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Spinal epidural lipomatosis in children with hematologic malignancies

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Summary

Background—Abnormal fat deposition in the epidural space or spinal epidural lipomatosis (SEL) due to corticosteroid treatment or obesity may cause obstruction to cerebrospinal fluid flow. Little is known about SEL in patients with hematologic malignancies who require frequent lumbar punctures and corticosteroid treatment that places them at risk.

Patients and methods—Records and radiologic images of patients with SEL and leukemia or non Hodgkin lymphoma (NHL) treated at a single institution from 1999–2009 were reviewed. Risk factors were compared with 405 control patients with leukemia.

Results—Fourteen patients with leukemia or NHL were diagnosed with SEL. The majority of patients underwent diagnostic imaging after unsuccessful lumbar punctures within one month of their primary diagnosis. Prior to SEL diagnosis, all patients received systemic and/or intrathecal corticosteroids. SEL diagnosis led to modification of intrathecal administration in 8 patients, including Ommaya reservoir placement in 4 patients. All patients completed protocol-specified chemotherapy without neurologic symptoms or surgical intervention. Risk factors for developing SEL include older age and high body mass index.

Conclusions—Investigation for SEL in leukemia or lymphoma patients with difficult lumbar punctures is warranted. Placement of an Ommaya reservoir may facilitate safe CNS-directed therapy in severely affected patients.

Keywords

spinal epidural lipomatosis; lumbar canal obstruction; difficult lumbar puncture; leukemia; lymphoma

Background

Patients with leukemia or lymphoma require multiple lumbar punctures (LPs) for diagnostic and therapeutic purposes [1]. Free-flowing cerebrospinal fluid (CSF) indicates satisfactory spinal needle placement. With optimal conditions of a well sedated and well positioned patient and an experienced clinician performing the LP, failure to obtain free-flowing CSF may indicate obstruction in the spinal canal. Congenital spinal canal stenosis, fluid collections or obstruction by tumors can impair CSF flow [2,3]. Another possible cause of obstruction is abnormal fat deposits in the epidural space, or spinal epidural lipomatosis (SEL).

SEL was first reported by Lee *et al.* in 1975 following renal transplantation[4]. Pathologic overgrowth of unencapsulated adipose tissue may cause direct mechanical compression of the spinal cord or nerve roots in the dural sac and vascular compromise[5,6]. Crowding of the dural sac may also impede collection of CSF by LP. SEL has been associated with long-term use of exogenous corticosteroids for solid-organ transplants and other conditions [7], endogenous steroid excess or hypercortisolism [8], drug induced lipodystrophy [9] and obesity [10]. Idiopathic (primary) SEL has been described in patients without known risk factors [5,11]. Patients with hematologic malignancies often receive long-term high-dose corticosteroid therapy, but with the exception of isolated case reports [12,13], this group is conspicuously missing from the SEL literature. Importantly, central nervous system (CNS)-directed therapy may be compromised by an inability to perform routine LPs successfully due to SEL. We review the clinical implications of this diagnosis in 14 patients with hematologic malignancies treated at a single institution.

Methods

Study population

Patients for review were identified through a search of the diagnostic imaging electronic records database [Cerner Classic PathNet and Cerner Millennium]. “Lipomatosis” was queried in spinal magnetic resonance imaging (MRI) electronic records for patients with leukemia and non-Hodgkin lymphoma (NHL) treated between January 1, 1999 and December 31, 2009. Overall, 14 patients were identified with a total of 22 diagnostic reports. For comparison of risk factors that may be associated with SEL, a cohort of 405 consecutive patients with newly diagnosed acute lymphoblastic leukemia (ALL) was analyzed. These patients were treated at on the Total study XV during a similar time period (June 2000 to October 2007)[14].

This study was approved by the St. Jude Children’s Hospital institutional review board.

Assessment and definition of potential risk factors

A retrospective review of medical records identified pertinent clinical and laboratory features, management and outcome. Treatment specific information included cumulative corticosteroid dose and total number of LPs with intrathecal medications prior to SEL diagnosis. All corticosteroid doses were converted to oral prednisone equivalent doses (mg/m²) for uniform analysis. All patients had at least 2 (range 2–22, median 4) successful LPs prior to SEL diagnosis.

