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Degenerative Lumbar Scoliosis-Anamnestic, Clinical and Radiological Evaluation of 80 Patients needing Fusion Surgery

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Study Design: Retrospective review.

Objective: To analysing the anamnestic, clinical and radiological data in patients with “de novo”-scoliosis at time of operative decompression and fusion of the curve

Summary of Background Data: In spine literature degenerative lumbar scoliosis (DLS) gains more and more importance because of its increasing effect on the morbidity of the elder population; most of the natural history studies either describe patients with different causes of adult scoliosis or with different clinical importance of this deformation, i.e. both patients with only radiologic changes and with severe clinical symptoms, but to our knowledge there exists no study of natural history of a collective of patients with DLS needing fusion of scoliotic curve.

Methods: This study demonstrates in a retrospective manner the preoperative data of 80 patients aged 49-86 (av. 69) years with DLS treated by fusion of the scoliotic curve. Investigation included anamnestic data (previous operations, pain, spinal claudication, flatback-symptoms), clinical symptoms (neurologic deficit, flatback), radiologic data like pre-existing vertebral fractures, extension and degree of scoliosis, convexity and both degenerative and rotational spondylolisthesis. MRI was rated concerning central spinal stenosis, foraminal stenosis and severe and/or Modic-1-disc degeneration. Bone density measurements at time of operation were evaluated if available.

Results: Our data showed a 5:1 majority of female patients, but no difference in the age at fusion. There was no statistical difference between left-and right sided curve. Cobb angle averaged 23° and varied from 12° to 48°.

87% complained of severe low back pain, 28% of severe and 31% of medium radicular pain; altogether 23% had both severe low back and sciatic pain, 11% severe claudication. 21% had motoric deficits, mainly L5. Flatback symptoms were evaluated as severe in 7% and medium in 24%.

50% of patients had degenerative spondylolisthesis, in 56% at L4.

56% demonstrated rotational instability, max. at L3/4.

92% had severe and/or Modic-1-degenerative disc disease, 58% concerning either L2/3 or L3/4. 80% had spinal canal stenosis (included 17% previously decompressed patients); 76% demonstrated foraminal stenosis; between L1 and L3 90% concavesided, below L3 at both sides. Bone-density measurements at time of operation demonstrated in 81% normal density or osteopenia and only in 19 % osteoporosis.

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Conclusion: Late-Onset scoliosis is a special form of spinal degeneration, mostly in female patients (5:1), in contrary to idiopathic scoliosis with the convexity in same percentage right and left sided. Extension of scoliosis at time of fusion was between 2 and 6 segments, medium 3; Cobb-angle at time of operation was 23° (12 - 48°); the most included segments are L2/3 and L3/4; 90% demonstrate either severe or Modic-1- disc degeneration in at least 1 segment (max. L2/3 and L3/4), 2/3 of patients show lateral instability (max.L3/4) and spinal canal stenosis (max. L4/5 and L3/4); degenerative spondylolisthesis can be seen in 50% of these cases, mainly at L4; foraminal stenosis within the primary curve down to L4 occurs nearly exclusively at the concave side, between L4 and S1 either the concave side of the secondary lumbosacral curve or both sides can be included. The number of severe degenerative disc diseases correlated with subjective flatback-symptoms, but there was no correlation between rotational instability and subjective flat-back-symptoms or rotational instability and neurologic deficit.

Level and number of degenerative changes support the hypothesis that DLS starts mainly at the level of L2-L4 with later occurring degenerative changes below L4 like in non-scoliotic forms of degeneration.

The main symptoms leading to decompression and fusion are lumbar pain (87%) by severe degenerative disc disease and instability, sciatic pain (28%), neurologic deficit (21%) and claudication (10%) by spinal canal- and/or foraminal stenosis, flatback-symptoms (7%) and of course combinations.

Key words: degenerative lumbar scoliosis, de novo-scoliosis, natural history**,** radiologic evaluation, fusion.

In the last two decades of the twentieth century the study of natural history of adult scoliosis demonstrated differences between adult idiopathic curves, curves caused by neurologic disease as Parkinson and lumbar degenerative lumbar (“de novo”) scoliosis as an own entity (1-4). Adult scoliosis demonstrates a highly reduced health perception in SF-36 score (4).

As DLS gaines more importance caused by increasing age of population, it seems important to evaluate the pathology especially at the time when decision to operation is made.

To our knowledge this is the first investigation of natural history concerning patients with DLS treated by fusion of the scoliotic curve.

