Spinal extradural angiolipoma: report of two cases and review of the literature

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Abstract Spinal angiolipomas are benign uncommon neoplasm composed of mature lipocytes admixed with abnormal blood vessels. They account for only 0.04–1.2% of all spinal tumors. We report two cases of lumbar extradural angiolipoma and review previously reported cases. We found 118 cases of spinal epidural angiolipoma (70 females and 48 males; age range 1.5–85 years, mean 44.03) spanning from 1890 to 2006. Prior to diagnosis 40.6% of the patients had weakness of the lower limbs. The interval between the initial symptoms and tumor diagnosis ranged from 1 day to 17 years (mean 20.2 months). Except for four cases diagnosed at autopsy, 109 patients underwent surgery and gross-total resection was performed in 79 cases (72.4%). Spinal angiolipomas are tumors containing angiomatous and lipomatous tissue, predominantly located in the mid-thoracic region. All angiolipomas show iso- or hyperintensity on T1-weighted images and hyperintensity on T2-weighted images and most lesions enhance with gadolinium administration. The treatment for spinal extradural angiolipomas is total surgical resection and no adjuvant therapy should be administered.

Keywords Angiolipoma · MR imaging · Spinal cord · Spinal epidural tumor · Spinal cord compression

Introduction

Spinal angiolipomas are benign tumors composed of both mature fatty tissue and abnormal vascular elements that represent a distinct clinical and pathological entity. These lesions are most commonly found in the subcutaneous tissue of the trunk and extremities, but other sites have been reported as well. They account for 0.04–1.2% of all spinal axis tumors, and are predominantly found in the epidural space, where they represent 2–3% of spinal tumors. Intracranial angiolipomas are extremely rare (4). It is not clear from the literature if the lesions originate in the epidural space and “infiltrate” the bone, if they originate in the bone and spread to the epidural space, or if they arise in both compartments simultaneously [72].

A review of the literature since 1892 to the present day revealed 123 cases of spinal angiolipomas (118 extradural and 5 intradural). The purpose of this paper is to report our two cases of SAL and review the pertinent literature.

Case reports

Case 1

A 16-year-old male with a 6-month history of low back pain, and worsening of the symptoms in the weeks prior to diagnosis presented to the study. The pain radiated down the posterior aspects of both legs, and increased with physical movement and walking. A neurological examination revealed spasm of the lumbar paravertebral muscles, bilateral Lasègue signs at 60°, and absence of the Achilles reflexes.

Noncontrast and gadolinium-enhanced MR studies of the lumbar spine showed an extradural lesion at L5–S1

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Fig. 1 Preoperative sagittal MRI T1-weighted image: **a** (TR 720, TE 80); **b** (TR 462, TE 80) showing a mixed epidural mass at L5–S1 level

level. On T1-weighted images the lesion was slightly inhomogeneous (Fig. 1). The T2 signal was heterogeneously hypointense.

The patient was submitted to a L5–S1 laminectomy with exposure of the dura and bilateral sacral roots. A 3×2 cm high vascularized mass was found pushing the duramater, and was totally excised with preservation of the roots. The patient was asymptomatic at 12-month follow-up.

Microscopic examination revealed mature adipose tissue and abnormal vascular channels with blood vessels of variable caliber and wall thickness which mostly filled with erythrocytes.

Case 2

A 45-year-old woman with numbness in both feet, leg weakness of 6 months which commenced during the fifth month of gestation complained of a sudden increased numbness and weakness in the right leg and aggravated low-back pain on the day before admission.

Examination revealed that she was obese, bilateral Lasègue sign at 30° , bilateral absence of the Achilles tendon reflex and perineal hypoesthesia.

MRI showed a posterior epidural mass extending from L5 to S1, predominantly on the right side (3×1.5 cm). The lesion was slightly inhomogeneous epidural mass, which is moderately hyperintense relative to spinal cord in T1 and T2 weighted images. Disc hernia was initially diagnosed, hence no contrast was administered. The L5–S1 disc showed a mild protrusion (Fig. 2). The patient was submitted to a L5–S1 foraminotomies, a slightly protruded L5–S1 disc and a soft brown extradural mass was observed

Fig. 2 Preoperative sagittal MRI T1-weighted image (TR 560, TE 14) (**a**), T2-weighted image (TR 400, TE 15); (**b**) showing an L5–S1 extradural slightly inhomogeneous epidural mass (arrows). Mild posterior protrusion L5–S1 disc

compressing the L5 and S1 roots; the operative field was enlarged, and the mass was excised from the dorsal dura without difficulty. The postoperative course was uneventful and at 6-month follow-up examination the patient had perineal hypoesthesia.

