Case Report

# A very rare spinal cord tumor primary spinal oligodendroglioma: A review of sixty cases in the literature


## ABSTRACT

Literature review. In this study, we evaluated a case of primary spinal oligodendroglioma (PSO) with a rare localization between L3 and S2, and also examined sixty cases in the literature in terms of demographic characteristics, clinical, radiological, and histopathological characteristics, and treatment planning. A case of PSO has been presented, and the relevant literature between 1931 and 2016 was reviewed. A total of 57 papers regarding PSO were found and utilized in this review. The main treatment options include radical surgical excision with neuromonitoring, followed by radiotherapy. Despite these treatment protocols, the relapse rate is high, and treatment does not significantly prolong survival. Oligodendrogliomas are rare among the primary spinal cord tumors. Oligodendrogliomas are predominantly found in the cervical spinal cord, thoracic spinal cord, or junctions during childhood and adulthood. Extension to the sacral region, inferior to the Conus, is very rare. Furthermore, of the sixty cases in the literature, the case we present here is the first to be reported in this particular age group. These localizations usually occur in the pediatric age group and after relapses. While for a limited number of cases the oligodendroglioma initiates in the thoracic region and reaches as far as L2, we encountered a case of an oligodendroglioma within the range of L3 to S2. Clinical findings are observed in accordance with location, and magnetic resonance imaging is the gold standard for diagnosis.



**Keywords:** Management, primary spinal oligodendroglioma, review


## INTRODUCTION

Primary spinal oligodendrogliomas (PSOs) are rare pathological entities. They constitute <2% of all intramedullary (IM) spinal tumors and sixty cases have been reported in the literature.[1-4] PSOs can occur in children and adults, and there is slight male predominance. Depending on the tumor's anatomical location, symptoms generally include motor deficits, sphincter dysfunction, pain, and sensory deficits. In rare cases, PSOs may involve the entire spinal cord, and emerge accordingly with a rise in intracranial pressure.[3-6] Magnetic resonance imaging (MRI) is the gold standard for diagnosis and surgical planning for the PSOs. Radiography or computed tomography should be performed in cases with very large tumors causing skeletal deformation. The brain and the entire spinal axis should be examined for any potential seeding.[3,4,6-9] Aggressive surgical tumor excision using microsurgical techniques and intraoperative electrophysiological monitoring is the main treatment for PSO. Tumor extirpation is not possible in the majority of cases due to the infiltrative nature of the tumors.[2-5,9-11] Although employing postsurgical chemotherapy (CMT) and/or radiotherapy (RT) is controversial, it is recommended for patients with a high relapse rate. Despite all current treatments, the prognosis for a PSO is poor.[4,5,7,12-14]


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| **DOI:** 10.4103/jcvjs.JCVJS_1_17 | |





**How to cite this article:** Hasturk AE, Gokce EC, Elbir C, Gel G, Canbay S. A very rare spinal cord tumor primary spinal oligodendroglioma: A review of sixty cases in the literature. J Craniovert Jun Spine 2017;8:253-62.

## MATERIALS AND METHODS

In this study, the literature was reviewed for PSOs. A detailed electronic search was carried out using the Medical Subject Headings term "PSOs" in the MEDLINE, PubMed, and Google databases for studies published from 1931 to 2016. We evaluated a case of PSO and examined sixty cases from the literature in terms of demographic, clinical, radiological, and histopathological characteristics and treatment planning. Table 1 summarizes the reviewed cases [Table 1].[1-57]

## CASE REPORT

A 28-year-old male patient with the complaints of leg weakness, headache, refractory constipation, and numbness in the legs was initially seen abroad. A mass was detected between L3 and S2 on an MRI scan, and the patient underwent two operations under general anesthesia. Nonetheless, over time, his symptoms became more severe, and he was admitted to our clinic. The first pathological report from the foreign clinic was insufficient material, and the second reported as a malignant tumor. In the MRI scans, a contrast-enhancing intradural mass between L3 and S2 causing contrast enhancement of bony tissues was detected [Figure 1]. The patient was operated on under standard conditions with neuromonitoring. While in a prone position, a midline skin incision was made. A needle biopsy (Bx) was performed from a 1-cm area in the sacral region, which was disproportionate to the size of the previous skin incision. No laminectomy had been performed along the length of the tumor. Hence, a laminectomy was performed without damaging the facets from L2 until the interior end of S2 and the dura was reached. The dura was cut through the midline, pads were placed around the tumor, and the tumor was suspended laterally. Hemorrhagic dark-colored tumor tissue that was soft and fragile with dirty-gray-colored regionswas removed from between the fibers of the cauda from the upper margin of the end of the cord to the lower margin of S2 [Figure 2]. The primary dural closure was performed using hemostasis and SF. No abnormalities were detected in theneuromonitoring records [Figure 3]. At a postoperative follow-up, the patient could move his four extremities and had a GCS score of 15. On postoperative day 1, the wound was clean, headache relieved, and the patient could defecate comfortably. The patient was then discharged from the hospital and followed-up as an outpatient. Histopathological assessment of the Bx indicated that the tumor was an oligodendroglioma; the tissue had highcellularityand composed of two types of cell components with well-defined cell borders, friedegg-like cells showing round cell nuclei, aclear, and slightly basophilic cytoplasm. The nuclei were mildly atypical, and mitotic figures were scarce. No capillary clusters or plexiform capillaries were observed. Hematoxylin and Eosin staining demonstrated typical oligodendroglioma, which could be classified as the WHO classification Grade II. Immunohistochemical staining of the cytoplasm was negative, except for focal positivity for glial fibrillary acidic protein (GFAP). A low proliferation index (<5%) was observed using Ki-67 immunohistochemistry [Figure 4].

