



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

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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 sequestered 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.

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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
Lichter 1989 [64]	54	M	L4-5	Myelogram	2 months	RP	L + E	Full recovery
Lutz et al. 1990 [67]	61	M	L2-3	CT + myelo/MRI	1 month	RP/LBP	L + E	Full recovery
Hirabayashi et al. 1990 [40]	30	M	L4-5	CT + myelo	5 days	CES	L + E	Full recovery
Sekerci 1992 [95]	58	M	L3-4	MRI, CT + myelo	4.5 months	CES	L + E	Improvement
Sakas 1995 [90]	70	M	L4-5	Myelogram	2 months	RP, LBP	L + E	Full recovery
Bonaroti and Welch 1998 [9]	51	M	L2-3	CT	10 weeks	CES	L + E	Improvement
Hodges et al. 1999 [41]	56	M	L4-5	MRI	12 h	LBP	2 level L + E + discectomy	Full recovery
Robe 1999 [88]	68	M	L3-4	MRI, MYL, CT	7 days	RP	L + E + discectomy	Full recovery
Neugroschl 1999 [78]	41	F	L3-4	CT + myelo + MRI	Acute	CES	L + E + discectomy	Full recovery
Saruhashi et al. 1999 [92]	57	M	L2-3	CT + myelo + MRI	15 days	RP, LBP	2 level L + facetectomy discectomy	Full recovery
Lisai et al. 2000 [65]	64	M	L2-3	CT + myelo + MRI	14 days	RP	Bilateral L + E	Full recovery
Dosoglu et al. 2001 [22]	44	F	L5-S1	CT + myelo + MRI	1 month	RP	L + E	Full recovery
Sen et al. 2001 [96]	63	M	L3-4	MRI	12 h	CES	L + facetectomy discectomy	Full recovery
Eysel 2001 [30]	47	M	L3-4	MRI	15 days	CES	Fenestration discectomy	Full recovery
Senel 2003 [97]	36	M	L4-5	MRI + CT	10 h	CES	L + discectomy bilateral	Full recovery
Kuzeyli 2003 [59]	45	M	L3-4	MRI + CT	8 weeks	CES	L + E	Full recovery
Kim 2003 [53]	37	F	L4-5	CT	7 weeks	RP	L + E	Full recovery
Kim 2004 [54]	41	M	L3-4	CT	11 weeks	LBP	L + E	Full recovery
Walsh 2004 [114]	44	M	L3-4	MRI	5 days	LBP	PHL + E	Full recovery
Tatti 2005 [105]	47	F	L2-3	MRI	4 months	CES	HL + E	Full recovery
Lakshmanan 2006 [60]	62	F	L1-2	MRI + CT	25 days	CES	HL + E	Full recovery
Chen et al. 2006 [13]	49	M	L4-5	MRI	15 days	LBP	L + E	Full recovery
Choi 2007 [16]	60	F	L3-4	MRI	4 days	RP/CES	2 level L + E	Subtotal recovery
Mobbs 2007 [72]	44	M	L4-5	CT	6 h	CES	L + E	Subtotal recovery
EL Asri 2008 [24]	62	M	L3-4	MRI	24 h	CES	L + E	Full recovery
Jose et al. 2008 [5]	54	M	L5-S1	MRI	2 days	CES	L + E	Improved
Derincek 2009 [19]	53	M	L3-4	MRI	2 days	CES	L + E	Full recovery
Nievas 2009 [80]	58	M	L4-5	MRI	1 month	RP	L + E	Full recovery
Kim 2010 [55]	28	F	L4-5	MRI	3 months	RP	L + E	Full recovery
Teufäc 2010 [106]	75	M	L2-3	MRI	2 weeks	RP	L + E	Improved
Eksi 2010 [23]	68	M	L4-5	MRI	3 weeks	RP	HL + E	Full recovery
Huang et al. 2011 [43]	74	F	L3-4	MRI	3 days	RP	PHL + E	Full recovery
Akhaddr 2011 [4]	42	M	L5-S1	MRI	1 week	RP	HL + partial facetectomy	Full recovery