Patients 2–19 years of age were classified as overweight (BMI between 85–95th percentile) or obese (BMI >95th percentile) at the time of malignancy diagnosis (<http://www.cdc.gov/obesity>). For older patients BMI>30 is considered obese and 25–29.9 overweight. Dyslipidemia was established using the National Cholesterol Education Program guidelines: total cholesterol > 200mg/dL; low-density lipoprotein (LDL) >130mg/

dL; high-density lipoprotein (HDL) <35mg/dL; and triglyceride > 130mg/dL (10–19 years old) or > 100mg/dL (0–9 years old) [15].

The definition of CNS involvement has been described previously [1]. An LP was classified as traumatic if the CSF contained at least 10 RBCs per microliter [16]. Finally, a “difficult LP” was defined as a procedure, under otherwise optimal conditions, that required multiple attempts (≥ 3), or where flow was slower than one drop of CSF every 3–5 seconds.

Radiology

MRIs were reviewed by a single neuroradiologist (KH). Sagittal T1 (TR 400–698, TE 9–13 msec) and T2 (TR 4000–6570, TE 82–116 msec) weighted images of the spine with 5 mm contiguous axial T1 (TR 400–1140, TE 10–12 msec) and T2W (TR 4380–9280, TE 89–90 msec) weighted images perpendicular to the vertebral bodies were acquired through areas of thecal sac narrowing. Fat saturation techniques were utilized in 7 cases. Measurements (in millimeters) of the width of the spinal canal at the point of maximal SEL (L4–5) were compared to the canal’s widest measurement (L2–L3). MRI at diagnosis of SEL and, when available, the most recent spinal MRI were reviewed for comparison. Images were also evaluated for congenital spinal canal narrowing, vertebral bony changes, or ligament hypertrophy that could contribute to canal narrowing independent of SEL by comparing measurements of the dural sac diameter at L2, L4, and L5 with published normal values [17]. Finally, visibility of CSF at L4–5 level despite the presence of SEL was noted

Statistical analyses

To study the association of clinical and laboratory risk factors with the development of SEL, 14 patients (SEL cases) were compared to 405 patients with newly diagnosed ALL (controls). The exact chi-square test was used for binary variables and the Wilcoxon signed rank test for continuous variables.

Results

Clinical Characteristics

From January 1, 1999 to December 31, 2009, 716 patients with ALL, 238 patients with acute myeloid leukemia (AML) and 174 patients with NHL were treated at St. Jude Children’s Research Hospital. Of these 1128 patients, 14 were diagnosed with SEL (incidence 1.2%). The 5 female and 9 male patients ranged in age between 3.9 and 22.6 years (median, 13 years) at the time of diagnosis of malignancy (Table 1). Ten patients were diagnosed with leukemia: 5 B-cell precursor ALL (including 2 with late marrow relapse), 4 T-cell ALL, and 1 AML. Four patients had NHL: 2 diffuse large B-cell lymphoma, one T-cell lymphoblastic lymphoma (SEL diagnosed at the time of early isolated CNS relapse), and one Burkitt lymphoma. Five patients (#1, 2, 3, 6, 14) had CNS2 disease at diagnosis. Initial lumbar puncture was traumatic with blasts in two patients (#1 and 6).

Diagnosis and Imaging of Spinal Epidural Lipomatosis

SEL was diagnosed in 10 patients within one month after starting therapy (range, 8 days to 30 days). The two patients with relapsed ALL were diagnosed with SEL at 18 months (#8) and 98 days (#9) after initiation of relapse therapy. Patient 13 with CNS relapse of NHL was diagnosed with SEL 118 days after initial diagnosis, but only 20 days after disease relapse.

The indication for MRI in the majority of patients (n=10) was “difficult LP.” In retrospect, one patient (#11) also complained of mild numbness in the extremities prior to SEL diagnosis; however, the neurologic examination was normal. Three patients had low back pain, one of whom (#3) presented at diagnosis with compression fractures due to presumed

leukemic infiltrates. One patient had MRI performed for signs of neurologic weakness while being treated for suspected sepsis (#14) and one patient with gluteal cleft drainage had imaging to rule out a fistula (#6).

Risk Factors

Five patients were overweight and three obese at the time of their malignancy diagnosis. At least one abnormality was noted in the lipid profiles of 10 patients in whom complete information was available at diagnosis of malignancy. Eight patients had low HDL levels (range 19–32), nine patients had high triglyceride levels (range 120–288), and one patient had a high total cholesterol level (202mg/dL).