The possibility of seeding was considered and brain, cervical, and MRI were performed for the brain and cervical and thoracic spinal cord. No pathologies were encountered [Figure 5]. Considering the current diagnosis of the patient, and that there were only sixty other cases in the literature, specialist

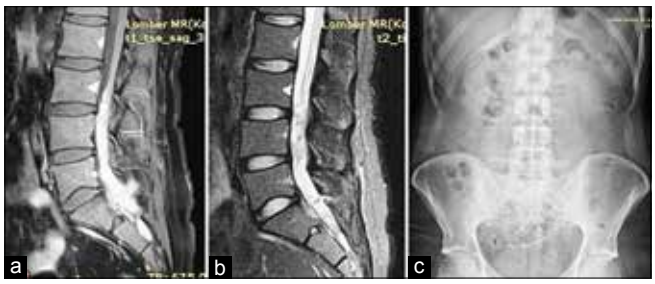


**Figure 1: (a) On the T1-weighted magnetic resonance imaging scan with contrast, a dense contrast-enhancing mass is seen spanning from the lower margin of L3 to S2. (b) In the T2-weighted magnetic resonance imaging scan without contrast, a nonhomogenous lesion is seen, including hypointense signal densities. (c) No distinct pathologies were observed on the anterior-posterior X-ray image**

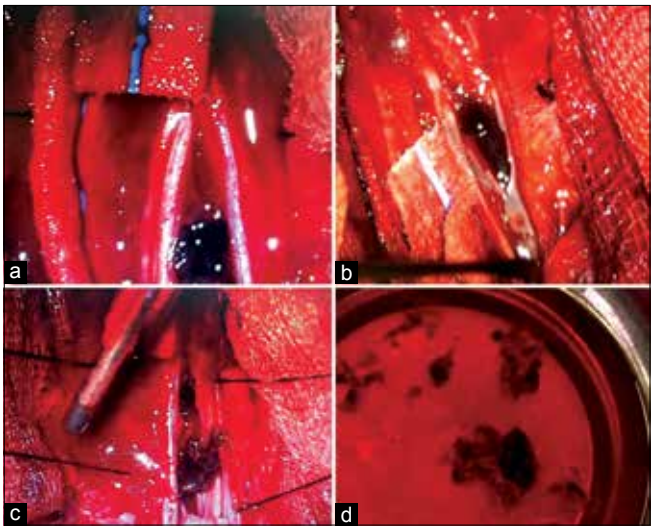


**Figure 2: (a) After opening the dura, hemorrhagic fragile tumor tissue was seen on the upper tumor margin. (b) As the dura opens distally, fragile tumor tissue can be observed coming away from the intradural space. (c) After the dura is opened and hung from the sides, gray-black-colored tumor tissue is visible between the cauda fibers. (d) An image is shown after the removal of tumor tissues**