PATIENTS AND METHODS

80 patients, average age 69 (49-86) years with de novo-scoliosis and severe symptoms leading to fusion of the scoliotic curve or decompression in combination with fusion, operated between 2002 and 2012 were evaluated retrospectively. There were 66 female and 14 male patients.

- Anamnesis was evaluated concerning previous operations, pain at time of fusion (lumbar, radicular or both), graded as severe (VAS >7) or medium (VAS 4-7), spinal claudication (<100m walking distance) and flatback-symptoms*.
- Clinical investigation concerned neurologic deficits and flatback-symptoms*.

*As we had lateral X-rays of the complete spine in standing position only in a part of the cases, flat-back was rated as severe, if this was the leading complain in anamnesis and/or clinic and/or a pedicle substraction osteotomy was done; it was rated as medium, if it was described in the anamnesis and/or clinically as one of the symptoms.

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- Radiologic examination was done looking at pre-existing vertebral fractures, extension of scoliosis, side of convexity, degrees of scoliosis according to Cobb, localisation of degenerative spondylolisthesis (>3mm) and rotational instability (>3mm).
- Bone-density-measurements according to WHO-classification at time of operation were available in 37 cases.
- MRI was investigated for central and foraminal stenosis and either severe or Modic-1-disc narrowing.

RESULTS

Relation female/male was 5:1, without difference between in the age of operation. 41% had previous operations (26 decompressions, 5 short fusions, 2 vertebroplasties). 87% patients complained of severe, 12% of medium low back pain, only 1% had no lumbar pain**.** 28% had severe and 31% medium radicular pain. In 23% both severe low back and radicular pain were the indication for operation. 10% suffered from severe spinal claudication, 21% had motoric deficits, in two thirds L5; Flatback symptoms were evaluated as severe in 7%, as moderate in 24%, 69% had no problems with flatback.

Bone-density measurements at time of operation demonstrated in 81% normal density or osteopenia and in 19% osteoporosis.

13 patients (16%) had previous fractures of lumbar or low thoracic vertebral bodies.

Side of convexity demonstrated no significant difference (52% left, 48,% right). Cobb angle averaged 23° and varied from 12° to 48°.

Table 1 demonstrates the levels of included segments.

Number of included segments: 4% 6 segments, 14% 5 segments, 26% 4 segments , 40% 3 segments, 16% 2 segments (table 2)

Cranial end vertebra of scoliosis were Th10 in 2,5%, Th11 in 8%, Th12 in 35%, L1 in 28%, L2 in 24% and L3 in 2,5% (table 3).

Degenerative spondylolisthesis occurred in 50% of patients, in 13% of patients in 2 segments (2%-L1, 6%-L2, 25%-L3, 56%-L4, 11%- L5, table 4). Altogether 56% of degenerative olisthesis were seen at L4.

Rotational olisthesis occurred in 56%, in 20% of patients in two or three segments (16% L1/2, 26% L2/3, 38% L3/4 and 20% L4/5, table 5).

92% had severe and/or Modic-1-degenerative disc disease, 58% concerning either L2/3 or L3/4.

63% of the MRI showed a spinal canal stenosis (7% L1/2, 24% L2/3, 32% L3/4, 36% L4/5, 1% L5/S1, table 6). Additional 17% had no spinal stenosis but decompression before, therefore 80% all together demonstrated spinal canal stenosis.

76% had foraminal stenosis; between L1 and L3 90% at the concave side, at L4 25% concavesided, 20% convexsided and 50% below scoliosis, at L5 without predominance of

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the side. Altogether 79% had their foraminal stenosis at the concave side (stenosis caudal of curve not included).

The existence of degenerative spondylolisthesis (DS) led to a higher percentage of neurologic deficits (73% of patients with DS had neurologic deficits versus 44% without DS).

6% suffered from severe flatback (needing pedicle-subtraction-osteotomy), 24% had moderate-medium grade problems with flatback. The number of severe degenerative disc diseases had a certain correlation to subjective flatback-symptoms (correlation coefficient 0,512).

There was no correlation between rotational instability and subjective flat-back-symptoms or rotational instability and neurologic deficit.

