

## Case Report

# Pigmented Villonodular Synovitis on Lumbar Spine : A Case Report and Literature Review

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Pigmented villonodular synovitis (PVNS) is a benign proliferative joint disease with an uncertain etiology that uncommonly involves the spine. We present a case of PVNS involving the lumbar spine. A 38-year-old male developed back pain and pain in both legs caused by a mass in the L4 region of the right lamina. After gross total tumor removal, the symptoms improved. The pathological finding was synovial hyperplasia with accumulation of hemosiderin-laden macrophages. He was diagnosed with PVNS and experienced no recurrence for up to 2 years after surgery. In this report, we review the previous literature and discuss etiology, clinical manifestations, diagnosis, and treatment.

**Key Words :** Pigmented villonodular synovitis · Lumbar spine · Giant cell tumor.

## INTRODUCTION

Pigmented villonodular synovitis (PVNS) is a benign proliferative disease of the synovial membrane with an uncertain etiology. This tumor is characterized by the presence of inflammation and hemosiderin deposition in the synovium. PVNS commonly involves the large appendicular joints, but the axial skeletal system is rarely involved<sup>20,22,34,44,47</sup>. After the first reported case of PVNS involving the spine at 1980<sup>29</sup>, only a few cases have been reported in the English-language literature. The etiology, natural history, and treatment of PVNS remain unclear. We present a case of PVNS and review the pertinent literature.

## CASE REPORT

A 38-year-old man had complained of an 8-month history of lower back pain and pain in both legs. His symptoms became particularly apparent after an exercise session one month prior to presentation when he noticed that claudication developed after a long walk. He had no antecedent trauma history. An initial neurological examination demonstrated full and symmetric muscle strength in the lower extremities. Sensation was intact to all modalities, and reflexes were symmetrical and nonpathological.

Plain radiographs of the lumbar spine revealed spondylolisthesis at L4-5. Magnetic resonance imaging (MRI) revealed a well-circumscribed 1.4-cm soft tissue mass in the region of the

right lamina of L4 (Fig. 1, 2). This lesion originated from the right superior articular process of the L4 vertebra with extension to the spinal canal, forming an extradural mass that compressed the thecal sac.

A midline incision and L3-4 partial hemilaminectomy with a right approach was performed to resect the mass. The ligamentum flavum was thickened with inflammatory changes. After removing the ligamentum flavum, a yellowish-dark colored mass attached to the thecal sac was noted. Gross total removal was performed without any damage to the surrounding tissues.

A histopathologic examination revealed a tumor composed of thick-walled blood vessels with extensive fibrosis and inflamed granulation tissue, and synovial hyperplasia with accumulation of hemosiderin-laden macrophages (Fig. 3). A diagnosis of PVNS was made.

The patient tolerated surgery well and made an uneventful recovery. The pain in the lower back and both legs improved significantly immediately after surgery, although there was some residual pain in the left calf. Follow-up contrast enhanced MRI performed 1 month after surgery revealed the absence of any residual mass (Fig. 4). No recurrence of symptoms was observed over the next 2 years.

## DISCUSSION

The World Health Organization classification defines diffuse

• Received : February 24, 2014 • Revised : April 6, 2014 • Accepted : August 14, 2014

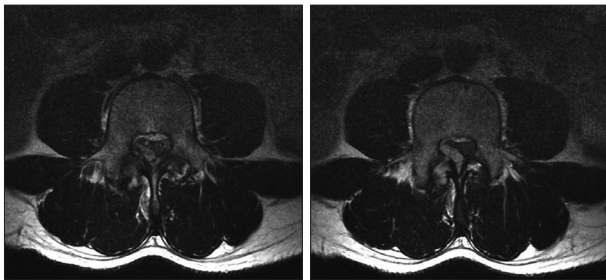
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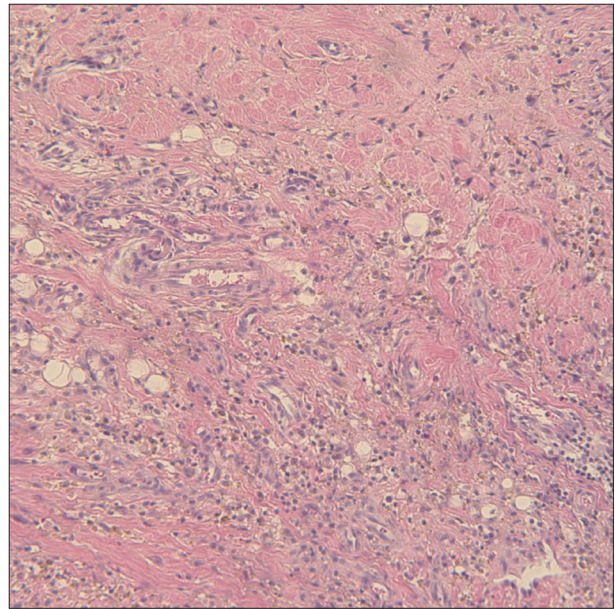
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**Fig. 1.** T2-weighted sagittal lumbar magnetic resonance image shows a well-circumscribed mass at the L4 epidural space compressing the spinal cord (arrow).



