5–T6	T3–T8	3 months	Spastic paraplegia	Yes	Anterior release, interbody fusion, posterior fusion	Significant recovery	6 months	–
Sariali et al. [16]	2009	14	M	28	T6–T7	T4–T8	–	Spastic paraparesis	Yes	Anterior spondylodesis, after 18 months – posterior spinal fusion of T2–L2	Spastic paraparesis of T8–T10	8 years	–
Mikhaylovskiy et al. [17]	2009	17	M	87	T9	T8–T12	–	Increased tendon reflexes in the legs, with traction – feet clonus	Yes	Posterior approach, segmental tool correction	Without negative changes	–	–

DM – dura mater.

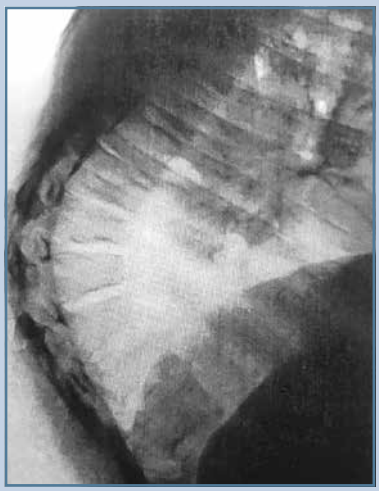

Fig. 1
Radiographic changes typical for Scheuermann's disease [10]

gery. The reason for the development of neurological symptoms was the magnitude (87°) and the progression rate of kyphotic deformity.

The data collected in the cumulative table shows that all the patients were young (from 14 to 21 years), while all 9 patients whose gender is known were men. The magnitude of the Cobb angle of kyphotic deformity is known only in six cases: it was averaged at 58.8° (28–87°). Such kyphosis can be called severe only with a long stretch. The position of the kyphosis apex ranges from T5 to T9. It means that it is limited, and the length of the curvature varies from three to six segments. The development time of neurological symptoms was from one to six months. During this time, in 10 cases out of 11, lower paraparesis or even paraplegia was developed. Five patients had pain syndrome. One patient did not suffer from it. The data concerning the rest of the patients is not available. All the patients underwent surgical treatment. The laminectomy was used only twice. In the first case, there was an improvement (the follow-up period was 9 months), in the other – worsening of the symptoms, which required repeated intervention. The last one was successful. In the remaining cases, different variants of spinal cord decompression and effective stabilization were used, which resulted in full or very significant recovery of lost functions within a timeframe of six months to five years.

Herniated thoracic intervertebral discs are very rare (this is not only about Scheuermann's disease). According to Court et al. [18], it is 1 case per 1,000,000 population. They are most often detected in patients aged 30–50 years, with an even gender distribution. In 75 % of cases, hernias are localized at the level of T7–T8 disc; another preferred localization is T11–T12, due to increased mobility and a relatively weak posterior longitudinal ligament in the area of this segment.

A peculiarity of herniated thoracic intervertebral discs is their tendency to calcification or even ossification. According to Quint et al. [19], the hernia calcification was found in 42 % of 168 operated patients. There is no data on the mechanism of calcification. The cases of self-existing regression of neurological symptoms of such hernias are described. An injury can be a triggering factor in up to 37 % of cases of herniated thoracic intervertebral discs. Court et al. [18] emphasize that the development of herniated thoracic intervertebral discs is typical for patients with Scheuermann's disease. Such hernias can reach large sizes. Hott et al. [20] defined the hernia as a giant, if according to CT or MRI, it takes more than 40 % of the spinal canal lumen. In such cases, calcification of the hernial contents is often noted. The volume and morphological changes in the disc tissue increase the risk of their intrusion through thinned and eroded dura. The frequency of this complication for giant calcified hernias varies from 15 to 70 %, and it is found only during surgery. Ossification of the posterior longitudinal ligament at the hernia level is likely to be detected radiologically and can imitate a calcified hernia [21].

The clinical course of the process is usually progressive. The diagnosis is confirmed on average 15 months after the first symptoms appear. The main manifestation is pain in the thoracic spine (92 %). The other, rarer, symptoms are: pain in the cervical and lumbar regions, in the area of the shoulder blades, headaches, radicular pain, and Horner syndrome. If the spinal cord is compressed, sensory and motor disorders develop, corresponding to the level of the involvement. Their development may be sudden, as a result of injury. In rare cases, occlusion of the anterior spinal artery is manifested by symptoms of transient or permanent paraplegia. The following reasons determine the weakness of the thoracic spinal cord [21]:

– a possible intradural sprain of the spinal cord, leading to its compression;
– thoracic kyphosis, pressing the spinal cord to the anterior wall of the spinal canal;
– odontoid ligaments that limit its mobility;
– large size of the spinal cord (6.5 × 8.0 mm) relative to the size of the spinal canal (16.8 ×17.2 mm),
– low vascularization of the spinal cord in the thoracic area.

Preoperative imaging.

Conventional spondylograms are most informative in the presence of calcified and ossified hernias. Accurate identification of the affected segment is available using MRI and CT, as well as the nature of the hernia, its position in the lumen of the spinal canal and volume. While planning a surgery, angiography is useful for identifying the Adamkiewicz artery, the largest of the anterior medullary arteries.

We found 24 descriptions of cases of herniated thoracic and lumbar intervertebral discs in patients with Scheuermann's disease, complicated by the neurological symptoms (Table 2).

The first description of a disc herniation in a patient with Scheuermann's disease belongs to Muller [22, 23].

Van Landingham [24] described a case of a herniated thoracic disc in a 17-year-old boy who woke up with a feeling of numbness in his legs below the hip joints. Neurological symptoms gradually increased. On radiographs – a picture of juvenile kyphosis, on myelograms – filling defects at the levels of T7, T8 and T9 discs. Laminectomy and extradural removal of the abnormal discs were done.