DISCUSSION

The development of DLS can be caused by asymmetric disc disease at the lumbar level (5). Yasuda et al. found, that wedging segments in DLS had greater motion than nonwedging segments, but osteophyte formation provided restabilization (6). Kobayashi et al. (7) found, that >20% decrease in unilateral disc height or more than 5mm longer osteophyte on one side led to increased incidence of de novo scoliosis. Genetic predisposition by various typical copy number variations is discussed by Shin et al. (8). According to Jimbo et al. (9) smaller L4-size, unilateral osteophyte formation and lateral disc wedgening raise the risk of developing DLS.

The search of serum biomarkers by Hosogane et al. (10) demonstrates elevated serum collagen type II and procollagen type II C-propeptides, meaning higher levels of type II collagen synthesis and degradation; they conclude, that this enhanced turnover may be related to the development and progression of DLS. Histologic studies demonstrate, that muscle degeneration was more common on the concave side (11).

The influence of osteoporosis is discussed differently; while Ding et al. find a negative correlation between the angle of scoliosis and bone density (12), Pappou et al. describe significantly lower hip bone mineral density in scoliotic patients, but no correlation between curve magnitudy and severity of osteoporosis (13); our data suggest, that osteoporosis does not seem to enhance the danger of DLS.

Different life expectancy alone cannot explain the much higher percentage of female patients with clinically important DLS, as we found no difference of age between the sexes at time of operation. Kilshaw et al. found in a survey of 2765 abdominal/kidney/bladder radiographs a significantly higher prevalence of scoliosis and lateral olisthesis in female patients (14). If there genetic and/or hormonal factors are responsible can only be discussed.

As factors predicting progression of DLS apical vertebral rotation, deep-seated L5-vertebra and asymmetrical change in the disc space above and below the L3 or apical vertebra are discussed (15). Mean progression is described with 2,5° per year for patients older than 69 years and 1,5° for younger patients (16). As factor of the side of convexity the height of the apical vertebra was described by de Vries et al (17), which could not be seen in our study; we found no special cause of developing left-or right sided curves.

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Our special questions were about the state of DLS, degenerative changes with concomitant clinical symptoms at the time, when pain and disability were at a point, that conservative treatment failed totally and patients needed fusion of the curve. Which degenerative

changes were responsible for the severe clinical impairment ? Are there specific changes of DLS or general changes like in other forms of spinal degeneration as degenerative spondylolisthesis or multiple DDD without scoliosis?

Our results suggest that both in majority of included segments and in majority of severe or Modic-1-disc-changes the segments L2/3 and L3/4 were predominant. Further on, the shortest curves have their upper end vertebra at L2, 50% of these curves including only 2 segments down to L4 with a difference between 23° Cobb-angle of curves beginning of L1 and 20° beginning at L2.

From these results one can conclude, that the deformity starts between L2 and L4 by unilateral disc-narrowing. Like in the study of Liu H. et al. 2003 (18) and Liu W. et al 2009 (19) foraminal stenosis within the primary curve (mainly L3 und L4) was seen with 90% nearly exclusively at the concave side, demonstrating that concavesided disc narrowing and development of osteophytes are the main cause of foraminal stenosis. As Ploumis et al.(20) we find with 79% of foraminal stenosis a very similar percentage concerning the concave side of the curve altogether. As Ploumis et al (21) and in contrary to a finite element study of Kim et al (22) we found no significant influence of lateral rotatory olisthesis on the development of neural canal stenosis or neurologic symptoms; on the other hand we could evaluate an increase of neurologic problems in case of a degenerative spondylolisthesis, occurring mostly at L4 influencing the L5-root.

Although these observations may be modified by the relatively high percentage of previous decompression operations they could support our hypothesis of developing degenerative curves between L2 and L4 with more severe disc degeneration at these levels and foraminal stenosis caused by this asymmetry, while below L4 besides unilateral narrowing especially degenerative spondylolisthesis with concomitant spinal stenosis is added as a second factor of neurologic impairment. Like in lumbar degeneration without development of scoliosis degenerative spondylolisthesis occurs most often at L4, indicating, that this form of instability is not dependent from the deformity itself, but from the special anatomy and physiology of the L4/5 segment.

There seems to be a rather low percentage of typical claudication symptoms compared to the number of central canal stenosis, according to the observation of Grubb et al. (23), who found in DLS a lack of the classic feature of relief in a sitting position.

Clinical symptoms of flatback were seen altogether in nearly one third of patients and are a special problem in DLS; severe cases need a pedicle subtraction osteotomy to get a correct sagittal profile, moderate cases need some PLIFs and/or TLIFs to correct lumbar hypolordosis. A certain dependency of flatback on the number of high-grade DDDs can be regarded.

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