**Table 1: Review of the literature of primary spinal cord oligodendroglioma[1-57]**

| Author | Age/sex | Symptom | Location | Histology/pathology | Treatment | Result |
|---|---|---|---|---|---|---|
| Kernohan *et al*., 1931[15] | | | | | | |
| Foerster and Gagel, 1934[16] | | | | | | |
| Oljenick, 1936[17] | 31/female | Weakness of left leg<br>Pain from hip to left knee<br>Weakness of both extremities | D7-D11 | M: Firm<br>Demarcation line, excised<br>*En bloc* (10 cm)<br>m: No calcium deposit | Total excision | Development? |
| Rasmussen *et al*., 1940[18] | | | | | | |
| Woods and Pimenta, 1944[19] | 43/female | Lower back pain and both sides sciatica pain | *Conus*<br>Cauda | M: Infiltrated<br>m: Malignant, no calcium deposit | Partial excision<br>RT | Died after 4.5 months |
| Russell and Bucy, 1949[20] | 31/male | Back muscle fasciculation<br>Deterioration and progressive weakness of legs<br>Lower extremity without RT<br>Pains in the pelvic cavity and the back | D8-D10 | M: Gelatinous pink wish<br>Grey infiltrating<br>m: No calcium deposit | Partial excision | Within 5 months |
| Love *et al*., 1951[21] | 44/male | Back pain | D8-D11 | m: No calcium deposit | Partial excision | |
| Padberg and Davıs, 1952[22] | 27/female | Back pain<br>First, weakness of the lower extremity and then numbness in the lower extremity<br>Hyperesthesia in the legs | C4-D1 | M: Firm, purple<br>Demarcated, removed<br>*En bloc* (9 cm) | Partial excision<br>RT no | 4.5 years of survival |
| Enestrom and Grontoft, 1957[25] | 37/female | Pain from lower back to hip<br>weakness of the lower extremity | D7 | M: Diffusely demarcated<br>m: No calcium deposit | Partial excision<br>RT no | 16 months without change |
| Kornyansky, 1959[26] | <16 | | | | Unspecified | |
| Amyes, 1960[27] | 26/male | Acute sciatic pain in the left<br>Sudden caudate syndrome | Phylum | M: Partly necrotic<br>Fresh and old hemorrhage | Total excision<br>RT | Development? |
| Coxe, 1961[28] | <15/ female | | Servico Thorax | Unspecified | | Development? |
| Klar and Henn, 1961[29] | 39/male | Upper abdominal pain<br>Progressive weakness<br>Starting as fasciculation in the left leg | D8-D11 | Unspecified | RT no | Died after 2 months |
| | 36/female | Pain | D10-L1<br>Extramedullary | Unspecified | RT no | Development? |
| Love and Rivers, 1962[30] | 36/male | Paresthesia in the left thumb and index, pain in the left shoulder<br>fasciculation in the left biceps | C4-C5 | M: Firm brownish-red<br>Encapsulated 3×2×1 cysts<br>m: No calcium deposit | RT no<br>Total excision | 31 years of survival |
| | 52/male | Back pain | D4-D5 | m: No calcium deposit | Bx<br>RT no | Died after 24 months |
| | 29/female | Sacral and two-sided sciatica pain<br>Weakness of the lower extremities<br>Numbness in the right leg | D5-D11 | M: Infiltrating cyst, syrinx<br>m: No calcium deposit | RT no<br>Bx | Died after 5 months |
| Shekhanov, 1964[31] | <16 | | | | Unspecified | |

*Contd...*

**Table 1: Contd...**

| Author | Age/sex | Symptom | Location | Histology/pathology | Treatment | Result |
|---|---|---|---|---|---|---|
| Slooff *et al*., 1964[32] | 45/female | Neck pain affecting right arm<br>Numbness in both hands<br>Hyperesthesia in both arms<br>Weakness of upper right extremity | C2-C5 | M: Firm, scarlet-red Demarcated, with extradural extension like a dumbbell neurofibroma<br>m: Malignant, no calcium deposit | Partial excision | Died after 21 days |
| | 40/male | Neck pain | C3-C7 | m: Malignant, no calcium deposit | Bx<br>RT | Died after 30 months |
| Backus, 1965[33] | Female | Pain | Cervical | | ? | |
| Broder, 1965[34] | 25/female | Weakness of both extremities | D6-D9 | | Partial excision | Died 6 days |
| Ortiz Gonzales *et al*., 1965[35] | 39/male | Weakness of the right leg | Phylum | M: Infiltrating calcified | Total excision | Change? |
| Nathoo and Halliday, 1967[36] | 45/female | Acute back pain two-sided Sciatica pain | Phylum | M: Firm fleshy Removed *en bloc* 5×2×2 | Total excision | Development? |
| Toso, 1967[37] | 8/male | Vomiting, headache, Kernig's sign | Cervical | No | no | Died after 3 months |
| O'Brien *et al*., 1968[38] | 16/male | Acute weakness of first the lower extremity then in the upper extremity | C2-L1 | | Bx<br>RT | Development? |
| | 8/female | Acute back pain | Entire Cord | M: Friable, infiltrating Hemorrhagic syrinx<br>m: No calcium deposit | RT<br>Bx | Died after 9 months |
| Pedersen, 1969[39] | 6/male | | D5 | | Partial excision RT? | |
| Garcia and Lemmi, 1970[40] | 16/female | Weakness of the left arm | C3-D3 | M: Gelatinous, orange Hemorrhagic<br>m: No calcium deposit | Partial excision RT | Deterioration within 9 months |
| Kernohan, 1972[42] | 18/female | Pain | L1-L3 *Conus* Cauda | M: Infiltrating<br>m: Malignant, no calcium deposit | Partial excision RT | ? |
| | 40/male | Pain | L2-L3 Phylum | m: No calcium deposit | RT Total excision | 22 years of survival |
| Hünig *et al*., 1974[43] | 16/male | | D11-L1 | | Partial excision RT | Died after 7 months |
| Maurice-Williams and Lucey, 1975[44] | 44/male | Hip pain<br>Femoral numbness<br>Progressive weakness | Lower Thoracic | M: Soft hemorrhagic<br>m: No calcium deposit | Bx<br>RT | Died after 5 years |
| Michel *et al*., 1975[45] | 13/male | Painless spasm in thoracolumbar Muscles<br>Meningeal syndrome | D10 | M: Gelatinous infiltrating<br>m: Calcium deposits | RT no<br>No surgery | Died after 23 months |
| Wöber and Jellinger, 1976[46] | 28/female | Lower back pain<br>Paresthesia in the right leg | D10-D11 | M: Vitreous white grayish Infiltrating syrinx<br>m: No calcium deposit | Partial excision RT no | Died after 2 months |
| Kordás *et al*., 1977[47] | <14 | | D12-L1 | Oligodendroglioma | Bx | Died |
| Ridsdale and Moseley, 1978[48] | 32/male | Headache, blurred vision | D12-L1 | Oligodendroglioma | Bx | Died |
| Fortuna *et al*., 1980[49] | 32/female | LBP, paraparesis | T10-T11 | Oligodendroglioma | Bx | NA |
| Guidetti *et al*., 1981[50] | NA/female | NA | NA | Oligodendroglioma | Partial excision | Died after 3 years |
| Alvisi *et al*., 1984[51] | | | | | | |
| Chen, 1988[52] | | | | | | |
| Pagni *et al*., 1991[53] | 13/male | Painful tetraparesis including kyphoscoliosis neck and shoulders | IM at C5-T2 | Oligodendroglioma | Partial excision | 2 year development |