The posterior longitudinal ligament was intact. Within four weeks, there was a complete recovery of spinal cord function. Almost a similar case was recorded by Roth et al. [25], but with the difference that the result of laminectomy was worsening of neurological symptoms: from paraparesis to paraplegia.

Bradford and Garcia [11] identified only 4 cases of disc herniation in Scheumann's disease with neurological symptoms in the literature and presented their observation. A spastic paraparesis was developed in a 16-year-old patient. The laminectomy of T7–T9 was performed; a hernia was found of T7–T8 disc. Dura dissection was not carried out; the strength increased in the postoperative period, but the spasticity remained. After 10 years, a full recovery was achieved.

Turinese and Raven [26] found a full recovery after the laminectomy of the spinal cord functions in the patient with the initial paraparesis from the level T7–T8.

Yablon et al. [27] described a 29-year-old patient with the spinal cord compression due to a combination of kyphosis and a herniated disc. The magnitude of kyphosis is only 14°. Myelography: defect of T7–T8. Costotransversectomy, anterior decompression without spinal fusion, full recovery. After 6 months, relapse, repeated transthoracic decompression, spinal fusion. Recovery was detected.

Lesoin et al. [28] published 6 cases of hernias (f/m – 2/4) of the thoracic intervertebral discs in patients with juvenile kyphosis. The age of patients varied from 27 to 61 years. There were neurological symptoms of varying severity in all cases, indicating ventral compression of the dural sac at the level of the apex of the kyphotic deformity (there is no data on the value of the Cobb angle in all cases). The authors emphasize that the pathogenetic function of Scheuermann's disease in such cases requires confirmation. According to them, the relationship between changes in the intervertebral discs and the neurological symptoms is shown in the literature by a very small number of observations, which preserves the popularity of Sheuermann's disease as a benign current disease. In such cases, the new methods of examination (myelography, CT) give surgeons serious grounds to resort to surgical treatment. Three of the six reported patients were operated from the posterolateral approach proposed by the authors. Laminectomy has to be left out because of the large number of negative results. Anterior approach is difficult and traumatic. The posterolateral approach leaves intact the neurovascular structures. This is a transverse arthropediculectomy providing tangential access to the disc. The muscles shift and do not intersect. The intervertebral foramen is opened at the beginning of the surgery, and the neurovascular structures are easily localized. The next step is an osteotomy of two vertebral bodies adjacent to the abnormal disc. It allows the disc to be removed without the mobilization of the dural sac and roots. The only disadvantage of the surgery is the relative instability at the level of intervention. In all cases, the authors used a short Harrington rod to stabilize the spine. There was a marked regression of neurological symptoms in all cases.

Bohlman and Zdeblick [29] operated 22 patients with hernias of the thoracic spine, two of whom were diagnosed with Scheuermann's disease. In the first case the costotransversectomy and spinal fusion were used. In the second case, the transthoracic approach, decompression, and spinal fusion were used. Transthoracic approach proved to be the best one.

Stambough et al. [23] defined two causes of the neurological symptoms development in Scheuermann's disease: 1) gibbus; 2) spinal cord compression by a displaced intervertebral disc. The recovery rate depends on the severity and duration of the symptoms. The published case is interesting for several reasons: hernias are formed at two levels. Moreover, their clinical significance is not equal, and a massive layer of epidural fat is located dorsally to the spinal cord, but does not compress it. Posterior spondylodesis as an addition to the ventral one is indicated for kyphoses heavier than 55°.

Bhojraj and Dandawate [30] presented 3 cases of thoracic disc herniation with the development of neurological symptoms. Excellent results were obtained. Posterior access with secure fixation offers a number of advantages: simplicity, fewer complications, solution of side effects (cysts, narrowing of the canal, internal gibbus). Posterior fusion is necessary. We need intraoperative spinal cord monitoring. All 3 cases were observed in the thoracolumbar junction. This is a unique observation.

Chiu and Luk [31] reviewed a case of paraparesis development due to the combination of two factors: a herniated disc and an intraspinal cyst in a patient with juvenile kyphosis.

Song and Yang [32] observed a rare case of traumatic disc herniation in a patient with a lumbar variant of Scheuermann's disease. A 24-year-old man went to the hospital due to severe pain in the thoracolumbar junction after a fall. No neurological symptoms were found. Radiographic diagnosis was: lumbar variant of Scheuermann's disease, as well as a body fracture of T12 vertebra. A disc herniation was identified in combination with the separation of the posterior part of the upper apophysis of the L1 vertebra, which resulted in the spinal cord compression. This type of hernia is called posterior retroextramarginal disc hernia. At the level of T12: a large Schmorl hernia with an extended U-shaped notch in the end plate which opens into the spinal canal. The patient refused the proposed admission to the hospital for a full examination. After 16 hours, he went to the hospital again with complaints of weakness in his right leg and inappropriate urination. Neurological diagnosis was spastic paraparesis from the level of L1. A lower paraplegia was found within 4 hours after the second visit. Surgery included laminectomy of T12, decompression of the dural sac, and posterior fusion using transpedicular fixation. After two years, incomplete regression was observed, rehabilitation continues. The authors suppose that in this case the rapid progress of paraplegia may be explained with the fact that with the already existing compression of the spinal cord, a traumatic hernia was formed

Table 2
Spinal cord compression by spinal disc herniation in patients with Scheuermann's disease (literature data)

