Neurosurgical Review
https://doi.org/10.1007/s10143-018-01065-1

REVIEW

Posterior epidural migration of herniated lumbar disc fragment: a literature review

Alaa Eldin Elsharkawy[1,2] · Anne Hagemann[3] · Peter Douglas Klassen[1]

Received: 1 July 2018 / Revised: 3 December 2018 / Accepted: 4 December 2018

Abstract

Herniated disc fragments' migration to posterior epidural locations is a very rare pathological condition, and the mechanism is not well understood. Posterior epidural migration may lead to serious neurologic problems; however, its diagnosis and treatment are challenging. We searched PubMed and Google Scholar, using various keyword combinations, and found 111 cases of posterior epidural disc migration in the lumbar region reported between 1973 and 2018. There were 89 (80.2%) men and 22 (19.8%) women. The mean age at surgery was 54.05 years (range, 26–83 years); the mean duration of complaints was 26.3 days. The locations were at the L3–L4 level in 41 cases (36.9%), the L4–L5 level in 37 (33.3%), the L2–L3 level in 21 (18.9%), the L5–S1 level in 8 (7.2%), and the L1–L2 in 4 (3.6%). The disc fragment appeared as hypointense and isointense in 60.3% and 33.8%, respectively, of cases in T1-weighted magnetic resonance imaging (MRI) and as hyperintense in 68.5% of cases on T2-weighted MRI. The initial symptoms were cauda equina syndrome, radiculopathy, and low back pain in 58 (52.2%), 52 (46.8%), and 12 (10.8%) patients, respectively. In addition, 107 patients (96.4%) underwent surgical treatment and 4 (3.6%) underwent conservative treatments, with total recovery, and subtotal recovery in 73 (65.8%), and 38 (34.2%), respectively. We found significant differences between patients at different ages with regard to the level of disc herniation ($\eta = 0.405$, $p = .001$): patients with a higher level of disc herniation were, on average, older. There was no significant difference in outcome between male and female patients, $\chi^2(1) = 0.591$, $p = .469$, or between patients with upper and lower lumbar spine, $\chi^2(1) = 0.027$, $p > .999$. Careful history documentation, clinical examinations, and contrast material–enhanced MRI with laboratory tests could help reveal herniated disc fragment and rule out several other pathological processes. In most cases, surgical treatment produced favorable outcomes.

Keywords Lumbar disc herniation · Disc fragment · Dorsal epidural

Introduction

Degenerative disc disease and its sequelae are common health problems [100]. The strong annulus fibrosis keeps the nuclear material inside the intervertebral spaces. Degenerative changes of the annulus may cause it to bulge or the nuclear material to herniate into the spinal canal [15]. Herniation (protrusion and extrusion) is defined as a localized displacement of disc material beyond the intervertebral disc space limits [31]. Extrusion of displaced disc material, which has completely lost any continuity with the parent disc, is defined as sequestration [22, 31]. According to Fardon et al., "The term migration may be used to signify displacement of disc material away from the site of extrusion, regardless of whether sequestrated or not" [31].

Disc sequestration accounts for about 28.6% of all disc herniations [93]. Herniated disc fragments migrate in all directions in the spinal canal; caudal and paracentral displacements are the most common patterns [18, 72].

The posterior epidural migration is a very rare pathological entity. Posterior epidural disc fragments were first reported in 1973 [66], but the reasons why disc fragments migrate posteriorly are not well understood. Posterior epidural migration may lead to serious neurological problems; however, it is difficult to diagnose and to treat.

✉ Alaa Eldin Elsharkawy
alaa.elsharkawy@icould.com; alaa-eldin.elsharkawy@hospital-lingen.de

[1] Department of Neurosurgery, Bonifatius Hospital, Wilhelmstr. 13, 49808 Lingen, Germany

[2] Neurosurgery Teaching Program, Traditional Medicine University, Yerevan, Armenia

[3] Department of Psychology, Bielefeld University, Bielefeld, Germany

Published online: 06 January 2019

Despite its importance, no large comprehensive studies of this type of migration have been reported. Only case reports and few case series are found in the literature [18, 28, 72]. Our aim is to review what is currently known about the pathophysiological features and treatment of posterior epidural migration.

Patients and methods

We performed PubMed and Google scholar searches using various combinations of keywords, including "posterior epidural migration of sequestered disc," "disc sequestration" "disc fragment," and "case report of unusual disc migration." Case reports of thoracic (6 cases) and cervical migration (3 cases) were excluded from this review. One Spanish case report was included because Spanish is the first language of the senior author of this article.

We reviewed all case reports about posterior migration of disc fragments, including those that were parts of case series that contained complete information about the position of disc fragments, clinical presentation, treatment, and outcome. Cases with incomplete data and with mainly dorsolateral disc fragment were excluded. We identified 120 papers, 111 of which were found to meet the study inclusion criteria. All cases are described in Table 1.

To compare outcomes between subgroups of patients, we used χ^2 tests. Coefficient η was used to describe the association between age and the level of disc herniation.

Results

We found 111 cases of posterior epidural disc displacement in the lumbar region reported between 1973 and 2018. Of the affected patients, 89 (80.2%) were men and 22 (19.8%) were women. The mean age at surgery was 54.05 years (range, 26–83 years); the mean duration of symptoms was 26.3 days (range, 0.25–300 days). (Cases of chronic low back pain lasting years and of acute pain of unknown duration were not included.)

The locations were at the L3–L4 level in 41 cases (36.9%), the L4–L5 level in 37 (33.3%), the L2–L3 level in 21 (18.9%), the L5–S1 level in 8 (7.2%), and the L1–L2 level in 4 (3.6%).

Magnetic resonance imaging (MRI) was performed in 95 patients (85.6%), computed tomographic (CT) scanning in 30 patients (27%), and CT and magnetic resonance myelography in 12 patients (10.8%). Ring enhancement was reported in 98.1% of patients who underwent imaging with contrast material. Disc fragments appeared as hypointense and isointense in 60.3% and 33.8% of cases, respectively, on T1-weighted MRI and as hyperintense in 68.5% of cases on T2-weighted MRI. Discography was performed in one case.

The presenting symptoms were cauda equina syndrome, radiculopathy, and low back pain (LBP) in 58 (52.2%), 52 (46.8%), and 12 (10.8%) cases, respectively. A history of trauma or lifting a heavy object was reported by 17 patients (15.3%). In addition, 107 patients (96.4%) underwent surgical treatment and 4 (3.6%) underwent conservative treatment; total recovery was achieved in 73 (65.8%) and subtotal recovery in 38 (34.2%). Of the surgical patients, 62 (57.9%) underwent laminectomy, 8 (7.4%) underwent multilevel laminectomy, and 7 patients (6.5%) underwent laminectomy plus fusion, additional discectomy underwent in 36 (33.6%). Details about the type of surgery and outcome are listed in Table 2.

Although posterior epidural migration was reported more often in male patients and in the upper lumbar spine (L1–L2, L2–L3, L3–L4), there was no difference in outcome between male and female patients, $\chi^2(1) = 0.591$, $p = .469$, or between those with affected upper and lower lumbar spine, $\chi^2(1) = 0.027$, $p > .999$. There were significant differences between patients of different ages with regard to the level of disc herniation ($\eta = 0.405$, $p = .001$): patients with a higher level of disc herniation were, on average, older (Tables 3, 4, and 5).

Discussion

Natural history and epidemiology of dorsal disc migration

Dorsal disc herniation is a very rare pathological entity. Most affected patients present clinically with serious pain or a neurological disorder and receive treatment, which makes it difficult to study the natural history. Furthermore, there are no data regarding disc posterior migration among nonsurgical patients and healthy individuals. Moreover, among surgical patients, an unusual disc migration is generally rare; Nievas et al. [80] reported an incidence of 0.4% among 3000 patients. In the case of posterior migration, study results varied greatly: Sengoz et al. [98] reported an incidence of 0.27%, Kahn et al. [51] reported 0.9%, and Akhaddar et al. [4] reported 1.04%. In most cases, the posterior migration site was at the L3–L4 level (75%) and the L4–L5 level (25%) [98].

The natural history of typical disc herniation is well studied; in most cases, it resolves and heals spontaneously [17]. Researchers have reported effective reduction and spontaneous absorption of typical disc sequestrations in 43 to 88% of affected patients and extruded discs with complete resolution in approximately 15% [14, 101].