**Fig. 2.** T2-weighted axial lumbar magnetic resonance image shows a mass originating in the right superior articular process of the L4 vertebrae and extending to the epidural space.



**Fig. 3.** The surgical specimen shows synovial hyperplasia containing round cells, foamy cells, and hemosiderin-laden macrophages.



**Fig. 4.** Postoperative T2-weight sagittal lumbar magnetic resonance image shows the absence of the lesion and postoperative changes.

PVNS as a diffuse-type giant cell tumor that differentiates from a giant cell tumor of the tendon sheath and localized PVNS<sup>19</sup>. The nomenclature that describes a synovial-type giant cell tumor reflects the uncertain pathogenesis and various clinical manifestations: tenosynovitis, diffuse type PVNS, xanthomatous giant cell tumor, histiocytic giant cell tumor, xanthoma, benign synovialoma, hemorrhagic villous arthritis, and localized or diffuse pigmented villonodular synovitis. Among them, most synovial-type giant cell tumors of the spine published to date have been called pigmented villonodular synovitis.

Furlong et al.<sup>20</sup> reported 15 cases of a synovial-type giant cell tumor of the vertebral column. Eight cases displayed a diffuse growth pattern, and only one showed typical PVNS morphology. One case of the remaining seven cases was a localized pattern and six cases were an intermediate growth pattern that was difficult to subclassify. Most reports of synovial-type giant cell tumors have excluded a description of the pathological subtype, perhaps because the pathological subtype of PVNS is not related to the surgical plan for spinal PVNS cases, unlike cases involving the appendicular joints. Appendicular tenosynovial giant cell tumors are typically a diffuse type that displays a higher recurrence rate than

that of the localized type. However, the small number of reported cases involving the spine and lack of information on subtype hinders typing.

The etiology of PVNS remains controversial. To date, hyperplasia, local metabolic disturbance, recurrent hemorrhage, and trauma have been mentioned as possible etiologic factors, while the inflammatory reaction and neoplasm are less clear and contentious<sup>2,21,23,50</sup>. The reason for the confusion concerning the causative factor of inflammation is that the cells involved are usually inflammatory cells, such as polyclonal CD8 positive T cells<sup>32,37</sup>, whereas the presence of trisomy 7 and clonal DNA rearrangements represent the neoplasm<sup>1,3,41</sup>.

**Table 1.** Clinical features and follow-up data of 63 previously reported pigmented villonodular synovitis cases of the spine