Authors	Age	Sex	Cobb angle	Kyphosis apex	Extent of kyphosis	Development time of symptoms	Pain	Neurological disorders (particular level)	Surgical intervention	Effect	Follow-up period	Myelography
Muller [22]	40	–	–	–	–	–	–	Spastic paraplegia (T10)	No data	Normal findings	–	–
Van Landingham [24]	17	M	–	–	–	4 weeks	–	Spastic paraparesis (T8–T10)	Laminectomy of T7–T8, elemination of two discs	Significant recovery	4 months	Filling dfects of T7 and T8
Roth et al. [25]	61	–	–	–	–	–	–	Spastic paraparesis (T9–T10)	Laminectomy of T9–T10	Paraplegia	–	–
Bradford, Garcia [11]	16	M	–	–	–	–	–	Spastic paraparesis	Laminectomy of T7–T8, elemination of two discs	Significant recovery	–	Filling defects of T7
Turinese, Raven [26]	20	–	–	–	–	–	–	Spastic paraparesis (T7–T8)	Laminectomy of T9–T10	Full recovery	–	–
Yablon et al. [27]	29	M	–	–	–	2 weeks	Yes	Spastic paraparesis (T8–T10)	Costotransversectomy, discotomy of T7–T8, anterior discotomy, interbody fusion	Full recovery, then worsening, after the second surgery almost full recovery	8 months	Filling defect of T7–T8
Lesoin et al. [28]	48	F	–	–	–	8 months	–	Paraparesis (T10)	Laminectomy of T8–T10	Worsening to parapplegia, then partial recovery	1 year	Full extradural block
	55	F	–	–	–	6 weeks	–	Paraparesis (T8–T9)	Transthoracic approach, elemination of a disc	Partial recovery	8 months	Calcified disc herniation T8–T9
	61	M	–	T10–T11	–	–	–	Spastic paraparesis	Posterior approach, hernia excision, Harrington contractors	Significant recovery	9 months	Spinal cord compression T11–T12
	27	M	–	–	–	–	Yes	Transient paraparesis (T9–T10)	Posterolateral approach, Harrington short rods	Full recovery	3 months	Contained disc herniation T8–T9
	30	M	–	–	–	1 year	Yes	Paraparesis	Posterolateral approach, Harrington short rods	Full recovery	3 months	Contained disc herniation T8–T9
	35	M	–	–	–	–	Yes	Spastic paraparesis (T8–T9)	Posterolateral approach	Significant recovery	3 months	Contained disc herniation T8–T9
Bohlman et al. [29]	38	F	–	–	–	6 weeks	Yes	Monoparesis (T11–T12; T12–L1)	Costotransversectomy, spinal fusion	Full recovery	36 months	–
	25	F	–	–	–	7 years	Yes	T8–T9	Transthoracic approach, anterior decompression T8–T9, spinal fusion	Significant recovery	24 months	–

Table 2. Breakover

Authors	Age	Sex	Cobb angle	Kyphosis apex	Extent of kyphosis	Development time of symptoms	Pain	Neurological disorders (particular level)	Surgical intervention	Effect	Follow-up period	Myelography
Stambough et al. [23]	21	M	35	Th6–Th8	Th5–Th12	12 weeks	No	Paraparesis (T8)	Transthoracic approach, anterior decompression T6, T7, spinal fusion	Minimal symptoms remained; the patient returned to work.	24 months	Spinal cord compression T6–T7; T7–T8
Bhojraj et al. [30]	16	M	–	–	–	1 month	Yes	Paraparesis (T11)	Laminectomy, bilateral posterolateral decompression, interbody fusion, TPF	Full recovery	36 months	Filling defect at the level of T11–T12
	25	M	–	–	–	4 months	Yes	Paraparesis (L1)	–	Full recovery	24 months	Filling defect at the level of T12–L1
	16	F	–	T11–T12	–	12 months	Yes	Paraparesis (T11)	–	Full recovery	15 months	Filling defect at the level of T11–T12
Chiu, Luk [31]	35	F	–	–	–	6 years	–	Paraparesis (L1)	Laminectomy, removal of an intraspinal arachnoid cyst, slightly improved manner of walking, anterior decompression after 3 months, removal of T11–L2 discs, anterior spine fusion	Significant recovery	24 months	Filling defect at the level of T12–L1; L1–L2
Song et al. [32]	24	M	–	–	–	16 hours	Yes	Paraparesis, paraplegia	Dorsal approach, decompression, TPF	Partial recovery	2 years	–
Chiche et al. [33]	40	F	43	–	–	2 hours after hospitalization	Yes	Flaccid paraplegia from the level L4	None	Partial recovery	3 months	MRI: no signs of spinal cord compression, signs of ischemia of T3–T8, hernias of T6–T7, T7–T8
	57	F	39	–	–	–	Yes	Paraparesis (Th10)	None	Partial recovery	3 months	MRI-signs of spinal cord compression
	32	M	47	–	–	–	Yes	Paraparesis (L2–L3)	None	Partial recovery	3 months	MRI-signs of spinal cord ischemia, disc herniation L2–L3
Zan et al. [34]	46	M	–	T9–T10	–	–	Yes	Brown-Sequard Syndrome	Posterior decompression, hernia removal, instrumental spinal fusion	Significant recovery, pain dissipated completely	–	Calcified hernia

TPF – transpedicular fixation.

by tissues from a large Schmorl's nodules, which opened into the spinal canal.

Chiche et al. [33] reported three cases of spinal cord ischemia due to compression of a herniated thoracic or lumbar intervertebral disc. In the first case, there was compression of a herniated disc of the Adamkiewicz artery or its branch with anterior spinal cord ischemia. The surgery was not performed due to the lack of prospects. The second case was a compression of the anterior spinal artery by a herniated disc T10–T11 from T10 to the conus. The third case was a compression of the Adamkiewicz or Deproqes-Gotteron artery. Operations were not carried out due to the absence of spinal cord compression and the risk of postoperative deterioration.