*Contd...*

**Table 1: Contd...**

| Author | Age/sex | Symptom | Location | Histology/pathology | Treatment | Result |
|---|---|---|---|---|---|---|
| Wang *et al*., 1993[54] | 3/male | Scoliosis | Thoracolumbar | AO | Partial excision<br>RT | NA |
| Lunardi *et al*., 1993[55] | <16 | Spinal deformity | | Oligodendroglioma | Partial excision<br>RT | Average period of 5 years |
| Cristante and Hermann, 1994[56] | | | | | | |
| Constantini *et al*., 1996[57] | <3 | Motor regression, gait abnormalities | | AO | Surgery | Median follow-up 76 months |
| Nam *et al*., 1998[13] | 38/male | In the scoliosis lower right extremity, monoparesis | IM with syringomyelia along the T4-*Conus* medullaris | AO | Partial excision<br>RT | 50 months nonprogressive TM |
| Ushida *et al*., 1998[14] | 12/male | Scoliosis, legs hyperreflexia in legs with hyperesthesia | IM with syringomyelia at C7-T12 | Oligodendroglioma | Partial excision | Recurrent TM after 10 months |
| Aman *et al*., 2000[10] | | | | | | Second PR and RT |
| Gilmer-Hill *et al*., 2000[11] | 4/male | Limited, hypotony ataxic gait | Primary IM oligodendrogliom at C6-T1 with gliomatosis | Oligodendroglioma | Bx | CMT, recurrence in the temporal lobe after 10 months<br>4th PR and RT (AO)<br>Recurrence in the occipital lobe 4 months after RT<br>5th PR and CMT (AO)<br>Stable 7 months after operation |
| Fountas *et al*., 2005[9] | 16/female | Neurogenic bladder, left ankle weakness and sensory deficit of S1 nerve root on the left | IDEM with extension at T11-L2 | AO | Totally excision | 28 months, no recurrence of tumor |
| Gürkanlar *et al*., 2006[7] | 56/male | Pain in both legs | IDEM at L1-L2 with partial invasion of the *Conus* | Oligodendroglioma | Totally excision | NA |
| Ramirez *et al*., 2007[8] | 22/male | Paresthesia, lower back pain and Brown–Sequard syndrome | IM with hematomyelia at C5-C7 | AO | Partial excision<br>RT | Brain met after 2 years |
| Tobias *et al*., 2008[6] | 8/male<br>24/male | Neck pain, weakness of the arm | C3-T12<br>C1-T12 | Oligodendroglioma<br>Anaplastic oligodendroglioma | Partial excision<br>Partial excision<br>*TMZ* | Mean of 3.4 years (range, 1-12) after surgery |
| Guppy *et al*., 2009[5] | 30/female | Nausea, vomiting, and severe headaches | C3-C4 | Oligodendroglioma Grade II | Partial excision<br>*TMZ* | |
| Wang *et al*., 2011[4] | 18/female | Back pain, weakness in the lower extremity, hyperalgesia underneath | IM with syringomyelia at T8-T10 | AO | Partial excision<br>RT | TM recurrence after 8 months<br>2nd PR and TMZ<br>No recurrence 1 year after TMZ |
| Yuh *et al*., 2015[2] | 24/male | Weakness in the left leg radicular pain in both legs | T4-T8 IM bulk | Grade II Oligodendroglioma | Subtotal excision<br>RT | Increased after 4 years fixed residue |
| Moorthy *et al*., 2015[3] | 44/male | Weakness of both lower limbs urinary incontinence | D11-L2 | Oligodendroglioma Grade II | | |
| Tunthanathip and Oearsakul, 2016[1] | 46/male | Neck pain, weakness of the left arm, sensorial deficit between C5 and T1 | IM with syringomyelia at C3-T4 | Oligodendroglioma | Partial excision<br>RT | Recurrent after 1 year |