Moreover, risk factors for posterior disc herniation are unknown and have been not studied. According to our review, 17 patients (15.3%) had a history of lifting heavy loads or other hard work. Previous studies showed that in case of usual disc herniation, occupational factors, such as heavy physical loading, may accelerate spinal degeneration and its sequelae

Table 1 All cases included in the review

Reference and years	Age	Sex	Level	Radiology	Duration	Clinical presentation	Surgery	Outcome
Lombardi 1973 [66]	58	M	L2–3	Myelogram	2 years	CES	L + E	Full recovery
	54	M	L4–5	Myelogram	2 months	RP	L + E	Full recovery
Lichtor 1989 [64]	61	M	L2–3	CT + myelo/MRI	1 month	RP/LBP	L + E	Full recovery
Lutz et al. 1990 [67]	30	M	L4–5	CT + myelo	5 days	CES	L + E	Full recovery
Hirabayashi et al. 1990 [40]	58	M	L3–4	MRI, CT + myelo	4.5 months	CES	L + E	Improvement
Sekerci 1992 [95]	58	M	L3–4	Myelogram	2 months	CES	L + E	Improvement
Sakas 1995 [90]	70	M	L4–5	CT	10 weeks	RP, LBP	L + E	Full recovery
Bonaroti and Welch 1998 [9]	51	M	L2–3	MRI	12 h	CES	2 level L + E + discectomy	Improvement
Hodges et al. 1999 [41]	56	M	L4–5	MRI	7 days	LBP	L + E	Full recovery
Robe 1999 [88]	68	M	L3–4	MRI, MYL, CT	Acute	RP	L + E + discectomy	Full recovery
	41	F	L3–4	CT + myelo + MRI	14	CES	L + E + discectomy	Full recovery
Neugroschl 1999 [78]	57	M	L2–3	CT + myelo + MRI	15 days	RP, LBP	2 level L + facetectomy discectomy	Full recovery
	64	M	L2–3	CT + myelo + MRI	14 days	RP	Bilateral L + E	Full recovery
Saruhashi et al. 1999 [92]	44	F	L5–S1	CT + myelo + MRI	1 month	RP	L + E	Full recovery
Lisai et al. 2000 [65]	63	M	L3–4	MRI	12 h	CES	L + facetectomy discectomy	Full recovery
Dosoglu et al. 2001 [22]	47	M	L3–4	MRI	15 days	CES	Fenestration discectomy	Full recovery
Sen et al. 2001 [96]	36	M	L4–5	MRI	10 h	CES	L + discectomy bilateral	Full recovery
Eysel 2001 [30]	45	M	L3–4	MRI + CT	8 weeks	CES	L + E	Full recovery
	37	F	L4–5	CT	7 weeks	RP	L + E	Full recovery
	41	M	L3–4	CT	11 weeks	LBP	L + E	Full recovery
Senel 2003 [97]	44	M	L3–4	MRI	5 days	LBP	PHL + E	Full recovery
Kuzeyli 2003 [59]	47	F	L2–3	MRI	4 months	CES	HL + E	Full recovery
	62	F	L1–2	MRI + CT	25 days	CES	HL + E	Full recovery
	49	M	L4–5	MRI	15 days	LBP	L + E	Full recovery
Kim 2003 [53]	60	F	L3–4	MRI	4 days	RP/CES	2 level L + E	Subtotal recovery
Kim 2004 [54]	44	M	L4–5	CT	6 h	CES	L + E	Subtotal recovery
Walsh 2004 [114]	62	M	L3–4	MRI	24 h	CES	2 level L + E	Full recovery
Tatli 2005 [105]	54	M	L5–S1	MRI	2 days	CES	L + E	Improved
	53	M	L3–4	MRI	2 days	CES	L + E	Full recovery
Lakshmanan 2006 [60]	58	M	L4–5	MRI	1 month	RP	L + E	Full recovery
	28	F	L4–5	MRI	3 months	RP	L + E	Full recovery
Chen et al. 2006 [13]	75	M	L2–3	MRI	2 weeks	RP	L + E	Improved
Choi 2007 [16]	68	M	L4–5	MRI	3 weeks	RP	HL + E	Full recovery
	74	F	L3–4	MRI	3 days	RP	PHL + E	Subtotal recovery
Mobbs 2007 [72]	32	M	L4–5	MRI	3 months	RP	Conservative	Full recovery
EL Asri 2008 [24]	42	M	L5–S1	CT	1 month	Radiculopathy	L + E discectomy	Subtotal recovery
	36	M	L5–S1	CT	1 month	CES	L + E discectomy	Subtotal recovery
Jose et al. 2008 [5]	45	M	L4–5	MRI	1 week	CES	HL + discectomy	Full recovery
Derincek 2009 [19]	60	F	L1–2	MRI	1 month	RP/LBP	L + E + discectomy	Full recovery
Nievas 2009 [80]	83	M	L3–4	MRI	2 months	RP, LBP	HL + E	Full recovery
	45	M	L4–5	MRI	4 weeks	RP, LBP	PHL + E	Full recovery
	67	M	L4–5	MRI	3 months	Radiculopathy	HL + partial facetectomy	Full recovery
	60	F	L5–S1	MRI	2 months	CES	HL	Full recovery
	59	F	L2–4	MRI	4 weeks	Radiculopathy	2 HL + discectomy	Full recovery
Kim 2010 [55]	73	M	L4–5	CT/MRI	Acute	CES	Bilateral laminotomy	Improved
Teufack 2010 [106]	49	M	L4–5	CT/MRI	1 week	RP, LBP	L + E	Full recovery
Eksi 2010 [23]	50	M	L3–4	MRI	20 day	RP	L + E	Full recovery
Huang et al. 2011 [43]	78	F	L3–4	MRI	10 years	Radiculopathy	L + fusion	Full recovery
Akhaddr 2011 [4]	60	F	L2–3	MRI	1 week	CES	L + E	Full recovery
	43	M	L5–S1	CT	2 months	CES	L + E + discectomy	Improvement
	48	M	L3–4	MRI	2 weeks	CES	L + E + discectomy	Improvement
	67	M	L3–4	CT	1 year	Radiculopathy	L + E + discectomy	Full recovery

Table 1 (continued)

Reference and years	Age	Sex	Level	Radiology	Duration	Clinical presentation	Surgery	Outcome
	59	M	L3–4	CT	1 month	Radiculopathy	L + E + discectomy	Full recovery
	35	M	L4–5	CT	3 months	CES	L + E + discectomy	Improvement
Sengoz 2011 [98]	43	F	L4–5	MRI	3 days	CES	Mini HL + E	Full recovery
	72	M	L3–4	CT	1 day	RP/CES	Mini laminotomy + E	Full recovery
	42	M	L3–4	MRI	10 days	CES	HL + E	Subtotal recovery
	44	F	L3–4	MRI	7 days	RP/CES	L + E	Full recovery
	54	M	L3–4	MRI	3 days	RP/CES	L + E	Full recovery
	55	M	L3–4	MRI	5 days	CES	L + E	Full recovery
	39	M	L3–4	MRI	4 days	Radiculopathy	L + E	Subtotal recovery
	34	M	L4–5	MRI	2 days	CES	L + E	Full recovery
Hur 2011 [44]	50	M	L1–2	MRI	10 days	RP/LBP	L + E	Full recovery
	69	M	L2–3	MRI	15 days	CES	Microdiscectomy	Improvement
	60	F	L3–4	MRI	5 days	Radiculopathy	L + E	Full recovery
Gonçalves 2012 [36]	51	M	L2–3	MRI	3 days	RP/LBP	HL	Improvement
Talavera 2012 [48]	76	M	L3–4	MRI	4 day	CES	L + fusion	Improvement
Ju et al. 2012 [115]	74	M	L2–3	MRI	Acute	CES	L + fusion	Full recovery
Rahimizadeh 2013 [85]	53	M	L3–4	MRI	1 day	CES	L + E + discectomy	Full recovery
	57	M	L4–5	MRI	Acute on top of chronic	Radiculopathy	L + E + discectomy	Full recovery
Türkoglu 2013 [111]	55	M	L2–3	MRI	12 day/acute	CES	L + E	Subtotal
Tarukado 2014 [103]	26	M	L4–5	MYO-MRI	3 days	Radiculopathy	Microendoscopy	Full recovery
Ba 2014 [6]	52	M	L4–5	CT, MRI	7 days	CES	L + E	Subtotal
	47	M	L4–5	MRI	2 months	CES	L + E	Subtotal
	48	M	L4–5	MRI	Long time	CES	L + E	Full recovery
	42	M	L4–5	MRI	2 months	CES	L + E	Subtotal
Yoo 2015 [115]	32	M	L2–3	MRI	2 weeks	RP, LBP	L + E	Full recovery
Tarukado 2015 [104]	83	M	L2–3	MRI	1 month	Radiculopathy	Conservative	Full recovery 1 month
	62	M	L2–3	MRI	Acute on top	Radiculopathy	Conservative	Full recovery 4 months
	79	M	L4–5	MRI	Acute on top	Radiculopathy	Conservative	Full recovery 6 months
	53	M	L3–4	MRI	5 days	Radiculopathy	L + E	Full recovery 3 months
Abe 2015 [1]	65	M	L3–4	MRI	Acute	Radiculopathy	HL + E	Improved
	73	M	L4–5	MRI	2 years	Radiculopathy	HL + discectomy	Improved
Bouya 2015 [10]	52	M	L3–4	MRI	1 week	Radiculopathy	L + E	Full recovery
Haddadi 2016 [37]	77	M	L4–5	MRI	Acute	CES	L + fusion	Full recovery
Li 2016 [62]	48	M	L5–S1	CT/MRI	30 day	Radiculopathy	L + fusion	Full recovery
Toktas 2016 [107]	60	M	L2–3	MRI	2 months	RP, LBP	L + E	Full recovery
Diyora et al. 2016 [21]	44	M	L4–5	MRI	2	CES	2 level L	Satisfactory
Ajayi et al. 2016 [3]	65	F	L3–4	MRI	30	CES	L + E	Improved
Kutty et al. 2017 [58]	48	M	L3–4	MRI	5	CES	L + E	Subtotal
	49	M	L3–4	MRI	14	CES/RP	L + E	Subtotal
Frati 2017 [32]	46	F	L2–3	MRT	Acute on top	Radiculopathy	L + fusion	Improved subtotal
Turan 2017 [110]	41	M	L4–5	MRI	3	CES	HL + discectomy	Excellent
	51	M	L3–4	MRI	2	CES	HL + E	Excellent
	56	M	L3–4	MRI	2	CES	HL + E	Good
	48	F	L4–5	MRI	3	CES/RP	HL + E	Good
	32	F	L5–S1	MRI	5	CES	HL + discectomy	Good
	70	M	L3–4	CT	21	CES	HL + E	Fair
	62	M	L3–4	MRI	10	Radiculopathy	HL + E	Poor
	28	M	L4–5	MRI	4	CES	HL + E	Good
	58	M	L2–3	MRI	14	Radiculopathy	HL + E	Fair