Histological examination of the tumor showed mature adipose tissue interlaced with numerous blood vessels ranging from distended capillaries, venules and blood-filled lakes to small arteries, no mitotic figures were observed, and angiolipoma was diagnosed (Fig. 3).

Results

All reported cases of spinal angiolipoma reported between 1890 and 2006 were reviewed; only cases in which authors

Fig. 3 The lesion contained mature adipocytes and small-medium caliber blood vessels indicative of angiolipoma (H&E $\times 80$) (Case 1)

Table 1 Summary of the previous reported cases of spinal epidural angiolipomas

Author	Year	Age/sex	First symptom	Duration (months)	Level	Axial location	Treatment	Surgical result
Berenbruch [12]	1890	16/M	Paraparesis	2.5	Thoracic	Not available	Surgery	Dead-necropsy
Liebscher [43]	1901	51/F	Numbness	54	T7–T8	Anterior	No surgery	Necropsy
Frazier and Allen [24]	1918	55/F	?	?	Thoracic	Unknown	Unknown	Unknown
Balado and Morea [9]	1928	20/F	Paraparesis	16	Midthoracic	Unknown	No surgery	Necropsy
Kasper and Cowan [36]	1929	6/M	Bleeding to lumbar puncture	4 d	C2–C8 and L3–S3	Unknown	No surgery	Necropsy
Petit-Dutaillis and Christoph [67]	1931	43/F	Paraplegic	12	T6–T10	Unknown	Surgery	Recovery
Ehni and Love [20]	1945	30/F	Paraplegic	12	T6–T8	Posterior	Surgery	Recovery
Ehni and Love [20]	1945	67/M	Paraparesis	12	T8–T9	Posterior	Surgery	Recovery
Bucy and Ritchey [15]	1947	33/M	Paraparesis	2.5	C6–T5	Posterolateral	Surgery + RT	Improved
Taylor et al. [90]	1951	51/F	Paraparesis	108	T3–T6	Posterior	Surgery	Improved
Taylor et al. [90]	1951	44/F	Paraparesis	24	T10–L1	Post-infiltrating	Surgery	Improved
Moscattelli and Merigliano [55]	1958	51/M	?	?	T4–T7	Posterior	Unknown	Unknown
Maier [49]	1962	1.5/F	Paraparesis	1	T7	Anterior-infiltrating	Surgery	Recovery
Gonzalez-Crussi et al. [28]	1966	20/F	LBP	36	L1–L4	Anterior-infiltrating	Surgery + RT	Recovery
Gagliardi and Gambacorta [25]	1968	30/M	Paraparesis	?	T3–T4	Posterior	Unknown	Unknown
Lo Re and Michelacci [47]	1969	16/M	Congenital	184	Cauda	Posterior	Surgery	Recovery
Lo Re and Michelacci [47]	1969	35/F	?	?	Lumbar	Posterior	Surgery	Recovery
Warot and Petit [95]	1969	57/F	Paraparesis	?	T4–T6	Posterior	Unknown	Unknown
Pearson et al. [66]	1970	17/F	TBP	1	T2–T5	Anterior-infiltrating	Surgery	Improved
Pearson et al. [66]	1970	22/M	TBP	12	T3–T9	Anterior-infiltrating	Surgery	Improved
Pearson et al. [66]	1970	44/F	Numbness	36	T7–T10	Posterior	Surgery	Recovery
Henry et al. [34]	1971	63/M	Paraparesis	4	Cervicothoracic	Posterior	No surgery	Dead for associated spinal glioblastoma. Necropsy
Giuffrè [27]	1971	36/F	?	?	T4–T8	Unknown	Unknown	Unknown
Bender et al. [11]	1974	50/F	Paraparesis	54	T6–T8	Posterior	Surgery	Recovery (12 years later recurrence)
Bender et al. [11]	1974	58/M	Paraparesis	5	T7–T10	Posterior	Surgery	Improved
Scanarini and Carteri [81]	1974	57/F	Paraplegic	12	T7	Posterior	Surgery	Recovery
Obrador et al. [57]	1977	43/F	Sciatalgia	6	T7–T9	Posterior	Surgery	Recovery
Occhiogrosso & Vailati [58]	1977	34/F	Numbness	12	T6–T8	Posterior	Surgery	Recovery
Cull et al. [18]	1978	50/F	Paraparesis	204	T8–T9	Posterior	Surgery	Recovery
Cull et al. [18]	1978	45/F	Paraparesis	?	T6–T8	Posterior	Surgery	Improved

