



IMED, INC.

1701 N. Greenville Ave. • Suite 202 • Richardson, Texas 75081
Office 972-381-9282 • Toll Free 1-877-333-7374 • Fax 972-250-4584
e-mail: imeddallas@msn.com

Notice of Independent Review Decision

DATE OF REVIEW: 07/01/08

IRO CASE #:

DESCRIPTION OF THE SERVICE OR SERVICES IN DISPUTE

Item in dispute: Anterior lumbar interbody fusion L4-S1, posterior lumbar decompression with posterolateral fusion and pedicle screw instrumentation at L5-S1.

A DESCRIPTION OF THE QUALIFICATIONS FOR EACH PHYSICIAN OR OTHER HEALTH CARE PROVIDER WHO REVIEWED THE DECISION

Board Certified Neurosurgeon

REVIEW OUTCOME

Upon independent review the reviewer finds that the previous adverse determination/adverse determinations should be:

Denial Upheld

INFORMATION PROVIDED TO THE IRO FOR REVIEW

1. MRI of the lumbar spine dated xx/xx/xx
2. Medical records Dr. dated 12/20/07 thru 06/09/08
3. Clinical note Dr. dated 12/30/07
4. Physical therapy record dated 01/31/08
5. Lumbar myelogram dated 02/04/08
6. Lumbar radiographs including flexion and extension dated 02/04/08
7. Psychiatric evaluation dated 03/19/08
8. Utilization review report dated 04/16/08
9. Letter of appeal dated 04/17/08
10. Utilization review determination dated 04/29/08
11. Letter of appeal dated 06/09/08
12. Independent diagnostic study review, Dr. dated 06/09/08
- 13. Official Disability Guidelines.**

PATIENT CLINICAL HISTORY [SUMMARY]:

The employee is a xx year old male who was reported to have sustained an injury to his low back on xx/xx/xx. On this date, the employee reported falling backwards on a ramp with the acute onset of low back pain which he described as a constant deep numbing ache with intermittent radiation to the bilateral lower extremities, left side greater than right, with associated numbness and tingling in a nondermatomal distribution.

The employee initially came under the care of Dr. who referred the employee for an MRI of the lumbar spine on 12/14/07. This study reported lumbar lordotic straightening suggesting muscular pain or spasm. At L2-L3 and L3-L4, there were 2-3 mm symmetric annular bulges, reduced interspace width by an estimated 50% and associated drying of the disc substance. At L4-L5, there was a prominent or estimated 7-8 mm posterior central to right paracentral discal substance protrusion that severely indented the expected thecal sac contours and severely stenosis the central canal. The interspace widths are reduced by 60-70% and drying of the disc substance was associated. At L5-S1, there was a 3-4 mm posterior central discal substance protrusion. Substance minimally indented expected thecal sac contours. Drying of the disc substance was associated.

The employee was subsequently seen in follow up on 12/30/07. The employee reported that he was having a lot of pain and difficulty sleeping. He had pain in the lumbar area and wanted some pain medications. He was diagnosed with contusion of the buttocks, low back strain, lumbar radiculopathy, and sprain/strains of the ankle and foot. He was provided oral narcotics, placed on modified activity, and was referred to Dr..

On 01/10/08, the employee was seen by Dr.. He reported the history above. It was reported that the employee was status post physical therapy with no significant improvement in his symptomatology. He currently described his pain level as 9/10 on the VAS scale with worsening symptomatology after prolonged sitting or standing. The employee denied worsening symptomatology with coughing, sneezing, or valsalva. He also described intermittent urinary incontinence with an inability to stop the flow. He denied bowel or bladder dysfunction at that time. His past medical history was reported to be noncontributory. His past surgical history was none. The employee was a smoker who smoked one pack per day and uses alcohol socially. On physical examination, the employee was alert and oriented times three. He had adequate attention span and an ability to concentrate. Cranial nerves II-XII were intact.