The total systemic prednisone equivalent corticosteroid dose received by patients with ALL or NHL (excluding those with relapse) prior to SEL diagnosis ranged from 355 to 1167 mg/m². The two patients with relapsed ALL received cumulative doses of 9808 and 9732 mg/m² respectively (5691 and 1759 mg/m² during retrieval therapy). Patient 13 with relapsed NHL received 505 mg/m² prednisone during retrieval therapy for a cumulative dose of 2249 mg/m². The patient with AML did not receive systemic corticosteroids.

All patients received intrathecal administration of hydrocortisone prior to SEL diagnosis. In 11 patients without relapse, the cumulative dose of intrathecal hydrocortisone ranged from 12 to 81 mg/m² (number of intrathecal treatments ranged from 2 to 9). Patients 8 and 9 received cumulative intrathecal doses of 405mg/m² and 304mg/m² respectively (235 and 48mg/m² during retrieval therapy). Patient 13 was initially treated with intrathecal methotrexate and cytarabine only, but during retrieval therapy he received a cumulative dose of 23 mg/m² intrathecal hydrocortisone before SEL diagnosis.

When compared to patients with newly diagnosed ALL, black race, older age, high BMI percentile for age and presence of CNS disease at diagnosis were identified as risk factors for developing SEL (Table 2). Patients with SEL had higher median HDL levels, but differences in the other parameters of the lipid profile did not reach statistical significance.

Radiology

Spinal epidural lipomatosis involved the posterior thoracic, lumbar, and sacral spinal canal in 13 patients; patient 8 had lumbar and sacral SEL only (Table 3). In 7 patients, SEL was also noted in the anterior portion of the lumbar canal (#2, 3, 5, 6, 11, 12, 13). In 5 patients, canal narrowing was compounded by congenital narrowing of the lumbar canal (#2, 7, 9, 10) or degenerative vertebrae (#8) when compared to age-matched normal references [17]. All patients except two (#2 and 13) had some degree of visible CSF in the lumbar region despite SEL compression of nerve roots.

Percentage of canal narrowing due to SEL was determined by comparing the canal width at L4-5 to the widest part of the canal in the lumbar region at L2 in the same patient. Age-matched normal reference values identified vertebral body diameter and dural sac diameter only and could not be used for comparison [17]. In our patient cohort, this ratio ranged from 0.52 to 1, indicating that the canal was narrowed by 0–48% by fat deposition at L4-5. All patients also had varying degrees of fat deposition at the L2 level, thus this ratio may not be truly reflective in some patients and underestimates the true extent of narrowing at L4-5. The presence or absence of anterior fat deposition was included as a marker of the severity of lipomatosis only since it does not interfere with the clinical procedure. These data are shown in Table 3.

Management of SEL

Ten patients were diagnosed with SEL due to difficulty in performing LP (#1, 2, 3, 4, 5, 7, 8, 11, 12, 13). Ommaya reservoir was placed for continued intrathecal therapy in 4 patients (#2, 4, 7, 13). Patient 4 had an almost 50% narrowing of the canal by SEL. Patients 2 and 7 had congenital stenosis combined with approximately 30% to 40% narrowing of the canal by SEL. MRI images of the spinal canal of patient 7 are shown in Figure 1. Patient 13 had ligamentum flavum hypertrophy in addition to SEL filling of the entire length of the lumbar canal that may have led to underestimation of narrowing at L4-5. Specifically at L4-5, sagittal images demonstrated compression of nerve roots and lack of visible CSF (Figure 2). Following review of MRIs, subsequent LPs were successful in patient 8 under fluoroscopy and in patients 3, 5, and 11 by inserting the spinal needle in the adjacent intervertebral space which demonstrated a lesser degree of obstruction. Since CSF was clearly visible at L4-5 for patient 12 despite obstruction by SEL and this patient with Burkitt lymphoma did not require frequent LPs, subsequent attempts did not require specific intervention. Patient 1 had 29% narrowing of the spinal canal by SEL, but since CSF was clearly visible at L4-5 and she did not have additional contributing factors to obstruction, no further intervention was required. None of the remaining four patients (#6, 9, 10, 14) in whom SEL was diagnosed for various other symptoms or as an incidental finding required interventions for intrathecal treatment.