Author (year)	Location	Age/sex	Clinical manifestation	Treatment	Clinical result	Follow up period
Teixeira et al. <sup>48)</sup> (2012)	C1-2	31/NA	None	GTR	NED	18 months
Hsieh et al. <sup>25)</sup> (2012)	L2-3	39/M	Localized Pain, radiculopathy	GTR	Recurrences	3 months
Okutan et al. <sup>38)</sup> (2012)	C6-T2	65/M	Localized pain	GTR	NED	6 months
Yener et al. <sup>51)</sup> (2010)	L2	66/F	Localized pain, claudication	GTR	NA	NA
del Carmen Baena-Ocampo et al. <sup>12)</sup> (2009)	T8-10	17/M	Paraplegia	GTR	NED	6 years
Pechlivanis et al. <sup>40)</sup> (2009)	C7-T2	35/M	NA	GTR	NED	2 years
Musuluman et al. <sup>33)</sup> (2009)	L4	59/F	Radiculopathy	GTR	NA	NA
Rovner et al. <sup>45)</sup> (2008)	L5	37/F	Localized Pain	GTR	NA	NA
Donna et al. <sup>5)</sup> (2008)	C1	43/M	None	GTR	NA	NA
Finn et al. <sup>18)</sup> (2007)	C2	82/F	Pathologic fracture, quadriplegia	STR	NED	2 years
Oda et al. <sup>35)</sup> (2007)	Sacrum	53/F	Radiculopathy	STR	1st recurrences	10 months
				GTR	2nd recurrences	7 months
				Chemo	NA	NA
Oe et al. <sup>36)</sup> (2007)	L4-5	43/M	Sciatica	GTR	NED	3 years
Hansen et al. <sup>24)</sup> (2007)	T6-7	17/M	Weakness	GTR	NA	NA
Hermann et al. <sup>26)</sup> (2007)	L3-5	50/M	NA	GTR	NED	2 years
de Zee et al. <sup>11)</sup> (2007)	T6-7	17/M	NA	GTR	NA	NA
Doita et al. <sup>16)</sup> (2005)	T7	26/M	Localized pain	GTR	NED	2 years
Furlong et al. <sup>20)</sup> (2003)	Sacrum	17/M	NA	GTR	NA	NA
	T4-5	21/F	NA	GTR	NA	NA
	C2-3	23/M	NA	STR	Recurrences	4 years
			NA	GTR+RT	NED	1 year
	C3-5	25/M	NA	GTR	NA	NA
	L4-5	25/F	NA	STR	Recurrences	2 years
			NA	GTR	Residual mass	NA
	L4	29/F	NA	En blot resection	NED	4 years
	C5-6	31/M	NA	GTR	NED	9 years
	C3-4	32/F	NA	GTR	NED	3 years
	C3-4	32/M	NA	STR	Recurrences	12 months
			NA	STR+RT	Residual (stable) mass	2 years
	C	35/M	NA	GTR+RT	NED	7 years
	C	39/F	NA	Biopsy	Recurrences	2 years
			NA	GTR	NED	10 years
	C5-6	39/F	NA	GTR	NA	NA
	C5-6	43/F	NA	STR	NA	NA
	C	44/M	NA	GTR+RT	NED	4 months
	C4-5	44/F	NA	Biopsy	NA	NA
Dingle et al. <sup>14)</sup> (2002)	C4	27/F	Localized pain	STR	Residual (stable) mass	2 years
Sampathkumar et al. <sup>46)</sup> (2001)	L5-S1	71/F	Radiculopathy	GTR	NED	3 years
Dimeco et al. <sup>13)</sup> (2001)	L3-4	70/F	Radiculopathy	GTR	NED	1 year
Bruecks et al. <sup>6)</sup> (2001)	T8	13/F	Myelopathy	GTR	NA	NA
Clerc et al. <sup>9)</sup> (1999)	L1-5	60/M	Radiculopathy, localized pain	Biopsy+RT	Recurrences	1 year
				STR+chemo	Residual mass	NA
Giannini et al. <sup>22)</sup> (1996)	C5	37/F	NA	GTR	NED	9 years
	L5-S1	29/F	NA	GTR	Recurrences	1 year
				GTR	NED	23 months
	C7	37/F	NA	GTR	NED	66 months
	C4-5	38/M	NA	GTR	Recurrences	1 year
				GTR	NED	55 months

**Table 1.** Continued

Author (year)	Location	Age/sex	Clinical manifestation	Treatment	Clinical result	Follow up period
	T11	40/M	NA	GTR	NED	64 months
	T3	21/F	NA	GTR	NED	53 months
	T7-8	23/M	NA	GTR	NED	42 months
	C5	43/M	NA	GTR	NED	36 months
	L3	42/M	NA	STR	Residual (stable) mass	14 months
	L3	67/M	NA	NA	NA	NA
	C6	26/M	NA	GTR	NED	11 months
	C5-6	44/M	NA	Biopsy+GTR	NED	6 months
Gezen et al. <sup>211</sup> (1996)	T9-12	19/M	Weakness	STR	Residual (stable) mass	16 months
Clark et al. <sup>81</sup> (1993)	T7-8	23/M	Weakness	GTR	NED	42 months
Titelbaum et al. <sup>491</sup> (1992)	L4-5	52/F	Radiculopathy	GTR	NA	NA
Mahmood et al. <sup>311</sup> (1992)	C6-7	43/F	None	GTR	NA	NA
Kuwabara et al. <sup>301</sup> (1992)	T8-11	25/F	NA	STR+RT+chemo	NED	7 years
Khoury et al. <sup>281</sup> (1991)	L4-5	61/F	Radiculopathy	GTR	NA	NA
	C4-5	84/M	Weakness	GTR	NA	NA
Karnezis et al. <sup>271</sup> (1990)	C6-7	37/F	Localized pain	GTR	NED	79 months
Retrum et al. <sup>431</sup> (1987)	L5-S1	81/F	Localized pain	GTR	NA	NA
Weidner et al. <sup>501</sup> (1986)	L5-6	48/F	Radiculopathy	GTR	1st recurrences	4 months
				GTR	2nd recurrences	4 months
				Radical resection	NED	2 months
	L2-5	34/F	Localized pain, radiculopathy	STR	NED	12 months
Pultizer and Reed <sup>421</sup> (1984)	C1-4	35/F	None	GTR	NED	11 years
	L5-S1	23/M	None	En bloc resection	NA	NA
Campbell and Wells <sup>71</sup> (1982)	L4-5	54/F	Localized pain	GTR	NED	31 months
Kleinman et al. <sup>291</sup> (1980)	C3-6	65/F	Localized pain, myelopathy	Biopsy	Residual (stable) mass	8 months