RT - Radiotherapy; NA - Not available; LBP - Low back pain; Bx - Biopsy; IM - Intramedullary; PR - Partial resection; AO - Anaplastic oligodendrogliomma; IDEM - Intradural extramedullary; CMT - Chemotherapy; TMZ - Temozolomide; TR - Total resection; M - Macroscopic; m - Microscopic

radiotherapists were consulted. A 45 Gy dose of RT was applied. The patient had no complaints during follow-up other than occasional lower back pain. The patient's early control lumbosacral MRI scan revealed that the tumor was resected and, apart from a small contrast-enhanced region in the vicinity of S2, typical postoperative changes were seen [Figure 6]. In the patient's 6-month and 15-month control MRIimages, apart from a contrast-enhanced S2 region with

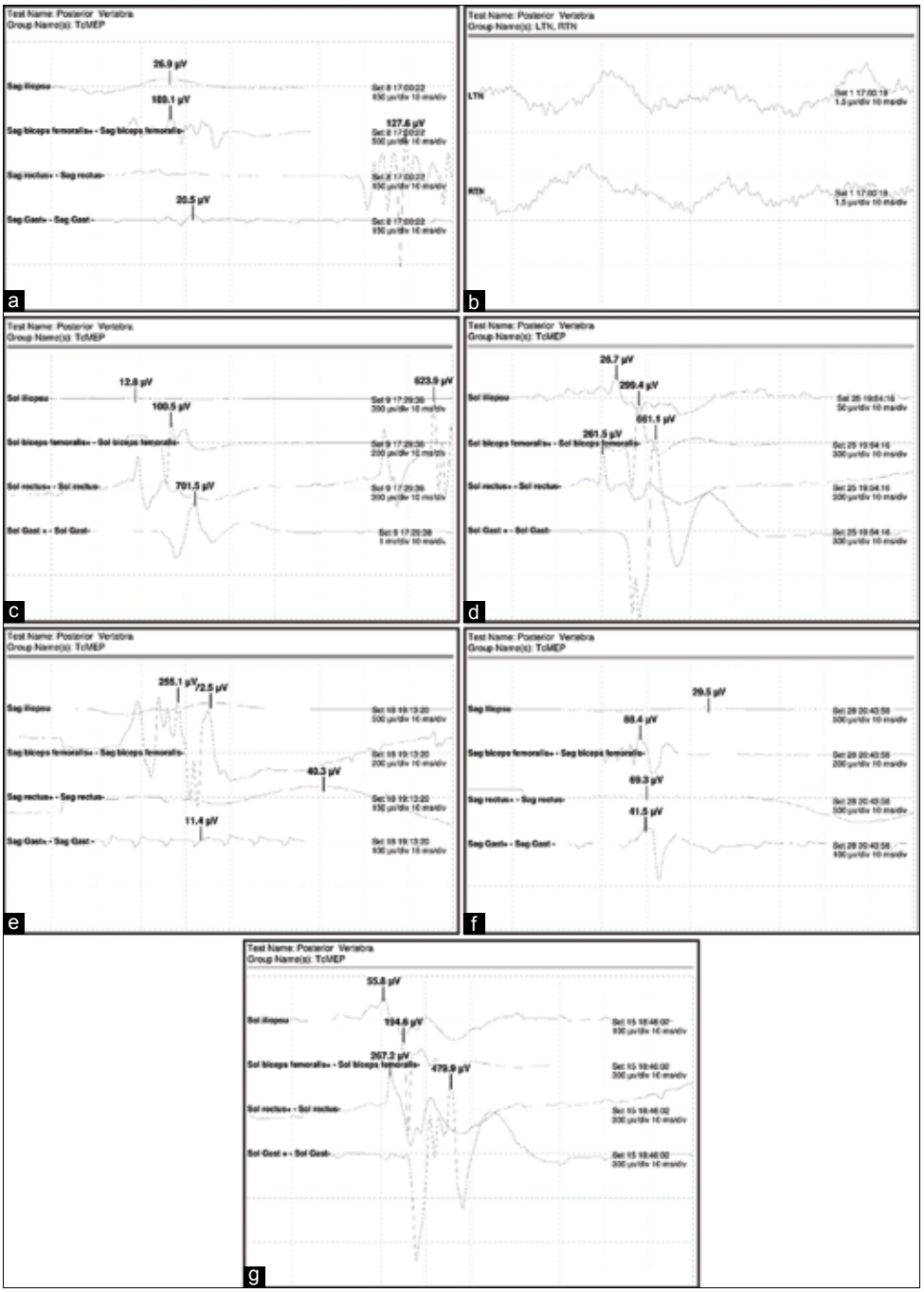


**Figure 3: (a) Basal somatosensory evoked potential (b) basal motor evoked potential (c) dural opening motor evoked potential, and (d) dural opening and motor evoked potential when the tumor area is exposed (e) motor evoked potential during tumor excision is shown (f) closing motor evoked potential is shown (g) exit somatosensory evoked potential and motor evoked potential values are shown**

changes in bone density, there were no radiological or clinical pathological findings [Figures 7 and 8].