Table 1 (continued)

Reference and years	Age	Sex	Level	Radiology	Duration	Clinical presentation	Surgery	Outcome
Takano 2017 [102]	78	M	L3–4	Disco/MR	Acute	CES/RP	3 level L + E	Full recovery
Kil 2017 [52]	57	M	L2–3	MRI	1 day	RP, LBP	L + E	Full recovery
Frioui 2018 [33]	29	M	L3–4	MRI	1 day	CES	L + D	Subtotal
Ozdemir 2017 [84]	46	F	L4–5	MRI	2 weeks	RP	PHL + E	Full recovery
Montalvo 2018 [73]	45	M	L4–5	MRI	28 days	CES	L + E	Full recovery
	33	M	L4–5	MRI	5 months	RP/LBP	L + fusion	Improved
Kim [56]	76	M	L2–3	MRI	5	CES	L + E	Full recovery
Hawkins et al. [39]	40	M	L4–5	MRI	7	CES	L + E	Full recovery
Elsharkawy 2018 [29]	78	M	L1–2	MRI	120	RP	Interlaminar + E	Full recovery

LBP low back pain, *RP* radicular pain, *CES* cauda equina syndrome, *L + E* laminectomy + extirpation, *HL* hemilaminectomy, *fusion* laminectomy + fusion, *PHL* partial hemilaminectomy

[113]. Kelsey et al. [50] reported that jobs involving lifting objects with a twisted back and straight knees were associated with a high risk for disc injury. Therefore, the natural history of dorsal disc herniation is still unknown.

Pathophysiology and mechanism of dorsal migration

Current knowledge of the mechanism behind the migration of the disc fragment is limited. Dorsal migration begins with a tear in the annulus. The most important contributing factors are formation of radial fissures; combinations of repetitive compression loading, bending, and axial torsion rotation; and compression [68, 82]. This tear leads to extrusion of the nuclear material from the disc [2]. The extruded fragment is always contained within the posterior longitudinal ligament on the posterior or posterolateral aspect of the disc and involves material from the nucleus pulposus [18, 47, 74]. Moreover, the annular fiber arrangement directs the disc herniation toward the exiting and traversing nerve roots [42, 108].

Dorsal migration is limited by certain anatomic barriers; the sagittal midline septum connects the posterior longitudinal ligament to the medial and lateral walls of the spinal canal and the nerve roots themselves [106]. This anatomic barrier may, however, not be enough to prevent dorsal migration if there is severe adhesion between the annulus fibrous and the dural sac; because of the location of the annulus tear near the pedicle and acute strong pressure, the disc material may be pushed to the dorsal side of the dural sac [75, 109]. The wide angle between the nerve root and the dural sac in the upper lumbar spine may also play a role in posterior migration [6]. Some authors have suggested that preexisting scoliosis in older patients may be a predisposing factor through ventral deviation development and facet joint rotations in one side [46]. Moreover, the general predisposing factors in disc herniation, such as hard work, body mass index, positive family history, lack of sports activities, and spinal manipulation, may play a role in posterior migration of disc fragments [26, 72, 89].

Why L3–L4 and males?

Consistent with published data, our review showed that L3–L4 was the level most frequently affected (approximately 40% of cases), and L4–L5 was second most frequently affected [29, 98, 110]. The reason is not exactly known; it may be a combination of degeneration in older patients, anatomy, and occupation. According to our review, the mean age of affected patients was about 54 years. Patients with herniated discs at the L3–L4 level or above were significantly older than those with herniation at L4–L5 or below [46]. Kanayama et al. [49] reported that the risk for degeneration in L3–L4 increases with age.

Table 2 Type of surgery and outcome

Surgical approach	Number and percentage
Laminectomy	62 (57.9%)
Multisegmental laminectomy	8 (7.5%)
Laminectomy with fusion	7 (6.5%)
Hemilaminectomy	21 (19.6%)
Laminotomy	4 (3.6%)
Interlaminar Fensterung	4 (3.7%)
Endoscopic	1 (0.9%)

Published data have shown that lumbar disc degeneration in younger people occurred more often at lower levels (L4–L5, L5–S1), and in older patients it occurred more often at higher levels (L1–L2, L2–L3, L3–L4) [70, 91]. Disc degeneration in older age may be associated with insufficient ligaments and other structures. Consistent with these data, we found significant differences in disc herniation levels according to patients' ages ($\eta = 0.405$, $p = .001$), in which patients with a higher level of disc degeneration had a higher mean age.

Anatomy of the L3–L4 level may play also a role: at this level, the spinal canal is larger and the intervertebral disc is more horizontal. Such configurations may, in association with the nerve root, be more conducive to epidural migration [62, 98]. Load with the aging process may be another factor; mathematical models showed that the load on L3 and L4 in sitting and standing positions with flexion is 2 and a half times that of the total body weight [45, 94]. Several studies demonstrated an association between heavy physical activity and disc degeneration [7, 112].

In general, spine compression is more severe in men. Studies have shown that the cross-sectional area of major trunk-loading muscles was smaller in women than in men; moreover, the directions of muscle force differ between men and women [11, 69, 79].

The range of motion also differs in relation to gender: males have more lumbar extension than females, and females have more lateral flexion or sideways movement of the spine [8]. Women also have significantly more lumbar lordosis than men [83]. This may indicate that the pushing forces in men are greater, which is conducive to migration of a disc fragment to posterior epidural spaces.

Table 3 Comparison of males and females with respect to the outcome

	Outcome		
	Full recovery *n* (%)	Subtotal recovery *n* (%)	Total
Male	57 (64.0%)	32 (36.0%)	89 (100%)
Female	16 (72.7%)	6 (27.3%)	22 (100%)
Total	73 (65.8%)	38 (34.2%)	111 (100%)

This table includes all patients (with and without surgery). An analysis including only patients with surgery ($n = 107$) leads to comparable results

Table 4 Comparison of groups "upper lumbar spine" and "lower lumbar spine" with respect to the outcome

	Outcome		
	Full recovery *n* (%)	Subtotal recovery *n* (%)	Total
Upper lumbar spine	43 (65.2%)	23 (34.8%)	66 (100%)
Lower lumbar spine	30 (66.7%)	15 (33.3%)	45 (100%)
Total	73 (65.8%)	38 (34.2%)	111 (100%)

This table includes all patients (with and without surgery). An analysis including only patients with surgery ($n = 107$) leads to comparable results. Upper lumbar spine: L1–2, L2–3, L3–4; lower lumbar spine: L4–5, L5–S1

Diagnostic studies

Early diagnosis of posterior epidural disc degeneration is essential for choosing the appropriate treatment protocol, preventing permanent neurological complications, and optimizing postoperative clinical outcomes [34, 71].