M : male, F : female, C : cervical, T : thoracic, L : lumbar, GTR : gross total removal, STR : subtotal removal, RT : radiation therapy, Chemo : chemotherapy, NED : no evidence of disease, NA : not available

We reviewed 63 cases of spinal PVNS reported in the English literature (Table 1). Median patient age was 37 years (range, 13–84 years), and males were younger (35 years) than females (39 years). Twenty-eight cases (44.4%) were identified in the cervical spine, 12 cases (19%) in the thoracic spine and 21 cases (33.3%) in the lumbar spine. As the lesion origin facet joint mostly contains synovium, spinal PVNS is typically located in the posterior element<sup>201</sup>. Rare cases have been reported in which spinal PVNS arose from the atlantoaxial or atlantooccipital joints, which are a tendon sheath or synovial membrane containing joints, and not a facet joint<sup>5,18,201</sup>. Additionally, spinal PVNS involving anterior spinal structures has been reported<sup>22,421</sup>. It is thought to arise from synovial membranes of the vertebral column accessory joints. Pain localized to the spinal region is the most common symptom. As PVNS tends to arise from the posterior elements, it is often accompanied by radicular pain or myelopathic deficits<sup>6,201</sup>.

The pathological findings of PVNS in the appendicular skeleton and spine are similar<sup>281</sup>. However, PVNS of the spine often exhibits less pigmentation than that in the appendicular joint<sup>391</sup>. The histological appearance is characterized by a proliferation of synovial cells and includes lipid and hemosiderin-laden macrophages, multinucleated giant cells, mixed round cell infiltration,

mononuclear polyhedral cells of fibrohistiocytic origin, and stromal and fibroblast cell proliferation<sup>4,101</sup>. Diffuse-type PVNS has greater vascularity, a greater number of hemosiderin-laden macrophages, and a typically villous pattern, which is often absent in localized PVNS<sup>341</sup>.

The best choice of diagnosis for PVNS is magnetic resonance imaging (MRI). MRI shows the mass as mixed signal intensity on T2-weighted images due to hemosiderin deposition and can reveal the extent of the lesion, bony erosion, and invasion. Differential diagnoses includes primary bone lesion, mesenchymal neoplasm and extradural masses, such as osteoblastoma, fibrohistiocytic tumor, schwannoma, hemangiopericytoma, tendinous xanthoma, and hypertrophic synovitis<sup>221</sup>.

The treatment goal for patients with PVNS is gross total excision of the mass with functional preservation and lowering of recurrence rate<sup>201</sup>. The local recurrence rate of spinal PVNS is 18–25% for all spinal cases<sup>221</sup>. It is similar with the rate of appendicular PVNS reported in different studies (0–50%)<sup>14,17,23,341</sup>. Various treatments for PVNS include surgery, radiation therapy, radioisotope infusions, and chemotherapy<sup>8,21,22,33,481</sup>. Surgical excision with the aim of gross total resection is accepted as the treatment of choice. However, after surgery, evidence is lacking concerning



the need for adjuvant treatment to lower the recurrence rate as well as the standard protocol for recurrent PVNS. The extent of resection is currently the only known influence on recurrence rate<sup>20,22</sup>. Among the 63 cases we reviewed, only four of 46 patients (8.7%) who underwent gross total resection recurred, whereas four of 10 patients (40%) who underwent subtotal resection recurred (Table 1). Three patients received adjuvant chemotherapy and six patients received radiotherapy (RT). No recurrence was found for patients who received adjuvant chemotherapy, but one patient who received RT recurred. The efficacy of the treatment is unclear because there were an insufficient number of patients who received chemotherapy or RT. Therefore, surgical resection remains the treatment of choice for PVNS, unless chemotherapy or RT is a treatment option

## CONCLUSION

PVNS involving the spine is rare. The recommended method for treating spinal PVNS is gross total removal. No consensus has been reached regarding the efficacy of chemotherapy or RT as adjuvant treatment, because only a few cases have been presented. Therefore, surgical resection should be considered the first treatment for PVNS. More case studies of spinal PVNS are needed to clarify treatment following surgery.

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