## DISCUSSION

Spinal IM tumors account for between 2% and 8% of all central nervous system tumors, and approximately 15% of primary intradural spinal tumors.[2-12] PSO is a very rare type oftumor and constitutes only 2% of all spinal tumors.[3-13] The first case of PSO was reported by Kernohan *et al*.[14-18] Subsequently, Foerster and Gagel, Rasmussen *et al*., and Henschen reported a total of five cases of PSO among a large clinical series.[19-24] Russel and Bucy reported another case, and a few years later, Kernohan and Sayre reported three cases of PSO.[19-30] Fortuna raised the total number of reported cases of PSO to 36 and they reported that PSOs represented only 1.6% of central nervous system oligodendrogliomas.[30-43] With the latest publications, including a few mini-clinical

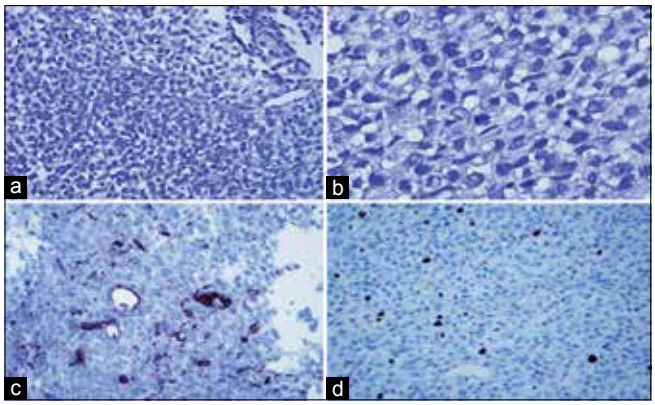

**Figure 4: (a and b) The typical circular and flattened appearance for an oligodendroglioma can be observed with uniform round nuclei and sparse or clear cytoplasm with perinuclear halos (a: ×200 and b: ×1000, respectively). (c) Glial fibrillary acidic protein staining showing positive focal immunoreactivity (×200). (d) Nuclear immuno-expression of Ki-67 is shown in a few neoplastic cells (×400)**

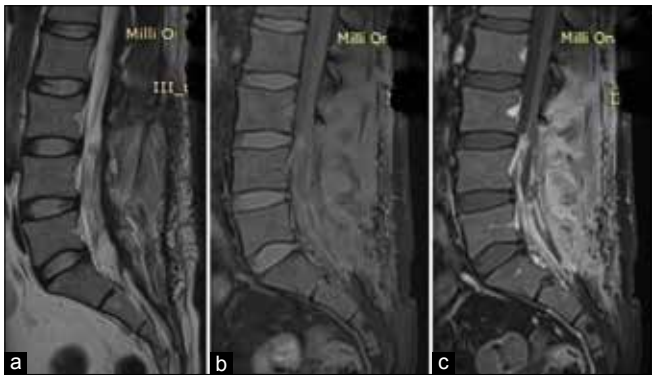

**Figure 6: (a) In the early postoperative period T2-weighted sagittal magnetic resonance imaging scan section without contrast, it can be seen that the tumor tissue has been removed. (b) In the postoperative early period T1-weighted sagittal magnetic resonance imaging scan section without contrast, it can be seen that the tumor tissue has been removed. (c) In the postoperative early period T1-weighted sagittal magnetic resonance imaging scan section with contrast, early postoperative changes can be seen**

series and cases, the total number of reported cases of PSO is 60.[1-14,43-57] Fortuna *et al*. found that PSO cases were equally distributed between both sexes and the literature to date indicates no significant sex predilection. Although IM spinal cord tumors appear more frequently in pediatric than adult patients, adult PSOs are more common than pediatric PSOs.[1-13,45-49] The clinical presentation of a PSO is similar to that of other IM spinal tumors, i.e., PSOs do not present with unique characteristic. PSO symptoms depend mostly on the anatomical site of the tumor and usually develop over a period spanning months to years. Symptom durations are shorter in pediatric patients than in adult patients.[1-13,47-52] Regional back pain and sensory disturbances are the most frequent complaints, while motor and sphincter deficits occur later.[1-10] More infrequent symptoms include kyphoscoliosis, raised intracranial pressure, and spastic tetraparesis due to