	36	M	L4-5	MRI	3 months	RP	HL	Full recovery
	45	M	L4-5	MRI	1 month	CES	2 HL + discectomy	Full recovery
	60	F	L1-2	MRI	1 month	Radiculopathy	Bilateral laminotomy	Improved
	83	M	L3-4	MRI	2 months	RP/LBP	L + E	Full recovery
	45	M	L4-5	MRI	4 weeks	RP, LBP	L + E	Full recovery
	67	M	L4-5	MRI	3 months	RP, LBP	L + E	Full recovery
	59	F	L2-4	MRI	2 months	RP, LBP	L + E	Full recovery
	73	M	L4-5	CT/MRI	4 weeks	CES	L + E	Full recovery
	49	M	L4-5	CT/MRI	Acute	RP, LBP	L + E	Full recovery
	50	M	L3-4	MRI	20 day	RP	L + E	Full recovery
	78	F	L3-4	MRI	10 years	Radiculopathy	L + fusion	Full recovery
	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
Sengoz 2011 [98]	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
	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
	51	M	L2-3	MRI	3 days	RP/LBP	HL	Improvement
	76	M	L3-4	MRI	3 days	CES	L + fusion	Improvement
	74	M	L2-3	MRI	4 day	CES	L + fusion	Full recovery
	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
	55	M	L2-3	MRI	12 day/acute	CES	L + E	Full recovery
	26	M	L4-5	MYO-MRI	3 days	Radiculopathy	Microendoscopy	Subtotal
Türkoglu 2013 [111]	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
	32	M	L2-3	MRI	2 weeks	RP, LBP	L + E	Full recovery
	83	M	L2-3	MRI	1 month	Radiculopathy	Conservative	Full recovery
	62	M	L2-3	MRI	Acute on top	Radiculopathy	Conservative	1 month
	79	M	L4-5	MRI	Acute on top	Radiculopathy	Conservative	Full recovery
	53	M	L3-4	MRI	5 days	Radiculopathy	L + E	4 months
	65	M	L3-4	MRI	Acute	Radiculopathy	HL + E	Full recovery
Abe 2015 [1]	73	M	L4-5	MRI	2 years	Radiculopathy	HL + discectomy	Improved
	52	M	L3-4	MRI	1 week	Radiculopathy	L + E	Improved
	77	M	L4-5	MRI	Acute	CES	L + fusion	Full recovery
	48	M	L5-S1	CT/MRI	30 day	Radiculopathy	L + fusion	Full recovery
	60	M	L2-3	MRI	2 months	RP, LBP	L + E	Full recovery
	44	M	L4-5	MRI	2	CES	L + E	Full recovery
	65	F	L3-4	MRI	30	CES	L + E	Satisfactory
	48	M	L3-4	MRI	5	CES	L + E	Improved
	49	M	L3-4	MRI	14	CES/RP	L + E	Subtotal
	46	F	L2-3	MRT	Acute on top	Radiculopathy	L + fusion	Subtotal
Frati 2017 [32]	41	M	L4-5	MRI	3	CES	HL + discectomy	Improved subtotal
	51	M	L3-4	MRI	2	CES	HL + E	Excellent
	56	M	L3-4	MRI	2	CES	HL + E	Excellent
	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
	Turan 2017 [110]	62	M	L2-3	MRI	Acute on top	Radiculopathy	Conservative
79		M	L4-5	MRI	Acute on top	Radiculopathy	Conservative	Full recovery
53		M	L3-4	MRI	5 days	Radiculopathy	L + E	6 months
65		M	L3-4	MRI	Acute	Radiculopathy	HL + E	Full recovery
73		M	L4-5	MRI	2 years	Radiculopathy	HL + discectomy	3 months
52		M	L3-4	MRI	1 week	Radiculopathy	L + E	Improved
77		M	L4-5	MRI	Acute	CES	L + fusion	Full recovery
48		M	L5-S1	CT/MRI	30 day	Radiculopathy	L + fusion	Full recovery
60		M	L2-3	MRI	2 months	RP, LBP	L + E	Full recovery
44		M	L4-5	MRI	2	CES	L + E	Satisfactory