Table 1 continued

Author	Year	Age/sex	First symptom	Duration (months)	Level	Axial location	Treatment	Surgical result
Shuangshoti and Hongsgabhabhas [85]	1979	45/M	?	?	T10–T11	Posterior	Surgery	Recovery
Schiffer et al. [84]	1980	48/F	Numbness	36	T10–L1	Anterior-infiltrating	Surgery	Recovery
Goyal [29]	1980	67/F	Paraparesis	2	?–T10	Posterolateral	Surgery	Dead
Miki et al. [53]	1981	46/F	Numbness	24	T3–T5	Posterior	Surgery	Recovery
Padovani et al. [61]	1982	50/M	Paraparesis	12	T4–T6	Posterolateral	Surgery	Recovery
Padovani et al. [61]	1982	52/M	Paraparesis	5	T4–T6	Posterior	Surgery	Improved
Hanakita and Koyama [33]	1982	42/M	Gait disturbance	12	T4–T5	Posterior	Surgery	Recovery
Pasquier et al. [65]	1984	48/F	?	?	T8–T10	Posterior	Surgery	Improved
Butti et al. [16]	1984	44/M	Paresthesia right arm	2	C6–C7	Posterior	Surgery	Recovery
Butti et al. [16]	1984	50/F	Numbness in legs	4	L4–L5	Posterior	Surgery	Recovery
Bardosi et al. [10]	1985	46/M	LBP	24	T11–L2	Posterior	Surgery	Recovery
Von Hanwehr et al. [94]	1985	35/M	Numbness of right foot	12	T6	Anterior-infiltrating	Surgery	Recovery
Haddad et al. [32]	1986	34/M	Numbness of legs	18	T5–T6	Posterior-infiltrating	Surgery	Improved
Haddad et al. [32]	1986	22/M	LTP	30	T7–T8	Posterior	Surgery	Recovery
Griebel et al. [30]	1986	53/F	Paraparesis	24	T5–T6	Posterior	Surgery	Recovery
Nishiura et al. [56]	1986	42/M	Numbness in legs	5	T3–T6	Posterior	Surgery	Improved
Nishiura et al. [56]	1986	24/M	LBP	36	L5–S1	Anterolateral	Surgery	Recovery
Nishiura et al. [56]	1986	45/M	Numbness in legs	6	T3–T6	Posterior	Surgery	Improved
Matsushima et al. [51]	1987	41/F	Paraparesis	2	T9–T10	Posterior	Surgery	Recovery
Rivkind et al. [75]	1986	52 M	Paraparesis	0.5	T7	Anterolateral	Surgery	Recovery
Poon et al. [70]	1988	65/F	TBP	6	T8–T9	Posterior	Surgery	Recovery
Parizel [64]	1989	37/F	?	?	T4–T8	Unknown	Surgery	Recovery
Anson et al. [5]	1990	58/F	TBP	3	T2–T6	Posterolateral	Surgery + RT	Recovery
Anson et al. [5]	1990	65/F	Paraparesis	2 d	T1–T8	Posterior	Surgery	Unchanged
Kuroda et al. [41]	1990	73/F	Numbness in left leg	?	T4	Posterior-infiltrating	Surgery	Improved
Weill et al. [96]	1991	46/F	Paraparesis	12	T7–T10	Posterior	Surgery	Recovery
Mascalchi et al. [50]	1991	42/F	Numbness of left toe	12	T5–T6	Posterior	Surgery	Recovery
Rubin et al. [77]	1992	58/M	Paraparesis	24	T8–T10	Posterior	Surgery	Improved
Stranjalis et al. [88]	1992	68/F	Paraparesis	12	T5–T6	Posterior-infiltrating	Surgery	Improved
Pagni and Canavero [62]	1992	56/F	LBP	144	L3	Anterior	Surgery	Recovery
Pagni and Canavero [62]	1992	59/F	LBP	224	L4–L5	Anterior	Surgery	Recovery
Mimata [54]	1992	60/M	?	48 m	T5–T8	Unknown	Surgery	Recovery
Yamashita et al. [99]	1993	57/M	Gait disturbance	24	T3–T9	Posterior	Surgery	Recovery