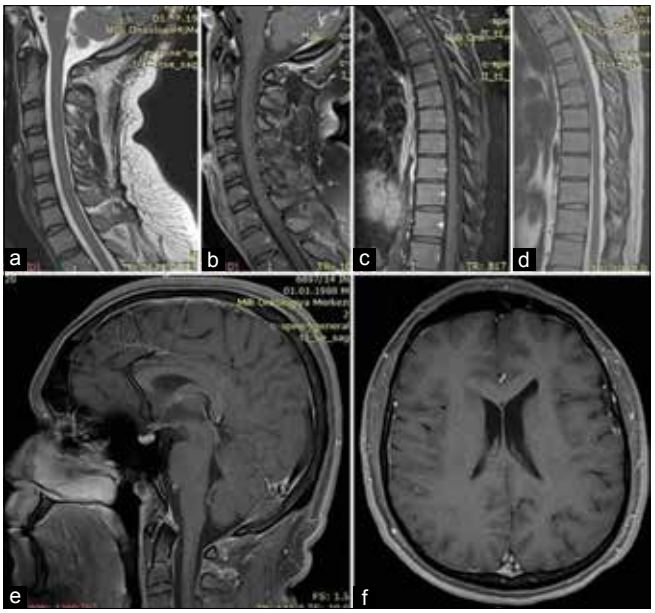

**Figure 5: No pathology was observed in the patient's seeding scans in the (a) T2-weighted sagittal cervical magnetic resonance imaging, (b) T1-weighted sagittal cervical magnetic resonance imaging, (c) T1-weighted sagittal thoracic magnetic resonance imaging; (d) T2-weighted sagittal thoracic magnetic resonance imaging (e) T1-weighted sagittal brain magnetic resonance imaging (f) T1-weighted axial brain magnetic resonance imaging**

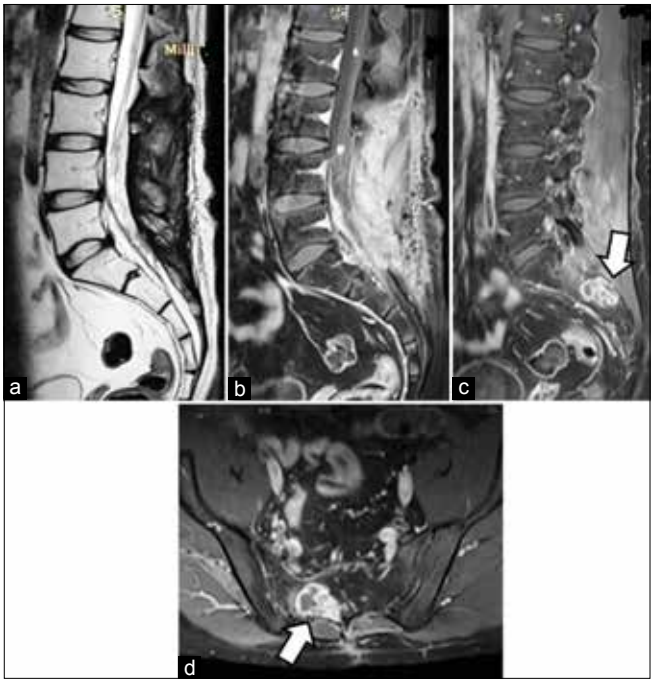

**Figure 7: In the postoperative 6-month (a) T2-weighted sagittal magnetic resonance imaging scan section without contrast, it can be seen that the tumor tissue has been removed (b) T1-weighted sagittal magnetic resonance imaging scan section with contrast, no residue contrast enhancement is observed in the intradural space (c) T1-weighted sagittal magnetic resonance imaging scan section with contrast, contrast enhancement is seen in the sacral region and bone (d) T1-weighted axial magnetic resonance imaging scan section with contrast, contrast enhancement is seen in the sacral region and bone**