Zan et al. [34] operated a 46-year-old patient with the Brown-Sequard syndrome and pelvic organ impairment. MRI showed signs of severe spinal cord compression by slipped disc herniation of T9–T10. Anterior discectomy and interbody fusion were done. The positive changes were noted after 3 months – a change on the Frankel scale from C to D.

Twenty of the described 24 patients were operated. However, in three cases there was no surgery (21–23), and in one case there was no data on the intervention (1). The average age of patients was 31.6 years (from 16 to 61 years). There were 10 women and 12 men out of 22 patients whose gender was mentioned in the publications. The Cobb angle of thoracic kyphosis was reported in only four cases and averaged 35.6° (14–47°). The deformity height and the extent of the deformity are specified only in single works. In 13 patients, a pain syndrome of varying severity was found. Almost all the patients had severe neurological symptoms - paraparesis. In one case, there was paraplegia. The complication's duration of development varies greatly: from a few hours to seven years. The compression level of the dural sac: from T7 to L4. In seven cases, the surgery was carried out to remove the hernia by a laminectomy approach. In two (16, 19) of them, it was completed by anterior spinal fusion. The result in both cases was successful: significant or complete regression of neurological symptoms. In the remaining five (2–5, 7) cases, the results were mixed: three improvements and two aggravations of the complication. In 13 cases (6, 8–15, 17, 18, 20, 24) the hernia removal was accomplished by spinal fusion (anterior bone or dorsal instrumentation), and in all cases significant or complete recovery of the lost functions was fulfilled. The postoperative supervision period varied from 3 to 6 months.

A spinal extradural cyst and its related syndrome have been described by Elsberg et al. [35]. They presented 4 cases found among the description of 250 spinal tumors. The authors were confident that this was the first publication devoted to such a pathology. However, a year later, Lehman discovered three much earlier papers (Schlesinger, 1898; Krauss, 1907; Mixter, 1932) and added two observations of his own [36]. Elsberg et al. [35] emphasized that spastic paraplegia develops in adolescence, while the pain syndrome is not prevailing, and sensitive disorders are rare or completely absent. The X-ray shows the widening of the interarch gap (the Elsberg–Dyke symptom), and the arches themselves are narrowed and atrophic (Fig. 2). Elsberg et al. believed that the cyst was the result of a herniated protrusion of the arachnoid membrane through a defect in dura mater or congenital DM diverticulum. In 1937 Cloward and Bucy [37] when discussing this problem, suggested, that such cysts are the result of evagination of both arachnoid membrane and dura mater. Furthermore, they believed that cyst-associated kyphosis was the result of the cyst's compression of the venous trunks draining the vertebral bodies. To their opinion, kyphoses that are not associated with such cysts are the result of venous stasis in the vertebral bodies.

Adelstein [38] described 16 cases of cysts before 1941. Two of them included identified kyphosis and were supplemented by the author's own observation. Only 10 of the patients were under the age of 20. The rest individuals were older. The duration of the symptoms varied from 2 months to 13 years, usually more than a year. The removal of the cyst, in most cases, resulted in recovery. The cyst itself contained clear CSF in a connective tissue capsule with villi from the arachnoid membrane. Typically, the cysts spread over 2-4 spinal segments. Most of the Adelstein patients (11 out of 17) showed an increase in the width of the spinal canal. Wise and Foster [39] in their review characterized 33 cases of cysts (23 – in men, 10 – in women), and in 19 of them it was developed against the background of juvenile kyphosis. The patients with Scheuermann's disease always had a cyst located in the thoracic segment of the spinal canal. In 1959 Nugent et al. [40] в 1959 г. described 7 cases of cysts. One of them was in the presence of Scheuermann's disease.

While describing the case of an extradural cyst in 1970, Bodosi [41] concluded that histological examination of its wall does not explain the mechanism of development. The presence of comorbid juvenile kyphosis is not causally associated with the cyst. The combination of the two pathological conditions and especially the progression of kyphosis after removal of the cyst confirm the need for spinal fusion surgery.

Fiss et al. [42] reported a case of extradural lumbar meningocele, similar to a cyst and causing rapidly progressive paraplegia due to local compression of the dural sac. During the surgery, a grayish epidural sac was revealed, unrelated to the neural structures and compressing the dural sac dorsally. During its puncture, a large amount of clear cerebrospinal fluid was obtained. Its cavity was connected with the subarachnoid space; it was removed; the DM was stitched up. Two weeks later, a complete regression of neurological symptoms was observed.

Park et al. [43] observed a 15-year-old patient with spastic paraplegia and a cyst, identified by MRI. It sizes were 14.5 × 7.4 × 75.0 mm at the level of L1–L2 vertebrae. This cyst spread from the epidural space to both intervertebral foramina. During the surgery, two small holes were found connecting the cyst cavity and the subarachnoid space, through which the cerebrospinal fluid was released with each respiratory excursion. These foramina should be carefully sutured. The authors emphasize that the

etiology of extradural arachnoid cysts remains unknown. Some regard them as the result of DM congenital defects. Meanwhile, it is known that cysts of IA type according to Nabors et al. [44] can be associated with Scheuermann's disease. This is the type of cyst presented in this observation. According to some authors [37], such cysts can cause the development of juvenile kyphosis owing to an impairment of the venous outflow from the vertebral bodies due to the pressure of the cyst itself.

Classification of dural cysts according to Nabors et al. [44]:

1. Extradural cyst without inclusion of fibers of spinal nerve roots:

IA – extradural meningeal cyst;

IB – sacral meningocele.