Cervical range of motion was full. Lumbar range of motion was decreased in forward flexion secondary to pain. Motor examination revealed 4/5 strength in the tibialis anterior, EHL, and gastrocnemius muscle on the left, otherwise 5/5 throughout. Deep tendon reflexes were 1+ in the left ankle jerk, otherwise 2+ throughout and symmetrical. Plantar responses were flexor bilaterally. Gait was antalgic. The employee had difficulty with toe and heel walking, less difficulty with tandem walk. Straight leg raising was positive bilaterally at 50 degrees. Sensory examination revealed a hypoesthetic region in both the L5 and S1 distributions on the left to pinprick and light touch. Radiographic examination MRI of the lumbar spine was reviewed dated 12/14/07, which was reported to demonstrate a severe central disc herniation at L4-L5 approximately 7-8 mm with near complete canal compromise; however, nondiagnostic due to motion artifact on axial images. There was decreased disc height and disc desiccation noted at L4-L5 as well as bilateral foraminal stenosis in the left side greater than right with Modic type 2 changes paracentrally and toward the left with left sided foraminal stenosis and indentation of the left S1 nerve root sheath. There was decreased disc height and disc desiccation at L5-S1 as well as Modic changes type 2. The employee was diagnosed with lumbar disc displacement, lumbar mechanical lowback pain, lumbar radiculitis, and lumbago. Treatment options were discussed with the employee from doing nothing to continuing physical therapy to possible evaluation for epidural steroid injections to possible surgical intervention. The employee was subsequently recommended to continue physical therapy, undergo evaluation for epidural steroid injections, and undergo a CT myelogram.

On 01/31/08, the employee was seen in physical therapy and noted to have completed seven visits of physical therapy at that time.

On 02/04/08, the employee was referred for lumbar myelogram and CT. The report of lumbar myelogram indicated an attempt was made at an L2 lumbar puncture but CSF could not be aspirated, and therefore, a second stick was made at the L2 level. Again no CSF could be aspirated; however, the lateral film documented that the needle was in the subarachnoid space, and therefore, contrast was injected and found to be in a normal location in the subarachnoid space. The myelographic images demonstrated no contrast opacification below the L4-L5 interspace. Flexion and extension views demonstrated no abnormal subluxation. Due to the employee's size, images were of limited quality. There were mild posterior indentations of the thecal sac noted at L2-L3 and L3-L4. The post myelogram CT indicated a mild diffuse disc bulge present. The overall canal diameter was within normal limits. Posterolateral disc bulging did result in mild bilateral neural foraminal narrowing at L1-L2. At L2-L3, there was a moderate diffuse disc bulge of the annulus fibrosis present. The overall canal

diameter was adequate. Posterolateral disc bulging was present without significant neural foraminal encroachment. At L3-L4, there was a moderate diffuse bulge of the annulus fibrosis. There was moderate degenerative facet and ligamentum flavum hypertrophy present. There was concentric encroachment of the thecal sac and bony AP diameter was adequate, although the thecal sac diameter was diminished with a measurement of 8 mm. Posterolateral disc bulging mildly narrowed the neural foramina. At L4-L5, vacuum disc phenomenon was present. Due to the presence of a large disc herniation, the thecal sac was not identified nor was it opacified with contrast material making the dimensions of this presumed herniated disc impossible to accurately quantify. Sagittal images demonstrated a soft tissue protrusion from the interspace consistent with a disc herniation. The thecal sac was not definitely identified on the axial images, and it was presumably severely compressed. There was a severe degenerative facet hypertrophy present. Posterolateral disc bulging or herniated disc material severely narrowed the neural foramina. In the paramidline images, there was soft tissue attenuation which extended caudally which may be related to volume averaging of the thecal sac, although caudally extruded disc material cannot be identified. At L5-S1, there was a retrolisthesis of L5 relative to S1. There was prominent anterior osteophyte formation present. A moderate broad-based traction disc bulge was present. Of note was a soft tissue attenuation extending into the left L5 neural foramen. This may be related to extruded disc material, although a neoplastic process could not be excluded. Disc bulging at the L5-S1 level narrowed the AP canal diameter although within normal limits. There was severe narrowing of the left subarticular and lateral recess as well as moderate bilateral neural foraminal narrowing. Seven views of the lumbar spine were also performed on this date. This study demonstrated no abnormal subluxation on flexion and extension. Even on these delayed flexion and extension views, no intrathecal contrast was present below the L4-L5 interspace.

On 02/22/08, the employee was seen by Dr. in follow up. There was no improvement in his condition. He continued to report constant deep numbing ache with intermittent radiation of the bilateral lower extremities, left greater than right. His physical examination was grossly unchanged with the exception of 3+/5 strength in the tibialis anterior and EHL and gastroc on the left. Dr. reviewed the imaging studies and opined that at L5-S1 there was a retrolisthesis of 3-4 mm of L5-S1 with an associated disc protrusion of 5 mm with bilateral foraminal stenosis, left side greater than right. He reported there was severe left sided foraminal and lateral recess stenosis as well. There was decreased disc height at both L4-L5 and L5-S1 with Modic type 3 changes. He opined that due to the failure of conservative care including physical therapy, current neurologic status with evidence of cauda equina syndrome with urinary

incontinence, evidence of severe canal stenosis with complete myelographic block at L4-L5, bilateral foraminal stenosis at L4-L5 and L5-S1, retrolisthesis of L5 on S1 with evidence of segmental instability, that the employee would benefit from an anterior lumbar interbody fusion at L4-L5 and L5-S1 with posterior lumbar decompression with posterolateral fusion and pedicle screw instrumentation at L4-L5 and L5-S1. The employee was reported to be willing to proceed with operative intervention.