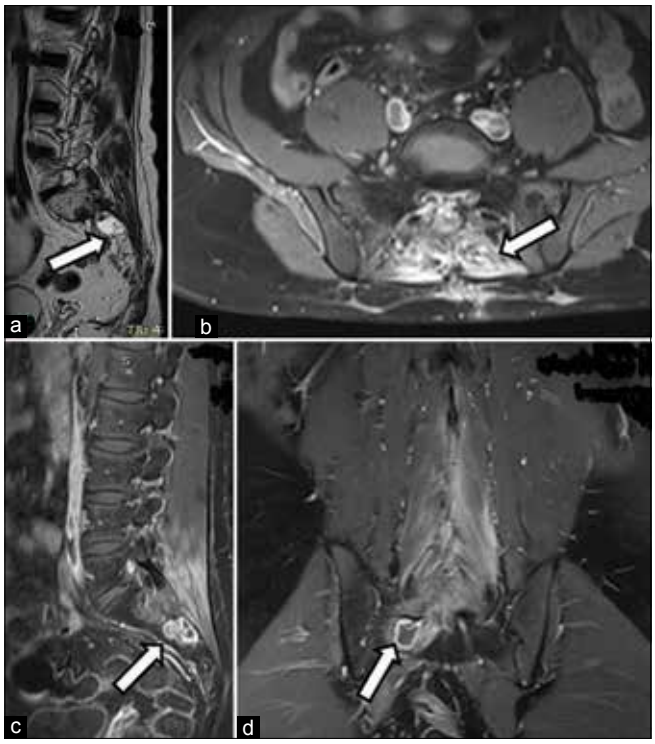


**Figure 8: In the postoperative 15-month (a) T1-weighted sagittal magnetic resonance imaging section without contrast, it can be seen that the tumor tissue sacrum (b) T1-weighted axial magnetic resonance imaging section with contrast, contrast enhancement is observed in the sacrum (c) T1-weighted sagittal magnetic resonance imaging section with contrast, contrast enhancement is seen in the sacral region and bone (d) T1-weighted coronal magnetic resonance imaging section with contrast, contrast enhancement is seen in the sacral region and bone**

osseous changes.[1-13,47-54] In the vast majority of reported cases of PSO, the tumor in the spine is between one and five segments in length. There are some cases in which the tumor occupied >10 consecutive vertebrae, and the frequency of these cases in young adolescents and pediatric patients has been reported. The most frequently reported anatomical locations for a PSOs are the cervical and thoracic regions. Only a few cases with involvement inferior to the conusmedullaris and sacrum have been reported, which including pediatric and relapse cases.[1-13,45-52] The case present here of a young adult patient with a primary diagnosis of a tumor reaching the sacrum is rare. Contrast MRI is the gold standard for the diagnosis of PSOs and appropriate surgical planning. Usually, heterogeneous hypointense or isointense lesions are observed in T1-weighted images, and a hyperintense lesion is observed in T2-weighted images.[1-11,48-56] The tumor margins are usually poorly demarcated, whereas in low-grade lesions, the tumor periphery may be well defined.[1-14] PSOs demonstrate mild to moderate nonhomogenous spotty enhancement. Hypointense areas may be present due to intramural bleeding foci and hemosiderin deposition. Cystic components or cystic necrotic areas may occasionally be observed, particularly in cases of high-grade PSO. The brain and entire spinal axis should be scanned due to the tendency of PSOs to spread through the cerebrospinal fluid.[1-13,45-54] The use of electrophysiological monitoring in surgical procedures minimizes neuronal tissue damage and maximizes tumor tissue removal. A laminectomy should be performed to cover the lower and upper tumor margins and intraoperative ultrasound is essential in identifying the tumor after opening the dura in the midline. The midline may be difficult to find sometimes owing to the rotation of the spinal cord. Moreover, the location of the posterior median sulcus can be determined approximately by observing the dorsal root entrance zones on both sides, or by observing the very small vessels emerging from the midline. A midline myelotomy should begin where the cord is the widest to reveal the entire tumor. Furthermore, the vessels intercrossing the midline of the cord can be safely coagulated and. After defining the tumor margins, it is important to identify the normal cord and tumor margins. A total excision of the tumor should be performed for all cases; however, in most cases, this is very difficult.[1-14] Pediatric patients may specifically require a laminoplasty instead of a multilevel laminectomy or the addition of lateral mass screws or rods for cervical or cervicothoracic tumors and the application of screws and rods to the lower levels or pedicles. An early postoperative MRI is important for identifying the tumor or postoperative hematomas.[1-13,47-52]

PSOs are usually solid mass tumors. They are yellow-gray, pink-gray or, less frequently, even reddish. Occasionally, the center of a PSO may show necrosis, cystic degeneration, and calcification at an average rate of 30%.[1-14] Microscopic analyses reveal that PSOs are typically composed of hyperchromatic neoplastic cells with small spherical nuclei and mitotic figures depending on the tumor's degree of differentiation. Immunohistochemical analysis of PSOs reveal no or mild reactivity for GFAP because oligodendrocytes possess no cytoplasmic intermediate filaments.[1-13,46-57] Using fluorescence *in situ* hybridization, an observation of 1p and 19p deletions in PSOs can better indicate response of the PSO to treatment. The use of adjuvant treatment in patients undergoing surgery for PSOs remains controversial. RT has been associated with the development of postradiation myelopathy and/or radiation-induced vertebral column deformities.[1-8,49-57] These adverse events are more frequent and more severe among pediatric patients.[1-14] Therefore, the decision to administer postresection RT should be determined carefully and individually for each patient, considering several parameters such as the patient's age, neurological status, tumor grade, tumor location, the extent of resection, and the genetic and histological characteristics of the tumor. Authors have reported several years of postoperative survival;[1-14]

however, in their cases, the prescription of RT and CMT failed to prevent tumor dissemination. Therefore, the use of post-resection RT should be limited to adults or older adolescent cases with a subtotal resection of high-grade tumors, and the RT doses should not exceed 40–50 Gy. CT with or without RT is promising. Nonetheless, despite all these treatments, the average survival for a patient with a PSO is 28.6 months.[1-14,48-57]

## Financial support and sponsorship

Nil.

## Conflicts of interest

There are no conflicts of interest.

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