2. Extradural cyst with the inclusion of fibers of the spinal roots.

3. Intradural cyst.

We found only 3 cases of neurological symptoms in patients with Scheuermann's disease and verified spinal epidural lipomatosis. The small number of observations does not allow for their analysis (Table 3).

It should be noted that in all cases of examination of the contents of the spinal canal (myelography, MRI), it was possible to visualize the cause of the neurological symptoms development. This refers to all the listed mechanisms of spinal cord compression.

In the last decade, the interest of researchers in the problem of spinal epidural lipomatosis, that is, the increased content of fat in the spinal epidural space, has increased. There is a common combination of spinal epidural lipomatosis with steroid therapy, obesity, and Cushing's syndrome. Spinal epidural lipomatosis can also be idiopathic. Normally, it is asymptomatic, but with an increase in the amount of adipose tissue, compression of the dural sac is likely with the development of neurological symptoms.

Abul-Qasim et al. [45] investigated the content of adipose tissue in the epidural space in 87 individuals (29 patients with Scheuermann's disease and 58 controls). Using MRI, they found that juvenile kyphosis is accompanied by the development of spinal epidural lipomatosis in 41 % of cases (in the control group – in 3 %). The amount of kyphotic deformity correlates with the amount of epidural adipose tissue. The authors conclude that MRI should be performed in the preoperative stage normally in all cases of Scheuermann's disease. Since the sagittal diameter of the spinal canal in Scheuermann's disease and in the general population is almost the same, patients with an increased amount of epidural fat theoretically have a risk of spinal cord compression and disruption of its blood supply. This condition may occur in the postoperative period with an edema of the epidural tissue. The authors proposed that the increase in the amount of epidural fat may contribute to the progression of kyphotic deformity in Scheuermann's disease.

Zhang et al. [46] analyzed the effect of epidural lipomatosis on the development of kyphotic deformities of the spine (not only Scheuermann's disease). It emerged that that epidural lipomatosis is a common pathological condition in patients with congenital, juvenile and tuberculous kyphosis. Surgeons should be aware of this, as extra fat can cause spinal cord compression and neurologic impairment.

Kim et al. [47] gave an overview of the problem of spinal epidural lipomatosis. Possible causes include: intake of steroid drugs, endogenous steroid hormone diseases, obesity, surgical triggering and idiopathic conditions. Immunodeficiency syndrome and Scheuermann's disease may also be involved in pathogenesis. Steroid-induced forms affect mainly the thoracic spine and are most likely to develop paraplegia. Treatment is prescribed by the cause of the pathology: weight loss, withdrawal of steroid medications, treatment of endocrine pathology, and surgical decompression of the dural sac. The goal is to decrease the amount of adipose tissue in the epidural space and eliminate neurological symptoms. Surgical treatment is effective, but may be accompanied by increased mortality associated with the initial condition of patients.

Bruns and Heise [48] described a case of paraparesis in a 17-year-old boy. The paraparesis developed as a result of spinal cord compression and was treated with anterior decompression and posterior spinal fusion by Harrington contractors. According to the authors, one of the possible causes of neurological symptoms in Scheuermann's disease is intraspinal lipoma.

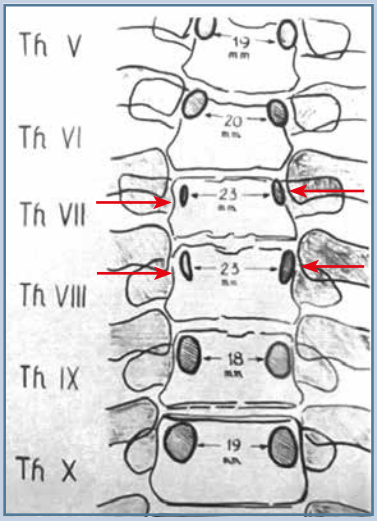

Fig. 2
Widening of the interarticular spaces, thinning of the pediculum; red arrows – a symptom of Elsberg – Dyke [41]

The thoracic form of Scheuermann's disease can cause the development of a very rare neurological complication. Soper [49] described 10 cases of shift of a fragment of the annular apophysis of the vertebral body into the spinal canal. They all were located in the lumbar spine (L2–L5). The author considered this shift as a result of apophysis damage. Out of 10 patients four were teenagers, and the rest were adults. Clinically, the shift was expressed by pain and neurological symptoms: sensitivity disorders, muscle weakness despite the patients were neurologically intact. Surgical treatment (removal of the shifted fragment of the apophysis and of the corresponding intervertebral disc) allowed to completely stop the symptoms. Three adult patients in the thoracic region showed changes typical for Scheuermann's disease. The author suggests that patients with juvenile kyphosis have a tenden-

cy to the shift of the fragment of the apophysis dorsally. Ryan and Taylor [12] believed that such conditions are a peculiarity of Scheuermann's disease in relation to the lumbar spine.

Demiroz et al. [50] were the first to study the frequency of intracanal abnormalities in patients with Scheuermann's kyphosis. In 7 out of 132 patients (5.8 %), syringomyelia was found. The localization of the anomaly in five cases was lumbar, in two – thoracic (the total spread is from T9 to L4 vertebra). The width of the cavity ranged from 3 to 5 mm. All of them were located centrally. No correlation was found with the magnitude of the kyphotic strain. All patients were neurologically intact. Additional neurosurgical intervention was not required in any of the cases. Spinal deformity as a result of surgical correction was decreased from 75° to 32° on average. There were no neurological complications. MRI should be part of the obligatory preoperative examination.