Outcome

None of the patients required surgical procedures, such as decompression, for management of SEL. Follow up imaging was available for five patients (#2, 4, 6, 12, 13) at various time points after SEL diagnosis (range, 1 to 22 months). Due to limited follow-up studies, the natural history of SEL could not be studied, though all five patients demonstrated gradual improvement in the extent of SEL (data not shown).

All patients continued protocol specified chemotherapy without alterations in doses of systemic corticosteroids or CNS-directed therapy due to SEL. Twelve patients are in remission from their malignancy for a median of 2.4 years (range, 0.4 to 7.5 years). Patient #14 with AML developed bone marrow relapse and subsequently died. Patient #13 with relapsed NHL also died of progressive disease.

Discussion

Frequent lumbar punctures are essential in the management of childhood hematological malignancies [1]. This procedure may be complicated by red blood cell contamination or poorly flowing CSF. Previously we have identified risk factors that may be modified to reduce the incidence of traumatic taps [18]. In this retrospective review we identify another potentially modifiable factor, SEL, that may lead to difficult LPs. Though SEL is an uncommon condition with only 104 cases in a recent literature review [7], we have identified 14 cases in our leukemia/lymphoma treatment population over the past decade. Since SEL can impede the optimal delivery of intrathecal medications, we identified risk factors of steroid administration, age, race, and obesity as well as guidelines for management in this treatment population.

A limitation of this retrospective review is the lack of use of an established grading system for measuring extent of SEL in pediatrics. An objective MRI grading system for adults with SEL has been previously published [19]. This technique utilizes linear measurements in the axial plane, parallel and tangent to the superior end plate of S1 vertebral body. Conventional MRI does not capture images in this specific plane, and it was not feasible to reconstruct the images in the axial plane while maintaining imaging detail. Thus, in our analysis, we

quantified the extent of SEL by comparing the widest measurement at L2 with the width of the canal at maximal compression specifically focusing at the clinically relevant L4-5 level. In our patient cohort, SEL compromised the width of the lumbar canal by a range of 13–48%. When coupled with co-morbidities such as congenital spinal canal narrowing, degenerative vertebrae or ligament hypertrophy, this presents a potentially significant obstacle to subsequent intrathecal treatment. Capturing spinal images utilizing techniques similar to Borre *et al* could improve comparison to adult grading systems for future patients, but a pediatric grading system has not yet been established and would likely be more beneficial in correlating SEL with management considerations in this population. While the most common presenting features of SEL in adult patients are neurologic symptoms of back pain and sensory and/or motor difficulties [7], children may present with more subtle findings. In the study by Kano *et al* [20], an MRI was performed in children receiving chronic steroid therapy for renal disorders if they were obese or if they complained of symptoms of SEL, such as back pain or numbness. SEL was diagnosed in 5 of 125 (4%) patients who all had clinical symptoms and 4 were obese. The true incidence of subclinical SEL may have been higher. In our study, the majority of patients with SEL did not exhibit symptoms and were diagnosed after difficult or failed LP attempts.

In addition to steroid treatment, obesity has been linked to SEL. In our cohort, 8/14 patients were overweight or obese. Abnormal lipid profiles are a significant co-morbidity with obesity and were investigated as additional risk factors for development of SEL. Of the 10 patients with available lipid profiles, all patients had at least one abnormal value, but it was not possible to determine if all panels were obtained during a fasting state. In addition, lipid panels were not obtained after initiation of corticosteroids, thus we could not ascertain whether deranged lipids post corticosteroids further promoted abnormal fat deposition in the spinal canal. Finally, we cannot exclude familial influences on development of dyslipidemia.

Current evidence does not suggest that the duration, dose, and type of corticosteroid administered influences the development of SEL [21]. All patients in the study were diagnosed with SEL after undergoing at least 2 successful LPs, and the majority of patients (10/14) were diagnosed within one month from the time of initial diagnosis. However, this first month of therapy features intensive systemic and intrathecal steroid administration in previously healthy patients (i.e. steroid-naïve). Childhood leukemia therapy, particularly steroid use, is an established risk factor for developing obesity [22,23]. It is possible that accumulation of fat in the spinal canal as SEL is a direct extension of the typical truncal obesity that results from exogenous corticosteroid therapy.

We chose a cohort of patients with newly diagnosed ALL treated on a contemporary clinical trial during a similar time period to identify risk factors associated with development of SEL in children with hematologic malignancies. Though this comparison is limited due to the small number of SEL cases, heterogeneous diagnoses and treatment, children that are older, black race and overweight were significantly over-represented in the SEL cohort.