Contrast material–enhanced MRI is the best means of diagnosis [29, 37, 85]. Our review showed that MRI was performed in most cases, and the disc fragments appeared hypointense on T1-weighted images, hyperintense in 80% of T2-weighted images, and of varying intensity in the other 20% [51]. In short-T1 inversion recovery (STIR) MRI, the fragments appear hyperintense because of increased regional blood perfusion in those areas [13].

MRI is helpful in outlining areas of spinal cord compression and may show the route of migration in the form of a tract-like enhancement extending from the outer aspect of the disc to the posterior epidural space [13, 99]. Despite the fact that MRI is an essential tool for diagnosing migrating posterior disc fragments, the appearances of disc fragments are not specific and may be similar to those of other posterior epidural

Table 5 Association between disc level and age

	Age		
Level of disc	*N*	*M*	SD
L1–2	4	62.50	11.59
L2–3	21	59.76	11.55
L3–4	41	57.44	12.52
L4–5	37	48.08	14.48
L5–S1	8	44.88	9.09
Total	111	54.04	13.79

pathological lesions; therefore, MRI may be inconclusive, especially without disc degeneration [80] (Fig. 1).

The native CT scan was the main diagnostic tool in 11% of the cases, mainly because CT scanning is fast and overall available; MRI may be unavailable in emergency situations and was not available for some earlier cases. In detecting typical disc herniation, CT scanning had 81.3% sensitivity and 77.1% specificity [57]. CT scanning combined with MRI may be used to detect calcification, which may help with differential diagnosis.

Myelography was one of the diagnostic tools in our review, especially in earlier cases; blockage of contrast medium at the level of disc herniation or sequestration was the main finding. Myelography was estimated to have 75.7% sensitivity and 76.5% specificity [57]. Myelography mainly reveals only the mass effect and compression of the dural sac and does not provide any information about the nature of the lesion.

Discography has shown some advantage in detecting the disc fragment origin through contrast medium leakage from the disc into the posterior dural space. This appearance may confirm that the posterior mass is part of the disc [102]. However, discography is an invasive procedure and may accelerate disc degeneration, even though small-bore needles and low-pressure injection are used. It may also cause disc herniation [12].

Electromyography was also used to diagnose posterior disc migration; if preoperative diagnosis is difficult, electromyography can reveal neural damage and nerve compression [56]. Electromyography is mainly an adjunct diagnostic method and more accurate in detecting compression of the nerve roots [63].

Collection of information such as history, clinical examination findings, and laboratory data raises the chances for accurate diagnosis. Published data have shown that up to 80% of cases can be diagnosed appropriately through MRI, along with clinical history and laboratory data [27, 37, 85]. Therefore, intraoperative findings may be suspect, but the final diagnosis depends on histopathological examination [102].

Differential diagnosis

In most cases, posterior epidural disc herniation manifests as an emergency; it should be diagnosed quickly, and emergency surgery may be performed. In this situation, establishing the appropriate diagnosis is difficult [32].

The differential diagnosis includes conditions with features similar to posterior epidural mass, which include degenerative (synovial cyst, and facet joint osteophyte), infective (epidural abscess), neoplastic (meningioma, metastasis, lipoma, lymphoma, hemangioma), and miscellaneous pathologic processes (postoperative fibrosis) [25, 61].

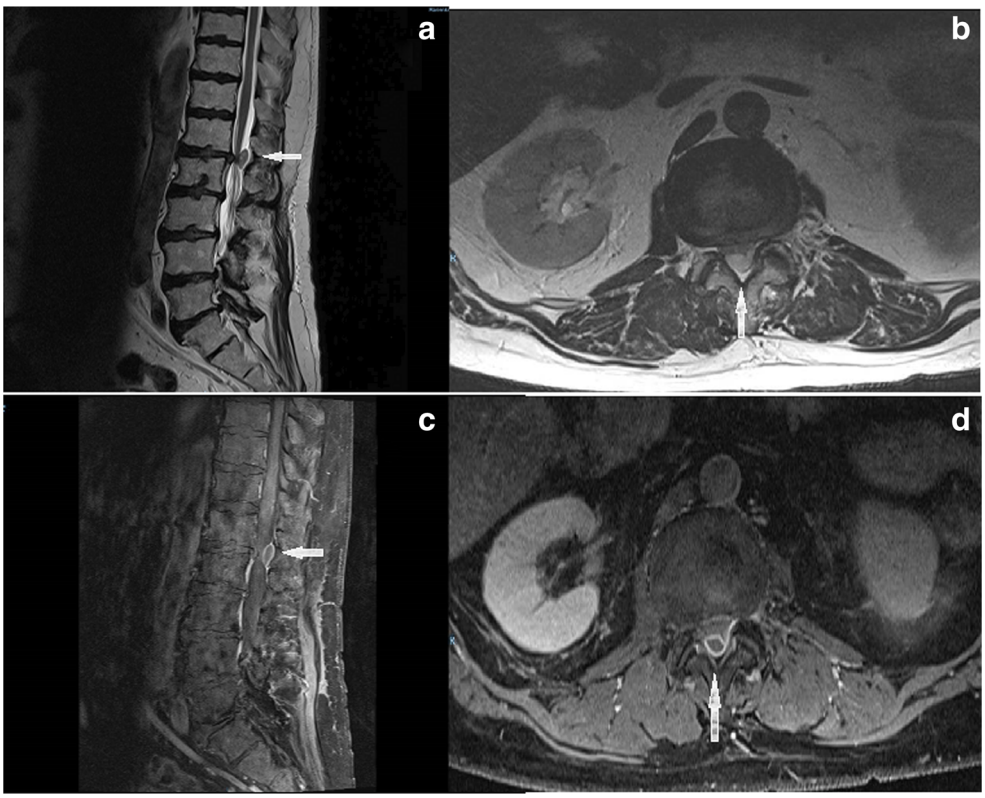

Fig. 1 MRI. (1a) Sagittal T2-weighted image. (1b) Axial T2-weighted image showing a posterior mass at level L1–2. (2a, 2b) Sagittal and axial T1-weighted image with contrast showing a ring enhancement (Elsharkawy 2018)

Synovial cysts may be observed on MRI, depending on the cyst content. T2-weighted MRI commonly reveals fibrosis or calcified hypointense capsules, and there may be adhesions to adjacent neural structures. Ligamentum flavum cysts are usually hemorrhagic and not connected to the facet joint [34]. Posterior lipomatosis is usually hyperintense on T1- and T2-weighted MRI [34].

Contrast material–enhanced MRI usually reveals solid and homogeneous enhancement in cases of meningiomas and hemangiomas. Abscesses and hematomas usually demonstrate peripheral rims of enhancement with an associated infectious illness or history of trauma. Spondylodiscitis may be associated with the epidural abscess; initial manifestations include fibrosis in 66% of cases, and 5.5% of affected patients have a history of epidural injection [86].

Hematoma appears as a biconvex lesion with a variable signal on T1-weighted images and has hypointense foci on a heterogeneous hyperintense background on T2-weighted images [34]. Bleeding or anticoagulation disorders, epidural anesthesia, trauma, and pregnancy are risk factors associated with epidural hematoma [76]. Hematoma shows different signals on MRI, depending on duration of bleeding [34].

In cases of malignant neoplasm, history and general examination usually reveal multisystem involvement. Based on associated infectious illness, history of trauma, clinical examination findings, and radiologic shape of the mass and form of contrast enhancement, the appropriate diagnosis can be reached in most cases [19, 32, 67].

Clinical manifestations

The clinical manifestations of posterior disc fragment include no typical features [73]. Therefore, the clinical presentation is variable, from lumbago without neurologic deficits to cauda equina syndrome [29, 51]. We found that approximately 50% of affected patients presented with cauda equina syndrome with neurologic deficits [32, 51].