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
Kim [56]	33	M	L4–5	MRI	5 months	RP/LBP	L + fusion	Improved
Hawkins et al. [39]	76	M	L2–3	MRI	5	CES	L + E	Full recovery
Elshtarkawy 2018 [29]	40	M	L4–5	MRI	7	CES	L + E	Full recovery
	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

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

	Outcome		Total
	Full recovery <i>n</i> (%)	Subtotal recovery <i>n</i> (%)	
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 <i>n</i> (%)	Subtotal recovery <i>n</i> (%)	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

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.

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

Level of disc	Age		
	<i>N</i>	<i>M</i>	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

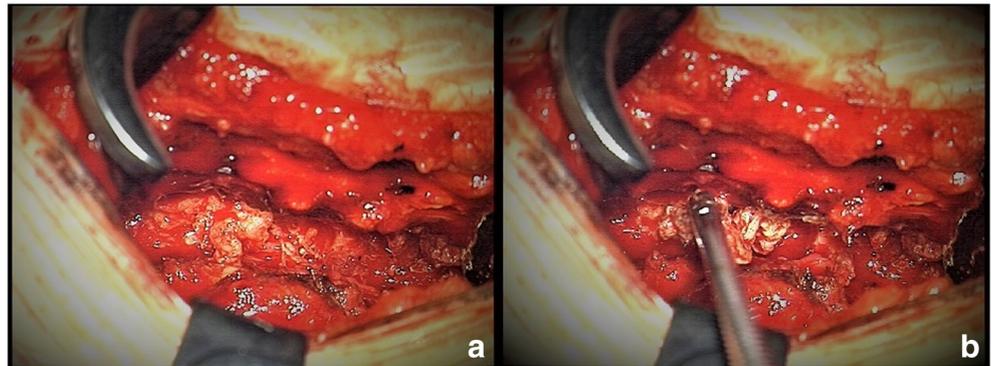
In most cases, surgery was the treatment of choice. Surgical management was carried out in 96% of patients in our reviewed cases.

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.



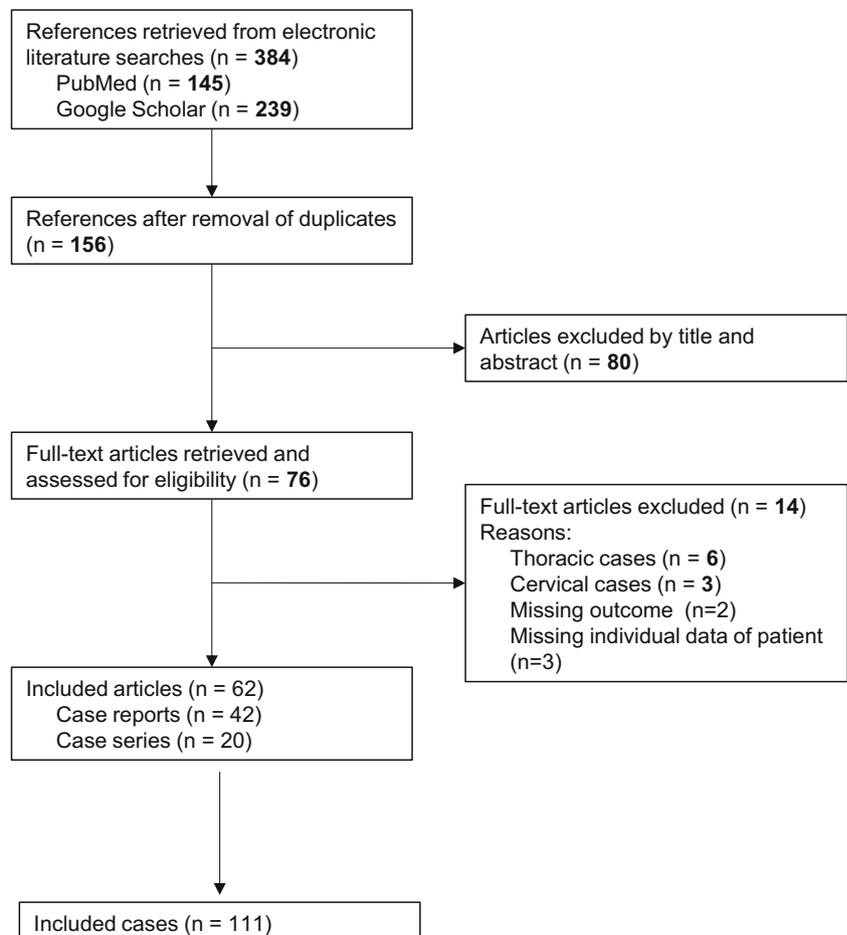
and disc spaces without traction on the neural structures, and minimize overlap syndrome and instability [32]. To reach this goal, individual patient features should be considered, and the surgical technique should be planned carefully to increase chances of better results [87].

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 2.3 cm 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 sequestered” [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)

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