The diagnosis of SEL and additional compounding factors led to changes in the administration of subsequent intrathecal chemotherapy for 8 patients including placement of an Ommaya reservoir in 4 patients and modification of the technique (fluoroscopy in one patient or level of the lumbar puncture in three patients). Patients investigated for “difficult LP” demonstrating canal narrowing >25% by SEL and compounding risk factors such as congenital spinal canal stenosis frequently required interventions. No additional surgical procedure was required for the management of SEL in our patients compared to an adult series where 75–95% of patients underwent surgical laminectomy and debulking for symptomatic relief [5,7]. Certain reports suggest that tapering the dose of corticosteroids may reduce complications of SEL [20,24]. This strategy is neither feasible nor

recommended for patients with hematologic malignancies. All patients in our study continued to receive protocol specified systemic and intrathecal steroids without progression of symptoms or extent of SEL (in follow up MRIs when available). None of the patients developed subsequent CNS relapse suggesting that CNS-directed therapy was not compromised due to SEL.

In conclusion, though SEL is a rare disorder, it can significantly impact intrathecal therapy required in patients with leukemia/lymphoma. Limited by the retrospective nature of this review, we recognize the need for further study in the pediatric oncology population including patients receiving corticosteroids for brain tumors or graft-versus-host disease. Heightened awareness of this diagnosis, especially in patients with difficult or “low-flow” CSF during routine lumbar punctures may help determine the true prevalence. Adoption of a more sophisticated imaging and grading technique, applied prospectively to patients imaged for a suspicion of SEL could lead to a better identification of patients at risk. It may also help in formulating guidelines for optimal management by identifying patients who need immediate intervention (i.e. ommaya reservoir placement) vs. those who could be managed conservatively. The recognition and appropriate management of SEL in patients with leukemia/lymphoma may help reduce unsuccessful LPs which can delay therapy or lead to traumatic lumbar punctures, directly impacting the quality of care for patients.

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Figure 1. Lumbar MRI views for patient # 7 (T1-weighted MRI on left, T2-weighted MRI on right). Black arrows demonstrate narrowing of the spinal canal and significant SEL (bright) in the lumbosacral region, and compression of cauda equina nerve roots (dark linear).

Figure 2. Axial T2 weighted MRI, L4-5 level for patient #13. Epidural fat completely surrounds and compresses spinal cord nerve roots (narrow arrows). Note facet hypertrophy (block arrows) which further narrows the canal. This patient also has abundant subcutaneous fat.

Table 1
Clinical characteristics at primary diagnosis, corticosteroid dose, and outcomes of patients with SEL

Patient	Primary diagnosis	Age (yrs)	Time to develop SEL	Indication for MRI	Cumulative steroids (mg/m ²)		BMI (overweight or obese)	Management of subsequent IT therapy	Outcome (duration of CR yrs)
					Systemic	Intrathecal			
1	Pre-B ALL	7.4	30 days	Difficult LP	1120	48	16.7	No change	CR (5.4)
2	Pre-B ALL	3.9	23 days	Difficult LP	867	64	17.4 (Overweight)	Ommaya	CR (2.4)
3	Pre-B ALL	13.3	25 days	Difficult LP	1167	77	32.1 (Obese)	LP at L3-4	CR (0.4)
4	T-cell ALL	10.5	24 days	Difficult LP	939	61	17	Ommaya	CR (3.5)
5	T-cell ALL	5.9	9 days	Difficult LP	355	63	13.2	LP at L3-4	CR (2.5)
6	T-cell ALL	16.7	20 days	Draining gluteal cleft	1095	63	27 (Overweight)	No change	CR (2.2)
7	T-cell ALL	18.5	21 days	Difficult LP	800	768	24.8	Ommaya	CR (0.9)
8	Relapsed pre-B ALL	22.4	[^] 18 mo	Difficult LP	9809	405	31 (Obese)	LP under fluoroscopy	CR (2.4)
9	Relapsed pre-B ALL	15.1	[^] 98 days	Low back pain	9732	304	27.8 (Overweight)	No change	CR (3.6)
10	DLBCL	11.3	25 days	Low back pain	879	51.7	14.8	No change	CR (7.5)
11	DLBCL	12.9	30 days	Difficult LP	1020	41	22.6	LP at L3-4	CR (1.7)
12	Burkitt lymphoma	8.5	8 days	Difficult LP	480	12	19.9 (Overweight)	No change	CR (4.3)
13	Relapsed T-cell NHL	22.6	[^] 20days	Difficult LP	22497	23	(Overweight)	Ommaya	PD, death
14	AML-M4	17.3	77 days	Neurologic weakness	0	36	42.3 (Obese)	No change	PD, death