In typical disc herniation cases, cauda equina syndrome is clinically rare; the incidence is 1.8 per million in the general population. In approximately 2 to 6% of cases, patients undergo lumbar disc surgery because of cauda equina syndrome [35]. However, the incidence of cauda equine syndrome is higher among posterior lumbar disc herniation cases [73]. The small size of the posterior epidural space is conducive to neural structure compression, which increases the chances of neurologic deficits [35, 51]. Cauda equina syndrome is caused by mechanical compression of the dural sac and neural structures, resulting in decreased blood flow and availability of nutrients, and in intraneural edema, which indirectly leads to ischemia and injury [87]. Bowel dysfunction and bladder paralysis are warning signs and usually indicate that emergency surgery is warranted [20].

We noticed in the review that there was no definition of cauda equina syndrome; several authors described the symptoms without clear definition. In general, nonspecific symptoms and signs of cauda equina syndrome may vary widely [77].

Our review showed that clinical presentation usually starts with acute LBP and sciatica over a period of hours to days and, in several cases, on top of chronic LBP [32, 104], followed by progressive neurologic and sphincter disturbances [16, 48, 54, 65, 88].

Treatment

Management of posterior epidural migration of lumbar disc fragments should start according to guidelines that apply in cases of ordinary disc herniation. However, approximately 50% of cases are emergencies, which make the decision-making and treatment planning challenging [32].

Conservative treatment

In our review, 4 cases were treated conservatively, and in one case, the patient's condition spontaneously improved [72, 104]. In principle, treatment should be conservative for all patients as long as they have no progressive neurological deficit or cauda equina syndrome. Pain killers, corticosteroids, and physical therapy are usually administered [38]. Patients undergoing conservative treatment need continuous follow-up [104].

Surgical treatment

In most cases, surgery was the treatment of choice. Surgical management was carried out in 96% of patients in our reviewed cases.

Several authors recommended early surgery as the first choice of therapy in patients with large sequestered disc fragments that had migrated posteriorly to prevent severe neurological deficits such as cauda equina and conus medullaris syndromes [51]. Our review showed, in agreement with published data, that surgical intervention resulted in a satisfactory outcome, which may be the reason for recommending surgery [15, 51, 105].

Surgical approach and strategies

The challenges encountered in the preoperative diagnostic studies continue during surgical planning. The surgical plan should guarantee maximal exposure of the pathologic process, avoid incidental durotomy, enable approach to the nerve root

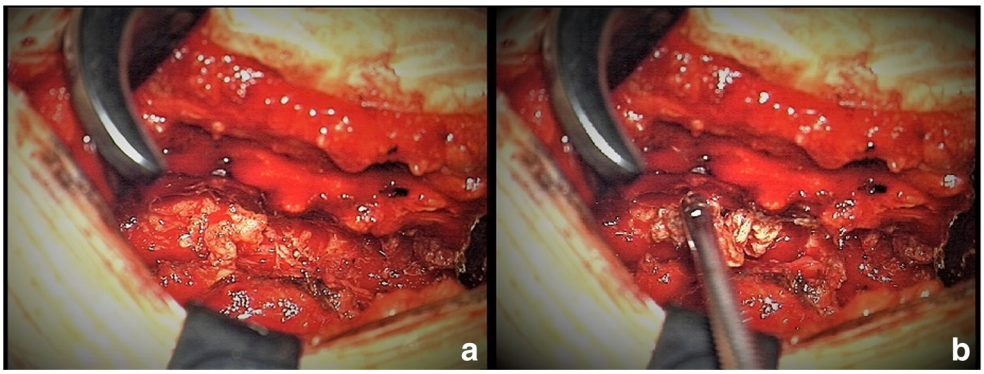

Fig. 2 (2a) Intraoperative picture showing a disc material extended to the ventromedial side of the disc after removal of the mass, (2b) showing a disc material after removal of the capsule membrane.

and disc spaces without traction on the neural structures, and minimize overlap syndrome and instability [32]. To reach this goal, individual patient features should be considered, and the surgical technique should be planned carefully to increase chances of better results [87].

Surgical strategies in our review varied from multilevel laminectomy with fusion to minimal invasive endoscopy [87]. Laminectomy and decompression were predominantly used in cases of posterior disc fragment (Table 1). Laminectomy ensures full exposure of the fragment and easier removal of the lesion, decreases the risk of incidental dural tear, minimizes the traction on the neural structures, and saves time, which is especially important in emergency situations [6, 32, 102].

Hemilaminectomy, mini-hemilaminectomy, and laminotomy were the second most common surgical techniques used to minimize instability and for bone removal [72, 80, 98, 110]. Laminectomy with various types of spine fusion, such as fusion with interspinous devices and dynamic fusion with rod and screws, was used in several cases to

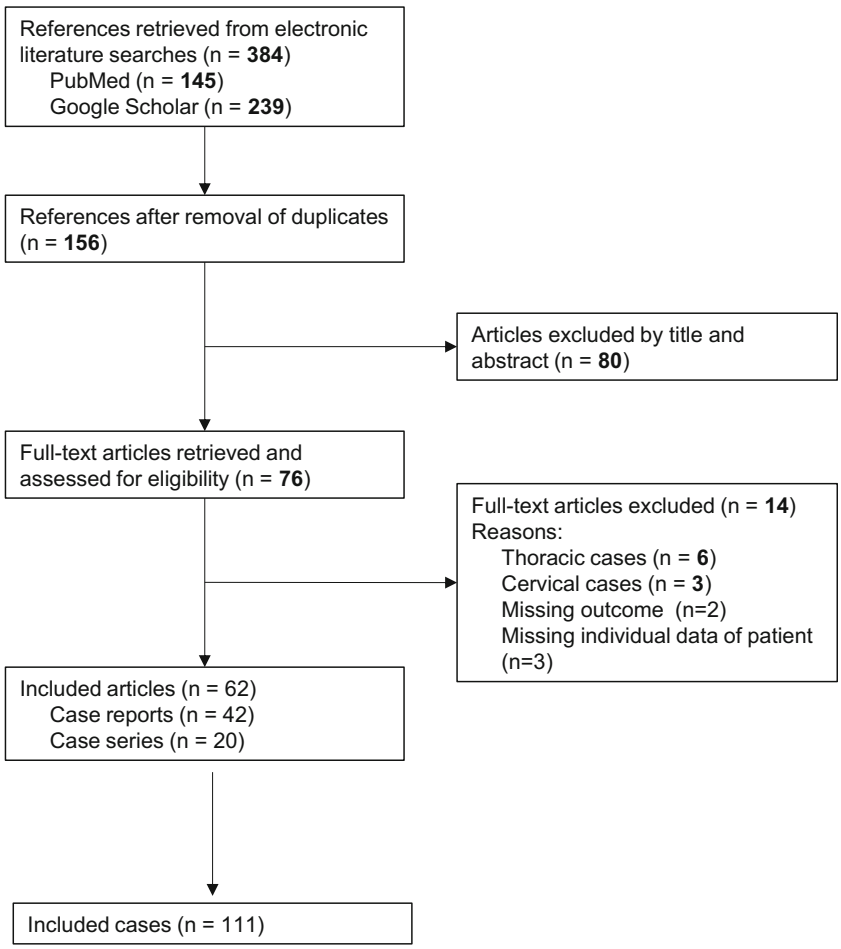

Fig. 3 Flowchart of the study

stabilize the spine, reduce load pressure over disc spaces and facet joints, and treat the possible overlap instability postoperatively [32, 37, 43, 48, 62, 74, 115].

Minimal invasive approaches, such as interlaminar and endoscopy, were reported in several selected cases, with success rates like those for laminectomy [22, 29, 44, 103].

Intraoperative findings

In our review, the ligamentum flavum was intact in all cases except one, in which ligamentum flavum perforation was noticed [84]. The ligamentum flavum showed degeneration changes and thickness [1].

Authors described the intraoperative disc fragment in different ways: as "free" [97], as "huge" [103], and as "big" [85]. Others gave exact measurements, such as "15 × 12 × 10 mm" or "20 × 13 × 10 mm" [105], or approximate measurements, such as "a 2.3cm sized mass" [1], "a 3 cm sized mass" [16], and "4 cm in length" [44]. Some authors described the disc fragment as an "elastic mass" [1], "hard" [55], "fat-like sequestrated" [44], and "semi-hard and capsulated mass" [29]. Surrounding and embedded tissues were described as "inflammatory" tissue [32], as richly vascularized fat tissue [16], as "encased in veins" [55], as granulation tissue [52], and as highly vascularized epidural fat [1]. Some disc fragments were found in posterolateral locations [39, 44, 53, 80, 84] and tracing their origins to the original disc was also described [29, 58, 66]. In other cases, authors described a thin film of disc material on the ventromedial side [29] and adherence of this material to the dural sac [32].