Few authors have studied the frequency of neurological symptoms in Scheuermann's disease, and the results are controversial. Murray et al. [51], studied the natural history of Scheuermann's disease. They reported about 15 % of patients with signs of involvement of the spinal canal contents in the pathologic process. In 1996–2021, 215 patients with juvenile kyphosis were operated in the Clinic of Pediatric and Adolescent Vertebrology of the Novosibirsk Research Institute of traumatology and Orthopaedics n.a. Ya.L. Tsivyan. Only one patient had neurotic symptoms in the preoperative stage (less than 0.5 %) [17]. Cho et al. [52] showed the results of surgical treatment of 69 patients. Only 6 (9 %) of them showed neurological symptoms of varying severity. It is worth noting that only one patient had severe myelopathy. The results of surgical treatment (posterior approach – 5 cases, posterio-anterior approach – 1) were successful, but the authors do not provide details for each patient. Main findings: preoperative neurological examination is obligatory. The X-ray findings do not correlate with the identified neurological symptoms. An unimpaired MRI result may be associated with a spinal cord lesion, and, conversely, pathologic changes may be observed in neurologically intact patients during MRI. The magnitude of kyphosis and the age of patients are not risk factors for the spinal cord compression development.

The issue of identifying and treating patients with Scheuermann's disease and neurological symptoms has not been discussed in the domestic literature. As for the foreign literature, it is mostly the description of single observations and small groups. This review of a small number of publications is not the first of its kind. However, we found it reasonable and useful to be written, since even in this local issue, new data are emerging which can change our ideas both quantitatively and qualitatively. We were able to find a description of 38 cases of Scheuermann's disease. The course of this disease was complicated by the involvement of the spinal canal contents in the pathological process. The data found here is more than in the previously published reviews. However, this is certainly not all of it. Even today not all sources are available for information breakthroughs. Nevertheless, we dare to think that the presented picture largely reflects the true situation. Thus, the most common cause of neurological complications in Scheuermann's disease are intervertebral hernias, the vast majority of which are localized in the thoracic spine. There are 24 such cases in our review. The spinal cord compression by the anterior wall of the spinal canal and the posterior leaf of the dura mater is in the second place (11 cases). The third place is occupied by very few cases of compression of the spinal canal contents by extradural spinal cysts (3 cases). The age of patients varies quite widely, but young people clearly prevail. A significant relationship between the magnitude of kyphotic deformity and the risk of complications could not be detected.

The recent literature contains references to other potential causes of the development of the discussed complication: spinal epidural lipomatosis, shift of the ring apophysis of the lumbar vertebra, and syringomyelia. These are very rare conditions, but a spine surgeon must be aware about them. The collective experience indicates the need for a full-fledged preoperative examination of patients with Scheeuermann's disease for the purpose of detailed visualization of the spinal canal contents and possible correction of surgical approach.

Surgical techniques themselves have evolved over the years. The realization came quite quickly that laminectomy as a surgical approach in certain situations gives the surgeon great opportunities. However, the specialists should not forget about the prevention of the pathological condition, which is defined in the works of Ya.L. Tsivyan "as a disease of the laminectomized vertebral column". He was one of the first to define this condition, its pathogenesis, symptoms, and treatment in detail, and in a differentiated way – in relation to adult patients and children [53]. The need for reliable spinal stabilization throughout the laminectomy defect is the key to deal with this challenge. The stabilization technique (anterior spondylodesis, dorsal fixation with the help of modern tools or a combination of them) is defined by the peculiarities of the pathological process, the preferences of the surgeon and many other important circumstances. On the present stage of development of our specialty, we have quite sufficient opportunities to solve various problems. The most significant thing is a thorough examination of the patient and a well – founded choice of treatment strategies.

Conclusion

Scheuermann's disease is one of the most common causes forcing the surgeon to consider indications for surgical correction of spinal deformity (after idiopathic scoliosis and congenital deformities). Its course is rarely complicated by the development of neurological symptoms. However, the causes of these complications are various and require the most thorough preoperative examination. This allows doctors to correctly plan the surgical intervention, to achieve the optimal therapeutic effect, while avoiding unexpected complications.

Table 3
Spinal extradural cyst in patients with Scheuermann's disease (literature data)

Authors	Age of the patient	Sex	Cobb, angle deg.	Kyphosis apex	Vertebral arch	Time of symptoms	Neurological disorders (particular level)	Pain	Surgical intervention	Effect	Follow-up period	Myelography
Fiss et al. [42]	31	M	–	L1–L2, Schmorl's nodes at these levels	–	4 weeks	L2	Yes	Laminectomy L1, L2, cyst removal	Full recovery	2 weeks	MRI – compression of *conus medullaris*
Park et al. [43]	15	M	–	–	–	2 weeks	Spastic paraparesis	–	Laminectomy T11–T12, cyst removal, transpedicular fixation	Partial recovery	12 months	Cyst 14.5 × 7.4 × 75.0 mm, spinal cord is compressed
Adelstein [38]	15	F	85	–	–	5 months	Spastic paraplegia	–	Laminectomy, cyst removal	Significant recovery, increased kyphosis	–	–