[^] from disease relapse

DLBCL = diffuse large B cell lymphoma, NHL = non-hodgkin lymphoma, LP = lumbar puncture, CR= complete remission, PD= progressive disease

Table 2
Risk factors associated with the development of SEL in children with hematologic malignancies

Variable	SEL patients		Control patients		P-value
	No. of patients (%)		No. of patients (%)		
Binary variables					
Race ^a	White	8 (57.1)	319 (78.8)		0.0312
	Black	6 (42.9)	70 (17.3)		
Gender	Male	9 (64.3)	225 (55.6)		0.59
	Female	5 (35.7)	180 (44.4)		
CNS status	CNS1	7 (50)	304 (75.1)		0.016
	CNS2	3 (21.4)	72 (17.8)		
	CNS3	2 (14.3)	9 (4.9)		
	Traumatic with blasts	2 (14.3)	20 (4.9)		
Treatment risk group ^b	Low risk	3 (30)	19 5(48.1)		0.34
	Standard/High risk	7 (70)	210 (51.9)		
	Underweight	1 (7.1)	52 (13.1)		0.0175
	Healthy weight	5 (35.7)	254 (62.7)		
Overweight	5 (35.7)	40 (9.9)			
Weight category	Obese	3 (21.4)	58 (14.3)		
Continuous variables					
Age		14	405		0.0001
	Median	13.1	5.4		
	Range	3.9–22.3	1.0–18.8		
High density lipoprotein	Median		262		0.015
	Range	29	3–61		
Low density lipoprotein		10	256		0.43
	Median	91	81.5		
	Range	57–131	15–236		
Triglycerides		10	267		0.58

	SEL patients		Control patients		P-value
Variable	No. of patients (%)		No. of patients (%)		
	Median		143		
	Range		35–917		132 83–288
Total cholesterol		10		265	0.29
	Median		136		
	Range		64–289		

^a 16 patients from control group with race classified as “other” were excluded

^b Patients with leukemia only

Table 3

Analysis of MRI findings for patients with SEL and comparison of canal widths

Patient	L4-5 fat posterior (mm)	L4-5 fat anterior (mm)	L4-5 canal (mm)	Widest canal (mm)	Ratio (available L4-5/widest canal)	% of canal narrowing at L4-5 compared to widest canal	Additional risk factors	CSF visible at L4-5	Longitudinal extent of SEL
1	7	n/a	5	7	0.71	29	none	Yes	TLS
2	9.9	8.6	3	5.2	0.58	42	Stenosis	No	T1-11; LS
3	9.4	3.7	9.4	10.1	0.93	7	none	Yes	TLS
4	8.4	n/a	6.3	12.1	0.52	48	none	Yes	TLS
5	11.1	2.8	7.2	13.7	0.53	47	none	Yes	T2-10; LS
6	6.3	6.3	7.3	8.4	0.87	13	none	Yes	T1-10; LS
7	11.5	n/a	6.3	8.9	0.71	29	Stenosis	Yes	T1-8; LS
8	5	n/a	7	13	0.54	46	DV; LFH	Yes	L4-S1
9	7.3	n/a	8.4	10.5	0.8	20	Stenosis	Yes	T3-9 L1-5 S1-2
10	7.5	n/a	8.3	8.3	1	0	Stenosis	Yes	TLS
11	3.1	6.6	6.6	9.7	0.68	32	none	Yes	T3-9; L2-5; S
12	5.4	9.7	5.4	8	0.68	32	none	Yes	T2-T11 L1-5 S
13	3.5	4.6	10.9	10.9	1	0	LFH	No	TLS
14	3.7	n/a	14.3	14.3	1	0	none	Yes	TLS

(DV = degenerative vertebrae, LFH = ligamentum flavum hypertrophy, T = thoracic, L = lumbar, S = sacral, SEL = Spinal Epidural Lipomatosis, csf = cerebrospinal fluid, T3 (et) denotes spinal region and level, i.e. 3rd thoracic vertebrae)