Rupture at the posterior longitudinal ligament [1] and annulus tear [52, 85] was detected. Other authors described the annulus as stretched more than usual [103]. Discectomy to remove degenerated disc material was done in about 30% of cases (Table 1). An empty disc was also reported [21], but dural tear due to disc fragment was not reported. The pathologic examination confirmed the presence of a disc fragment in all cases (Fig. 2).

Limitation of the results

General case report limitations included variations in diagnostic procedures, in assessment of outcome, in identification of level when the disc fragment was gigantic, in definition of cauda equina syndrome, and in the surgical approaches. Overinterpretation, misinterpretation, and a tendency to report only positive outcomes are common biases in case reports [81]. Figure 3 shows the flowchart of the study.

Conclusion

The diagnosis and treatment of posterior epidural disc fragments are challenging. It should be included in the differential diagnosis for patients with acute LBP and progressive neurologic impairments without infection or general illness. Careful history documentation, clinical examinations, and contrast material–enhanced MRI with laboratory tests could help reveal the presence of the disc fragment and rule out several other pathologic processes. Surgical treatment produces a favorable outcome in most cases.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval Approved from local ethics committee in hospital

Informed consent Not applicable (review)

References

1. Abe E, Uchikado H, Maruiwa H, Ichinomiya T, Fujiki M (2015) Two cases of dorsal epidural migration of lumbar disk fragments. Neurosurg Q 25(1):131–136
2. Adams MA, Hutton WC (1985) Gradual disc prolapse. Spine (Phila Pa 1976) 10(6):524–531
3. Ajayi O, Shoakazemi A, Tubbs RS, Moisi M, Rostad S, Newell DW (2016) Atypical presentation of a sequestered posterolateral disc fragment. Cureus 8(2):e502
4. Akhaddar A, El-asri A, Boucetta M (2011) Posterior epidural migration of a lumbar disc fragment: a series of 6 cases. J Neurosurg Spine 15(1):117–128
5. Ávila Ramírez J, Reyes Rodríguez VA, Ulibarri-Vidales M, Ávila Cervantes R (2008) Epidural dorsally sequestered lumbar disc herniation at L4-L5. Case report. Rev Mex Neurocienc 9:494–496
6. Ba MC, Kleib R, Sy C, Diabang J, Ndoye N, Thiam AB, Thioub M, Tine I, Badiane SB (2014) Lumbar disc hernia migrating to the epidural posterior space: a rare entity. Internet J Neurosurgery 10:1–6
7. Battié MC, Videman T (2006) Lumbar disc degeneration: epidemiology and genetics. J Bone Joint Surg Am 88(Suppl 2):3–9
8. Berryman Reese N, Bandy WD (2016) Joint range of motion and muscle length testing-E-book, 3th edition, st. Laouis, Missouri, ISBN: 0323291554
9. Bonaroti EA, Welch WC (1998) Posterior epidural migration of an extruded lumbar disc fragment causing cauda equina syndrome. Clinical and magnetic resonance imaging evaluation. Spine (Phila Pa 1976) 23(3):378–381
10. Bouya SM, Djoubairou BO, Okacha N, Gazzaz M, El Mostarchid B (2015) Posterior epidural migration of lumbar intervertebral fragment: case report. Pan Afr Med J 21:80
11. Brinckmann P, Hoefert H, Jongen HT (1981) Sex differences in the skeletal geometry of the human pelvis and hip joint. J Biomech 14(6):427–430
12. Carragee EJ, Don AS, Hurwitz EL, Cuellar JM, Carrino J, Herzog R, Herzog R (2009) 2009 ISSLS prize winner: does discography cause accelerated progression of degeneration changes in the lumbar disc. Spine (Phila Pa 1976) 34:2338–2345

13. Chen CY, Chuang YL, Yao MS, Chiu WT, Chen CL, Chan WP (2006) Posterior epidural migration of a sequestrated lumbar disk fragment: MR imaging findings. Am J Neuroradiol 27(7): 1592–1594
14. Chiu C-C, Chuang T-Y, Chang K-H, Wu C-H, Lin P-W, Hsu W-Y (2015) The probability of spontaneous regression of lumbar herniated disc: a systematic review. Clin Rehabil 29:184–195
15. Choi Y-S (2009) Pathophysiology of degenerative disc disease. Asian Spine J 3:39–44
16. Choi JY, Lee WS, Sung KH (2007) Intradural lumbar disc herniation–is it predictable preoperatively? A report of two cases. Spine J 7:111–117
17. Cribb GL, Jaffray DC, Cassar-Pullicino VN (2007) Observations on the natural history of massive lumbar disc herniation. J Bone Joint Surg (Br) 89–B:782–784
18. Daghighi MH, Pouriesa M, Maleki M, Fouladi DF, Pezeshki MZ, Mazaheri Khameneh R, Bazzazi AM (2014) Migration patterns of herniated disc fragments: a study on 1,020 patients with extruded lumbar disc herniation. Spine J 14(9):1970–1977
19. Derincek A, Özalay M, Şen O, Pourbagher A (2009) Posterior epidural mass : can a posteriorly migrated lumbar disc fragment mimic tumour, haematoma or abscess ? Acta Orthop Belg 75(3): 423–427
20. Dinning TA, Schaeffer HR (1993) Discogenic compression of the cauda equina: a surgical emergency. Aust N Z J Surg 63:927–934
21. Diyora BD, Giri S, Giri D, Nitin Kotecha SP (2016) An unusual case of dorsally sequestrated disk mimicking tumor with cauda equina syndrome. J Spinal Surg 1:15–17
22. Dösoğlu M, Is M, Gezen F, Ziyal MI (2001) Posterior epidural migration of a lumbar disc fragment causing cauda equina syndrome: case report and review of the relevant literature. Eur Spine J 10:348–351
23. Ekşi MS, Yener U, Akakin A, Akakin D, Konya D (2010) Posterior epidural disc herniation at L3-L4 mimicking a spinal tumor: a case report. J Neurosurg Sci 54:71–76
24. El Asri AC, Naama O, Akhaddar A, Gazzaz M, Belhachmi A, El Mostarchid B, Boucetta M (2008) Posterior epidural migration of lumbar disk fragments: report of two cases and review of the literature. Surg Neurol 70(6):668–671
25. El Khamary SM, Alorainy IA (2006) Case 100: spinal epidural meningioma 1. Radiology 241(2):614–617
26. Elfering A, Semmer N, Birkhofer D, Zanetti M, Hodler J, Boos N (2002) Risk factors for lumbar disc degeneration: a 5-year prospective MRI study in asymptomatic individuals. Spine (Phila Pa 1976) 27:125–134
27. Elgamri A, Sami A, Aqqad A, Hilmani S, Ibahioin K, Naja A, El Kamar A, El Azhari A (2009) Posterior migration of a lumbar disc herniation as a cause of cauda equina syndrome. J Radiol 90(6): 731–733
28. Elsharkawy AE, Avramidis P, Baume B, Gafumbegete E, Lange B, Klassen PD (2017) An intradural lumbar disc fragment with free migration: a case of a missed intradural disc herniation. Interdiscip Neurosurg Adv Tech Case Manag 7:17–21
29. Elsharkawy AE, Gafumbegete E, Klassen PD (2018) Posterior epidural migration of extruded lumbar disc fragment mimicking epidural mass: a case report. Interdiscip Neurosurg Adv Tech Case Manag 11:31-33
30. Eysel P, Herbsthofer B (2001) Dorsal compression of the epidural cord due to free sequestral lumbar prolapse. Arch Orthop Trauma Surg 121:238–240
31. Fardon DF, Milette PC, Combined Task Forces of the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology (2001) Nomenclature and classification of lumbar disc pathology. Recommendations of the Combined task Forces of the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology. Spine (Phila Pa 1976) 26:E93–E113
32. Frati A, Pesce A, Palmieri M, Vangelista T, Caruso R, Salvati M, Raco A (2017) Anterior-to-posterior migration of a lumbar disc sequestration: surgical remarks and technical notes about a tailored microsurgical discectomy. Case Rep Surg 2017:1762047
33. Frioui S, Khachnaoui F (2018) Posterior epidural migration of a lumbar disk: an entity not to ignore. Pan Afr Med J 29:59
34. Gala F, Aswani Y (2016) Imaging in spinal posterior epidural space lesions: a pictorial essay. Indian J Radiol Imaging 26(3): 299–315
35. Gardner A, Gardner E, Morley T (2011) Cauda equina syndrome: a review of the current clinical and medico-legal position. Eur Spine J 20:690–697
36. Gonçalves FG, Hanagandi PB, Torres CI, DelCarpio-O'Donovan R (2012) Posterior migration of lumbar disc herniation - imaging dilemma due to contrast contraindication: a case report. Radiol Bras 45(3):170–172
37. Haddadi K, Qazvini HRG (2016) Posterior epidural migration of a sequestrated lumbar disk fragment causing cauda equina syndrome in an old patient: a case report. Clin Med Insights Case Reports 9:39–41
38. Hagen KB, Hilde G, Jamtvedt G, Winnem MF (2002) The Cochrane review of advice to stay active as a single treatment for low back pain and sciatica. Spine (Phila Pa 1976) 27:1736–1741
39. Hawkins JC, Natkha VP, Seibly J (2018) Posterior epidural migration of a lumbar disc herniation causing cauda equina syndrome: a case report. Cureus 10:e2739
40. Hirabayashi S, Kumano K, Tsuiki T, Eguchi M, Ikeda S (1990) A dorsally displaced free fragment of lumbar disc herniation and its interesting histologic findings. A case report. Spine (Phila Pa 1976) 15:1231–1233
41. Hodges SD, Humphreys SC, Eck JC, Covington LA (1999) Posterior extradural lumbar disk fragment. J South Orthop Assoc 8:222–228
42. Hsu EW, Setton LA (1999) Diffusion tensor microscopy of the intervertebral disc anulus fibrosus. Magn Reson Med 41(5):992–999
43. Huang T-Y, Lee K-S, Tsai T-H, Su Y-F, Hwang S-L (2011) Posterior epidural migration of sequestrated lumbar disc fragments into the bilateral facet joints: case report. Neurosurgery 69(5):E1148–E1151
44. Hur JW, Lee JW, Rhee JJLH (2011) Posterior epidural migration of lumbar disc fragment: three cases and review of literature. Korean J Spine 8:66–69
45. Inoue N, Espinoza Orías AA (2011) Biomechanics of intervertebral disk degeneration. Orthop Clin North Am 42(4):487–499
46. Iwasaki M, Akino M, Hida K, Yano S, Aoyama T, Saito H, Iwasaki Y (2011) Clinical and radiographic characteristics of upper lumbar disc herniation: ten-year microsurgical experience. Neurol Med Chir (Tokyo) 51(6):423–426
47. Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS (1994) Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med 331:69–73
48. Jové Talavera R, Altemir Martínez V, Chárlez Marco A, Mas Atance J, Curiá Jové E, Aguas Valiente J (2012) Epidural posterior migration of a disc fragment. Rev Esp Cir Ortop Traumatol 56(3): 224–226
49. Kanayama M, Togawa D, Takahashi C, Terai T, Hashimoto T (2009) Cross-sectional magnetic resonance imaging study of lumbar disc degeneration in 200 healthy individuals. J Neurosurg Spine 11(4):501–507
50. Kelsey JL, Githens PB, White AA, Holford TR, Walter SD, O'Connor T, Ostfeld AM, Weil U, Southwick WO, Calogero JA