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References

1. **Scheuermann HW.** Kyfosis dorsalis juvenilis. Ugeskr Laeger. 1920;82:385–393.
2. **Sorensen KH.** Scheuermann's Juvenile Kyphosis: Clinical Appearances, Radiology, Etiology and Prognosis. Copenhagen: Munksgaard, 1964.
3. **Palazzo C, Sailhan F, Revel M.** Scheuermann's disease: an update. Joint Bone Spine. 2014;81:209–214. DOI: 10.1016/j.jbspin.2013.11.012.
4. **Wenger DR, Frick SL.** Scheuermann kyphosis. Spine. 1999;24:2630–2639. DOI: 10.1097/00007632-199912150-00010.
5. **MacEven W.** The surgery of the brain and spinal cord. Br Med J. 1888;2:302–309.
6. **Scheuermann H.** Roentgenologic studies of the origin and development of juvenile kyphosis, together with some investigations concerning the vertebral epiphyses in man and in animals. Acta Orthop Scand. 1934;5:161–220. DOI: 10.3109/17453673408991327.
7. **Kienbock R.** Rontgendiagnostik der Knochen- und Gelenkkrankheiten, Heft IV; Degenerative Wirbelsaulenerkrankungen, Berlin and Wien: Urban & Schwarzenberg, 1936.
8. **Lonstein JE, Winter RB, Moe JH, Bradford DS, Chou SN, Pinto WC.** Neurologic deficits secondary to spinal deformity. A review of the literature and report of 43 cases. Spine. 1980;5:331–355. DOI: 10.1097/00007632-198007000-00007.
9. **Dommisse GF.** The blood supply of the spinal cord. A critical vascular zone in spinal surgery. J Bone Joint Surg Br. 1974;56:225–235.
10. **Gulledge WH, Brav EA.** Non-tuberculous thoracic kyphosis with paraplegia: A case report. J Bone Joint Surg. 1950;32:900–903.
11. **Bradford DS, Garcia A.** Neurological complications in Scheuermann's disease. A case report and review of the literature. J Bone Joint Surg Am. 1969;51:567–572. DOI: 10.2106/00004623-196951030-00017.
12. **Ryan MD, Taylor TK.** Acute spinal cord compression in Scheuermann's disease. J Bone Joint Surg Br. 1982;64:409–412. DOI: 10.1302/0301-620X.64B4.7096412.
13. **Normelli HC, Svensson O, Aaro SI.** Cord compression in Scheuermann's kyphosis. A case report. Acta Orthop Scand. 1991;62:70–72. DOI: 10.3109/17453679108993097.
14. **Kapetanos GA, Hantzidis PT, Anagnostidis KS, Kirkos JM.** Thoracic cord compression caused by disk herniation in Scheuermann's disease: a case report and review of the literature. Eur Spine J. 2006;15(Suppl 5):S553–S558. DOI: 10.1007/s00586-005-0053-0.
15. **Putz C, Stierle I, Grieser T, Mohr G, Gerner HJ, Furstenberg CH, Wiedenhofer B.** Progressive spastic paraplegia: the combination of Scheuermann's disease, a short-segmented kyphosis and dysplastic thoracic spinous processes. Spinal Cord. 2009;47:570–572. DOI: 10.1038/sc.2008.133.
16. **Sariali E, Panier S, Glorion C.** Mechanical spinal cord compression at the apex of a kyphosis: a propos of one case. Review of the literature. Eur Spine J. 2009;18 (Suppl 2):S160–S164. DOI: 10.1007/s00586-008-0733-7.
17. Mikhailovsky MV, Udalova IG, Lebedeva MN, Sarnadsky VN. Scheuermann's kyphosis with neurological symptoms. Hir. Pozvonoc. 2009;(4):50–52. In Russian. DOI: 10.14531/ss2009.4.50-52.
18. **Court C, Mansour E, Bouthors C.** Thoracic disc herniation: surgical treatment. Orthop Traumatol Surg Res. 2018;104:531–540. DOI: 10.1016/j.otsr.2017.04.022.
19. **Quint U, Bordon G, Preissi I, Sanner C, Rosenthal D.** Thoracoscopic treatment for single level symptomatic thoracic disc herniations: a prospective followed cohort study in a group of 168 consecutive cases. Eur Spine J. 2012;21:637–645. DOI: 10.1007/s00586-011-2103-0.
20. **Hott JS, Feiz-Erfan I, Kenny K, Dickman CA.** Surgical management of giant herniated thoracic discs: analysis of 20 cases. J Neurosurg Spine. 2005;3:191–197. DOI: 10.3171/spi.2005.3.3.0191.
21. **Bouthors C, Benzakour, Court C.** Surgical treatment of thoracic disc herniation: an overview. Int Orthop. 2019;43:807–816. DOI: 10/1007/s00264-018-4224-0.
22. **Muller R.** Protrusion of thoracic intervertebral disks with compression of the spinal cord. Acta Med Scand. 1951;139:99–104.
23. **Stambough JL, VanLoveren HR, Cheeks ML.** Spinal cord compression in Scheuermann's kyphosis: case report. Neurosurgery. 1992;30:127–130. DOI: 10.1227/00006123-199201000-00025.
24. **Van Landingham JH.** Herniation of thoracic intervertebral discs with spinal cord compression in kyphosis dorsalis juvenilis (Scheuermann's disease); case report. J Neurosurg.1954;11:327–329. DOI: 10.3171/jns.1954.11.3.0327.
25. **Roth MD, Taylor TK.** Acute spinal cord compression in Scheuermann's disease. J Bone Joint Surg Br. 1982;64:409–412. DOI: 10.1302/0301-620X.64B4.7096412.
26. **Turinese A, Raven C.** Complicance neurologicere nella osteochondrite vertebral jiuvanile. Psichiat e Neuropatia Giom. 1966;23:215–238.
27. **Yablon JS, Kasdon DL, Levine H.** Thoracic cord compression in Scheuermann's disease. Spine. 1988;13:896–898. DOI: 10.1097/00007632-198808000-00004.
28. **Lesoin F, Leys D, Rousseaux M, Dubois F, Villete L, Pruvo JP, Petit H, Jomin M.** Thoracic disc herniation and Scheuermann's disease. Eur Neurol. 1987;26:145–152. DOI: 10.1159/000116327.
29. **Bohlman HH, Zdeblick TA.** Anterior excision of herniated thoracic discs. J Bone Joint Surg Am. 1988;70:1038–1047.
30. **Bhojraj SY, Dandawate AV.** Progressive cord compression secondary to thoracic disc lesion in Scheuermann's kyphosis managed by posterolateral decompression, interbody fusion and pedicular fixation. A new approach to management of a rare clinical entity. Eur Spine J. 1994;3:66–69. DOI: 10.1007/BF02221442.
31. **Chiu KY, Luk KDK**. Cord compression caused by multiple disk herniation and intraspinal cyst in Scheuermann's disease. Spine. 1995;20:1075–1079. DOI: 10.1097/00007632-199505000-00016.
32. **Song KS, Yang JJ.** Acutely progressing paraplegia caused by traumatic disc herniation through posterior Schmorl's node opening into the spinal canal in lumbar Scheuermann's disease. Spine. 2011;36:E1588–E1591. DOI: 10.1097/BRS.0b013e31820f6958.
33. **Chiche L, Carlier RY, Siahou D, Nataf A, Hugeron C, Palazzo C.** Spinal cord ischemia in Scheuermann disease: a report of three cases. Joint Bone Spine. 2017;84:345–348. DOI: 10.1016/j.jbspin.2016.10.007.
34. **Zan C, Yang X, Wu M, Jiao J, Qu Y, Zhang S.** Acute Brown-Sequard syndrome caused by thoracic disc herniation in atypical Scheuermann's disease: a case report. Int J Chin Exp Med. 2017;10:12720–12725.
35. **Elsberg C, Dyke C, Brewer E.** The symptoms and diagnosis of extradural cysts. Bull Neurol Inst NY. 1934;3:395–417.
36. **Lehman E.** Spinal extradural cysts. Am J Surg. 1935;28:307–322.
37. **Cloward RB, Bucy PC.** Spinal extradural cyst and kyphosis dorsalis juvenilis. Am.J Roentgenol Rad Ther. 1937;38:681–706.
38. **Adelstein LJ.** Spinal extradural cysts associated with kyphosis dorsalis juvenilis. J Bone Joint Surg. 1941;23:93–101.
39. **Wise BL, Foster JJ.** Congenital spinal extradural cyst. Case report and review of the literature. J Neurosurg. 1955;12:421–427. DOI: 10.3171/jns.1955.12.4.0421.
40. **Nugent GR, Odom GL, Woodhall B.** Spinal extradural cysts. Neurology. 1959;9:397–406. DOI: 10.1212/wnl.9.6.397.
41. **Bodosi M.** Die angeborene spinale Zyste. Acta Neurochirurgica. 1971;23:275–283. DOI: 10.1007/BF01401858.
42. **Fiss I, Danne M, Hartmann C, Brock M, Stendel R.** Rapidly progressive paraplegia due to an extradural lumbar meningocele mimicking a cyst. Case report. J Neurosurg Spine. 2007;7:75–79. DOI: 10.3171/SPI-07/07/075.