On 03/19/08, the employee was referred to, Ed.D, Psychologist, for preoperative psychiatric evaluation. Based upon the clinical interview and objective testing, there were no contraindications for surgery with this employee. He reported the employee displays an essentially normal mental status examination with none to mild symptoms of depression that are physiologically based.

On 04/16/08, a peer review was performed by Dr.. Dr. non-certifies the request for anterior lumbar interbody fusion L4-S1 with posterior lumbar decompression and posterolateral fusion with pedicle screw instrumentation, two day inpatient stay, and an assistant surgeon. Dr. reported that there was limited scientific evidence about the long-term effectiveness of fusion for degenerative disc disease compared with natural history, placebo, or conservative treatment. He indicated that the requesting physician had failed to demonstrate instability in the spine which would be required for a fusion. Based upon the clinical information submitted for this review and the evidence-based guidelines, the request was not certified.

On 04/17/08, Dr. submitted a letter of rebuttal. He reported that the employee had failed conservative care. He noted the employee's previous history; however, he reported that the employee had failed conservative care which included physical therapy, injection therapy, and concurrent pain duration of greater than six months.

A second request was submitted on 04/29/08. This case was reviewed by Dr. This request was non-certified. Dr. noted that the employee was reported on the surgeon's note as having a retrolisthesis and segmental instability. However, this was not reported either through the MRI or CT myelogram. Plain film studies revealed no subluxation on flexion and extension and no evidence of instability. He reported that the employee's examination and imaging studies would be conducive to a possible discectomy and decompression; however, given the lack of instability, fusion surgery was not warranted.

On 06/09/08, a letter of appeal was submitted by Dr.. Dr. cited the **Official Disability Guidelines** and reported that all pain generators were identified and treated, all physical medicine, manual therapy interventions were completed, radiographs demonstrating spinal instability and/or myelogram, CT myelogram or discography and

MRI demonstrating disc pathology, spine pathology was limited to two levels and psychosocial screen with confounding issues addressed. Dr. indicated a request was made for independent radiologist interpretation. He reported at L4-L5 there was a large disc bulge. The combination caused spinal canal stenosis and complete block of the flow of contrast on this employee's myelogram. At L5-S1, there was a 3 mm retrolisthesis of L5-S1 and a 4 mm annular disc bulge. He further reported in his letter that if in fact partial agreement for either the L4-L5 or L5-S1 was deemed to be the only levels that were concluded as supported by the submitted documentation, that he would accept and move forward in accordance with the determination. He further reported that if a partial approval was concluded, it was expected to create a potential failed back syndrome requiring a second surgery.

An independent review of the diagnostic tests was performed by Dr. on 06/09/08. Dr. opined that the employee had a congenitally small lumbar spinal canal, moderate disc desiccation and degenerative hypertrophic spondylosis at L2-L3, L4-L5, and L5-S1. There was mild disc desiccation and degenerative hypertrophic spondylosis at L1-L2 and L3-L4. There were multiple small Schmorl's node deformities along the vertebral body endplates from T11 through L4. The location and contiguity of these lesions was likely due to remote Scheuermann's disease. There was a mild 2 mm disc bulge at L1-L2. There was a moderate sized 4 mm disc bulge at L2-L3 which moderately effaced the thecal sac and mild central spinal canal stenosis and moderate narrowing in the lateral recesses in both foramina. She reported a mild 2 mm disc bulge at L3-L4 which mildly impinged upon the thecal sac. There was also mild degree of ligamentum flavum hypertrophy at this segment. There was a large disc bulge at L4-L5 which broadly impinged upon the thecal sac and both of the L5 nerve roots in the lateral recesses and the L4 nerve roots in both foramina. There was a mild degree of ligamentum flavum hypertrophy and moderate degenerative facet joint hypertrophy. The combination caused severe central spinal canal stenosis and effacement of both of the lateral recesses. It also resulted in a complete block in the flow of contrast on this employee's myelogram through this segment and nonopacification of the L4 and L5 nerve root sheaths. There was a grade I retrolisthesis of L5 on S1 and a moderate sized 4 mm disc bulge at L5-S1 which mildly impinged on the thecal sac and both the S1 nerve root sheaths in the lateral recesses. There was a mild degree of degenerative facet joint hypertrophy at this segment. There were Modic type II changes along the vertebral body end plates at L4-L5. There was moderate bilateral foraminal stenosis at L4-L5 and on the right at L5-S1. Mild bilateral foraminal stenosis at L2-L3 and severe left foraminal stenosis at L5-S1 was noted.