(1984) An epidemiologic study of lifting and twisting on the job and risk for acute prolapsed lumbar intervertebral disc. J Orthop Res 2(1):61–66
51. Khan Z, Sharafat S, Ali M, Haider A, Khanzada K, Siddique M (2013) Posterior epidural migration of herniated disc posterior epidural migration of herniated lumbar disc fragment: experience with 11 cases. Gomal J Med Sci 11:13–15
52. Kil J-S, Park J-T (2017) Posterior epidural herniation of a lumbar disk fragment at L2-3 that mimicked an epidural hematoma. Korean J Spine 14:115–117
53. Kim MS, Hur JW, Lee JW, Lee HK (2003) Posterior and lateral epidural migration of extruded lumbar disc fragments: case report. J Korean Neurosurg Soc 33:297–298
54. Kim JH, Kong MH, Lee SK, Song KY (2004) A case of posterior epidural migration of an extruded lumbar disc fragment causing cauda equina syndrome. J Korean Neurosurg Soc 35:442–444
55. Kim JS, Lee SH, Arbatti NJ (2010) Dorsal extradural lumbar disc herniation causing cauda equina syndrome : a case report and review of literature. J Korean Neurosurg Soc 47:217–220
56. Kim H, Kwon BS, Park J-W, Lee HJ, Lee JW, Lee EK, Park TJ, Kim HJ, Cho Y, Kim T, Nam K (2018) Posterior epidural migration of a lumbar intervertebral disc fragment resembling a spinal tumor: a case report. Ann Rehabil Med 42:621–625
57. Kim JH, van Rijn RM, van Tulder MW, Koes BW, de Boer MR, Ginai AZ, Ostelo RWGJ, van der Windt DAMW, Verhagen AP (2018) Diagnostic accuracy of diagnostic imaging for lumbar disc herniation in adults with low back pain or sciatica is unknown; a systematic review. Chiropr Man Therap 26:37
58. Kutty RK, Sreemathyamma BS, Peethambaran A, Jain SK, Kumar S (2017) PEMLIF—a tale of two stories. J Spine Surg 3: 498–503
59. Kuzeyli K, Çakr E, Usul H, Baykal S, Yazar U, Karaarslan G, Arslan E, Peksoylu B (2003) Posterior epidural migration of lumbar disc fragments. Spine (Phila Pa 1976) 28(3):E64–E67
60. Lakshmanan P, Ahuja S, Lyons K, Howes J, Davies PR (2006) Sequestrated lumbar intervertebral disc in the posterior epidural space: a report on two cases and review of the literature. Spine J 6(5):583–586
61. Lee JW, Cho EY, Hong SH, Chung HW, Kim JH, Chang K-H, Choi J-Y, Yeom J-S, Kang HS (2007) Spinal epidural hemangiomas: various types of MR imaging features with histopathologic correlation. AJNR Am J Neuroradiol 28(7):1242–1248
62. Li K, Li Z, Geng W, Wang C, Ma J (2016) Postdural disc herniation at L5/S1 level mimicking an extradural spinal tumor. Eur Spine J 25:80–83
63. Li W, Liu Y, Zheng C, Miao J, Chen H, Quan H, Yan S, Zhang K (2018) Diagnosis of compressed nerve root in lumbar disc herniation patients by surface electromyography. Orthop Surg 10: 47–55
64. Lichtor T (1989) Posterior epidural migration of extruded lumbar disk. Surg Neurol 32:311–312
65. Lisai P, Doria C, Crissantu L, Dore T, Spano G, Fabbriciani C (2000) Posterior epidural migration of an extruded free fragment from a lumbar disc herniation. J Orthop Traumatol 1:103–105
66. Lombardi V (1973) Lumbar spinal block by posterior rotation of anulus fibrosus. Case report. J Neurosurg 39:642–647
67. Lutz JD, Smith RR, Jones HM (1990) CT myelography of a fragment of a lumbar disk sequestered posterior to the thecal sac. AJNR Am J Neuroradiol 11:610–611
68. Marras WS, Lavender SA, Leurgans SE, Rajulu SL, Gary Allread W, Fathallah FA, erguson SA (1993) The role of dynamic three-dimensional trunk motion in occupationally-related low back disorders: the effects of workplace factors, trunk position, and trunk motion characteristics on risk of injury. Spine (Phila Pa 1976) 18(5):617–628
69. Marras WS, Jorgensen MJ, Granata KP, Wiand B (2001) Female and male trunk geometry: size and prediction of the spine loading trunk muscles derived from MRI. Clin Biomech 16(1):38–46
70. Martin MD, Boxell CM, Malone DG (2002) Pathophysiology of lumbar disc degeneration: a review of the literature. Neurosurg Focus 13(2):E1
71. Matsumoto T, Toyoda H, Terai H, Dohzono S, Hori Y, Nakamura H (2016) Utility of discography as a preoperative diagnostic tool for intradural lumbar disc herniation. Asian Spine J 10:771. https://doi.org/10.4184/asj.2016.10.4.771
72. Mobbs RJ, Steel TR (2007) Migration of lumbar disc herniation: an unusual case. J Clin Neurosci 14(6):581–584
73. Montalvo Afonso A, Mateo Sierra O, Gil de Sagredo Del Corral OL, Vargas López AJ, González-Quarante LH, Sola Vendrell E, Romero Martínez J (2018) Misdiagnosis of posterior sequestered lumbar disc herniation: report of three cases and review of the literature. Spinal Cord Ser Cases 4:61
74. Moore RJ, Vernon-Roberts B, Fraser RD, Osti OL, Schembri M (1996) The origin and fate of herniated lumbar intervertebral disc tissue. Spine (Phila Pa 1976) 21:2149–2155
75. Morizane A, Hanakita J, Suwa H, Ohshita N, Gotoh K, Matsuoka T (1999) Dorsally sequestrated thoracic disc herniation - case report. Neurol Med Chir (Tokyo) 39(11):769–772
76. Naidich TP (2011) Imaging of the spine. Saunders/Elsevier
77. Nater A, Fehlings MG (2015) The timing of decompressive spinal surgery in cauda equina syndrome. World Neurosurg 83:19–22
78. Neugroschl C, Kehrli P, Gigaud M, Ragragui O, Maitrot D, Manelfe C, Dietemann JL (1999) Posterior extradural migration of extruded thoracic and lumbar disc fragments: role of MRI. Neuroradiology 41(9):630–635
79. Ng JK, Kippers V, Richardson CA (1998) Muscle fibre orientation of abdominal muscles and suggested surface EMG electrode positions. Electromyogr Clin Neurophysiol 38(1):51–58
80. Nievas MNC, Hoellerhage H-G (2009) Unusual sequestered disc fragments simulating spinal tumors and other space-occupying lesions. J Neurosurg Spine 11(1):42–48
81. Nissen T, Wynn R (2014) The clinical case report: a review of its merits and limitations. BMC Res Notes 7:264
82. Norman R, Wells R, Neumann P, Frank J, Shannon H, Kerr M, The Ontario Universities Back Pain Study (OUBPS) Group (1998) A comparison of peak vs cumulative physical work exposure risk factors for the reporting of low back pain in the automotive industry. Clin Biomech 13(8):561–573
83. Norton BJ, Sahrmann SA, Van Dillen LR (2004) Differences in measurements of lumbar curvature related to gender and low Back pain. J Orthop Sport Phys Ther 34(9):524–534
84. Ozdemir B, Kanat A, Batcik OE, Erturk C, Celiker FB, Guvercin AR, Yazar U (2017) First report of perforation of ligamentum flavum by sequestrated lumbar intervertebral disc. J Craniovertebral Junction Spine 8:70–73
85. Rahimizadeh A, Rahimizadeh A, Soufiani H (2013) Posterior epidural migration of sequestered lumbar disc fragment causing cauda equina syndrome. Coluna/ Columna 12(1):78–80
86. Reihsaus E, Waldbaur H, Seeling W (2000) Spinal epidural abscess: a meta-analysis of 915 patients. Neurosurg Rev 23:175–204 discussion 205
87. Rhee JM, Schaufele M, Abdu WA (2006) Radiculopathy and the herniated lumbar disc. Controversies regarding pathophysiology and management. J Bone Joint Surg Am 88:2070–2080
88. Robe P, Martin D, Lenelle J, Stevenaert A (1999) Posterior epidural migration of sequestered lumbar disc fragments. Report of two cases. J Neurosurg 90:264–266
89. Saftić R, Grgić M, Ebling B, Splavski B (2006) Case-control study of risk factors for lumbar intervertebral disc herniation in Croatian island populations. Croat Med J 47:593–600