43. **Park HY, Lee SH, Kim ES, Eoh W.** Spinal extradural meningeal cyst and Scheuermann's disease: coincidence or causative factor. Childs Nerv Syst. 2012;28:1807–1810. DOI: 10.1007/s00381-012-1774-0.
44. **Nabors MW, Pait TG, Byrd EB, Karim NO, Davis DO, Kobrine AI, Rizzoli HV.** Updated assessment and current classification of spinal meningeal cysts. J Neurosurg. 1988;68:366–377. DOI: 10.3171/jns.1988.68.3.0366.
45. **Abul-Kasim K, Schlenzka D, Selariu E, Ohlin A.** Spinal epidural lipomatosis: a common imaging feature in Scheuermann disease. J Spinal Disord Tech. 2012;25:356–361. DOI: 10.1097/BSD.0b013e31822631d3.
46. **Zhang Z, Liu Z, Zhu Z, Qiu Y.** Spinal epidural lipomatosis – an easily ignored secondary intraspinal disorder in spinal kyphotic deformities. BMC Musculoscelet Disord. 2017;18:112–117. DOI: 10.1186/s12891-017-1467-7.
47. **Kim K, Mendelis J, Cho W.** Spinal epidural lipomatosis: a review of pathogenesis, characteristics, clinical presentation, and management. Global Spine J. 2019;9:658–665. DOI: 10.1177/2192568218793617.
48. **Bruns J, Heise U.** Spastische Paraparese bei Morbus Scheuermann. Eine Kasuistik. Z. Orthop Unfall. 1994;132:390–393. DOI: 10.1055/s-2008-1039842.
49. **Soper J.** Lumbar posterior vertebral spur. Australas Radiol. 1988;32:343–347. DOI: 10.1111/j.1440-1673.1988.tb02750.x.
50. **Demiroz S, Ketenci IE, Yanic HS, Bayram S, Ur K, Erdem S.** Intraspinal anomalies in individuals with Scheuermann's kyphosis: is the routine use of magnetic resonance imaging necessary for preoperative evaluation? Asian Spine J. 2018;12:697–702. DOI: 10.31616/asj.2018.12.4.697.
51. **Murray PM, Weinstein SL, Spratt KF.** The natural history and long-term follow-up in Scheuermann kyphosis. J Bone Joint Surg Am. 1993;75:236–248. DOI: 10.2106/00004623-199302000-00011.
52. **Cho W, Lenke LG, Bridwell KH, Hu G, Buchowski JM, Dorward IG, Pahys JM, Cho SK, Kang MM, Zebala LP, Koester LA.** The prevalence of abnormal preoperative neurological examination in Scheuermann kyphosis: correlation with X-ray, magnetic resonance imaging, and surgical outcome. Spine. 2014;39:1771–1776. DOI: 10.1097/BRS.0000000000000519.
53. **Tsivyan YaL.** Injuries of the Spine. Moscow, 1971:213–266. In Russian.

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