ANALYSIS AND EXPLANATION OF THE DECISION INCLUDE CLINICAL BASIS, FINDINGS AND CONCLUSIONS USED TO SUPPORT THE DECISION.

I would concur with the two previous reviewers in that anterior lumbar interbody fusion L4-S1 posterior lumbar decompression with posterolateral fusion and pedicle screw instrumentation at L5-S1 would not be supported by the submitted clinical information.

The submitted clinical records indicate that the employee sustained an injury to his low back as a result of a slip and fall occurring on xx/xx/xx. I would note that at approximately four months post date of injury, Dr. had submitted his initial request for operative intervention. The submitted clinical records indicate that at the time of initial consideration the employee had been treated with oral medications and physical therapy. The records allude to a possible referral to pain management for interventional procedures; however, there were no records which support this.

The employee was subsequently referred for CT myelography which is noted above and indicates very significant pathology at the L4-L5 level secondary to a disc herniation which resulted in myelographic contrast block. The employee is further noted to have multiple levels of degenerative changes most significant at L5-S1. On 02/04/08 the employee underwent dynamic radiographs which show no abnormal subluxation. The employee is reported to have a retrolisthesis of 3-4 mm of L5 on S1; again this was noted to be stable with no translation on dynamic studies.

The records indicate that the employee did undergo preoperative psychiatric evaluation and was cleared. The **Official Disability Guidelines** report that fusion should not be considered within the first six months of symptoms except for fracture, dislocation, or progressive neurologic loss. It reports indications for spinal fusion may include a neural arch defect, segmental instability that is objectively demonstrable, primary mechanical back pain; however, it notes in cases of worker's compensation employees outcomes related to fusion may have other confounding variables which may affect the overall success of the procedure which should be considered. They report that there is a lack of support for fusion for mechanical low back pain for subjects with failure to participate effectively in all active rehabilitation preoperatively with a total disability over six months and active psychiatric diagnosis. When recommended the ODG reports include all of the following that all pain generators are identified and treated, all physical medicine, manual therapy interventions are completed, x-rays demonstrating instability and/or myelogram, CT myelogram or discography and MRI demonstrating disc pathology, spine pathology is limited to two levels with confounding issues addressed and if the employee is a smoker he refrains from smoking for six weeks prior to surgery. Given the available medical records and the previous reviews, it would be noted that the employee does not have any instability although there is evidence of retrolisthesis of L5 on S1, and it has not conclusively been proven that the employee's pain generators are the L4-L5 or the L5-S1 discs. The employee has clear pathology on imaging studies which would account for his symptoms.

In conclusion, given the lack of documented instability, clear evidence of pathology and lack of documentation to support that the employee has failed all conservative care, the requested operative intervention would not be considered medically necessary.

A DESCRIPTION AND THE SOURCE OF THE SCREENING CRITERIA OR OTHER CLINICAL BASIS USED TO MAKE THE DECISION:

1. The Official Disability Guidelines, 11th edition, The Work Loss Data Institute.
2. The American College of Occupational and Environmental Medicine Guidelines; Chapter 12.
3. Deyo RA, Natchemson A, Mirza SK, Spinal-fusion surgery - the case for restraint, N Engl J Med. 2004 Feb 12;350(7):722-6
4. Gibson JN, Waddell G. Surgery for degenerative lumbar spondylosis: updated Cochrane Review. *Spine*. 2005 Oct 15;30(20):2312-20.
5. Atlas SJ, Delitto A. Spinal Stenosis: Surgical versus Nonsurgical Treatment. Clin Ortho Relat Res. 2006 Feb; 443:198-207.
6. Resnick DK, Choudhri TF, Dailey AT, Groff MW, Khoo L, Matz PG, Mummaneni P, Watters WC 3rd, Wang J, Walters BC, Hadley MN; American Association of Neurological Surgeons/Congress of Neurological Surgeons. Guidelines for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 7: intractable low-back pain without stenosis or spondylolisthesis. J Neurosurg Spine. 2005 Jun;2(6):670-2.