90. Sakas DE, Farrell MA, Young S, Toland J (1995) Posterior thecal lumbar disc herniation mimicking synovial cyst. Neuroradiology 37:192–194
91. Saleem S, Aslam HM, Rehmani MAK, Raees A, Alvi AA, Ashraf J (2013) Lumbar disc degenerative disease: disc degeneration symptoms and magnetic resonance image findings. Asian Spine J 7:322–334
92. Saruhashi Y, Omura K, Miyamoto K, Katsuura A, Hukuda S (1999) A migrated lumbar disc herniation simulating a dumbbell tumor. J Spinal Disord 12:307–309
93. Schellinger D, Manz HJ, Vidic B, Patronas NJ, Deveikis JP, Muraki AS, Abdullah DC (1990) Disk fragment migration. Radiology 175:831–836
94. Schultz AB, Andersson GBJ, Haderspeck K, Örtengren R, Nordin M, Björk R (1982) Analysis and measurement of lumbar trunk loads in tasks involving bends and twists. J Biomech 15(9):669–675
95. Sekerci Z, Ildan F, Yüksel M, Gül B, Kiliç C (1992) Cauda equina compression due to posterior epidural migration of extruded lumbar disk. Neurosurg Rev 15:311–313
96. Sen O, Volkan Aydin M, Erdogan B, Yildirim T, Can er H (2001) Cauda equina syndrome caused by posterior epidural migration of an extruded lumbar disc fragment. Turk Neurosurg 11:108–110
97. Şenel A, Çokluk C, Çelik F (2003) Posterior epidural migration of extruded lumbar disc mimicking epidural mass: case report. Turk Neurosurg 13:3–4
98. Sengoz A, Kotil K, Tasdemiroglu E (2011) Posterior epidural migration of herniated lumbar disc fragment. J Neurosurg Spine 14(3):313–317
99. Sze G, Krol G, Zimmerman RD, Deck MD (1988) Malignant extradural spinal tumors: MR imaging with Gd-DTPA. Radiology 167(1):217–223
100. Taher F, Essig D, Lebl DR, Hughes AP, Sama AA, Cammisa FP, Girardi FP (2012) Lumbar degenerative disc disease: current and future concepts of diagnosis and management. Adv Orthop 2012: 970752
101. Takada E, Takahashi M, Shimada K (2001) Natural history of lumbar disc hernia with radicular leg pain: spontaneous MRI changes of the herniated mass and correlation with clinical outcome. J Orthop Surg (Hong Kong) 9(1):1–7
102. Takano M, Hikata T, Nishimura S, Kamata M (2017) Discography aids definitive diagnosis of posterior epidural migration of lumbar disc fragments: case report and literature review. BMC Musculoskelet Disord 18(1):151
103. Tarukado K, Tono O, Doi T (2014) Ordinary disc herniation changing into posterior epidural migration of lumbar disc fragments confirmed by magnetic resonance imaging: a case report of a successful endoscopic treatment. Asian Spine J 8(1):69–73
104. Tarukado K, Ikuta K, Fukutoku Y, Tono O, Doi T (2015) Spontaneous regression of posterior epidural migrated lumbar disc fragments: case series. Spine J 15(6):57–62
105. Tatli M, Güzel A, Ceviz A, Karadağ O (2005) Posterior epidural migration of sequestered lumbar disc fragment causing cauda equina syndrome. Br J Neurosurg 19:257–259
106. Teufack SG, Singh H, Harrop J, Ratliff J (2010) Dorsal epidural intervertebral disk herniation with atypical radiographic findings: case report and literature review. J Spinal Cord Med 33:268–271
107. Toktaş ZO, Yilmaz B, Konya D, Yapicier O, Demir MK (2016) Posterior epidural migration of lumbar disc fragment as an unusual ring-enhancing mass. Spine J:e31–e32
108. Tsuji H, Hirano N, Ohshima H, Ishihara H, Terahata N, Motoe T (1993) Structural variation of the anterior and posterior anulus fibrosus in the development of human lumbar intervertebral disc. A risk factor for intervertebral disc rupture. Spine (Phila Pa 1976) 18:204–210
109. Turan Y, Yilmaz T, Gocmez C, Ozevren H, Kemaloglu S, Teke M, Sariyildiz MA, Ceviz A, Temiz C (2015) Posterior epidural migration of a sequestered lumbar intervertebral disc fragment. Turk Neurosurg 27(1):85–94
110. Turan Y, Yilmaz T, Gocmez C, Ozevren H, Kemaloglu S, Teke M, Sariyildiz MA, Ceviz A, Temiz C (2017) Posterior epidural migration of a sequestered lumbar intervertebral disc fragment. Turk Neurosurg 27(1):85–94
111. Türkoglu E, Karavelioglu E, Oral N, Metin Sanli Ahmet SZ (2013) Cauda equina syndrome due to posterior squestered lumbar disc herniation: a rare case report and MRI findings. J Turkish Spinal Surg 24:165–168
112. Vernon-Roberts B, Moore RJ, Fraser RD (2007) The natural history of age-related disc degeneration: the pathology and sequelae of tears. Spine (Phila Pa 1976) 32(25):2797–2804
113. Videman T, Battié MC (1999) The influence of occupation on lumbar degeneration. Spine (Phila Pa 1976) 24(11):1164–1168
114. Walsh AJ, Martin Z, McCormack D (2004) Cauda equina syndrome seconday to posterior epidural migration of a lumbar disc fragament: a rare phenomenon. Eur J Orthop Surg Traumatol 14: 30–31
115. Yoo YS, Il JC, Kim SW, Kim DM (2015) Posterior epidural migration of an extruded lumbar disc mimicking a facet cyst: a case report. Korean J Spine 12(1):12–14