Table 1 continued

Author	Year	Age/sex	First symptom	Duration (months)	Level	Axial location	Treatment	Surgical result
Shibata [83]	1993	38/F	Paraparesis	6	T4–T6	Unknown	Surgery	Recovery
Preul et al. [72]	1993	45/F	Numbness in legs	24	T7–T11	Posterior	Surge	Recovery
Preul et al. [72]	1993	58/M	TBP	?	T3	Posterior-infiltrating	Surgery	Unchanged
Michilli et al. [52]	1993	12/M	LBP	1 s	T5–T10	Posterior	Surgery	Recovery
Sakaki et al. [79]	1993	67/M	Bilateral sciatica	60	T12–L1	Lateral	Surgery	Recovery
Fernandez et al. [22]	1994	14/F	Paraplegic	Hours	T5	Posterior	Surgery	Improved
Fernandez et al. [22]	1994	28/M	Paraplegic	Hours	C7–T4	Posterior	Surgery	Unchanged
Turanaz et al. [92]	1994	25/M	Paraparesis	1	T6–T8	Anterior-infiltrating	Surgery	Improved
Balbo [9]	1995	41/M	Paraparesis	4	T6–T7	Posterior	Surgery	Recovery
Bouramas [14]	1995	27/F	Numbness in legs	2	T2–T8	Posterior	Surgery	Recovery
O'Donovan [59]	1996	54/M	TBP	1	T3–T9	Posterior	Surgery	Recovery
Trabulo et al. [91]	1996	26/F	TBP	8	T2–T9	Posterior	Surgery	Recovery
Trabulo et al. [91]	1996	72/M	Paraparesis	24	T6	Posterior-infiltrating	Surgery	Recovery
Provenzale & McLendon [73]	1996	38/F	LBP	36	Lumbar	Posterior	Surgery	Unknown
Provenzale & McLendon [73]	1996	61/F	Paraparesis	24	Thoracic (2 tumors)	Posterior	Surgery	Unknown
Provenzale & McLendon [73]	1996	42/F	TBP	24	Thoracic	Posterior	Surgery	Unknown
Krishnan et al. [39]	1996	55/F	Paraparesis	24	T6–T9	Posterior	Surgery	Recovery
Boockvar et al. [13]	1997	34/F	TBP	5	T3–T9	Posterior	Surgery	Recovery
Shuangshoti & Lerdlum [86]	1997	21/M	Paraparesis	2	T1–T2	Posterior + osteochondroma	Surgery	Recovery
Sakaïda [78]	1998	72/M	Numbness in legs	4	T3–T5	Anterior-infiltrating	Surgery	Recovery
Labram et al. [42]	1999	40/F	TBP	6	C6–T3	Posterolateral	Surgery	Recovery
Labram et al. [42]	1999	68/F	TBP	6	T5–T10	Posterior	Surgery	Recovery
Labram et al. [42]	1999	17/M	TBP	12 h	C5–T3	Circumferential	Surgery	Recovery
Turgut [93]	1999	54/F	Paraparesis	5	T4–T9	Posterior	Surgery	Recovery
Kujas [40]	1999	67/F	Paraparesis	6	T6	Anterior	Surgery	Worsened
Öge et al. [60]	1999	72/M	Paraparesis	4 d	T2–T5	Posterior	Surgery	Recovery
El Abbadi [21]	1999	38	Paraparesis	?	T10–T11	Posterior	Surgery	Recovery
Bailey et al. [7]	2000	44/F	Numbness in legs	6	T3–T8	Posterior	Surgery	Recovery
Al-Anazi et al. [2]	2000	38/F	Numbness of both legs	?	T5–T9	Posterior	Surgery	Recovery
Andaluz et al. [4]	2000	24/F	Paraparesis	3	T7–T10	Posterior	Surgery + embolization	Recovery
Andaluz et al. [4]	2000	39/F	Numbness in legs	6	T6–T8	Posterior	Surgery	Recovery
Andaluz et al. [4]	2000	59/F	Paraparesis	?	T4–T6	Posterior	Surgery	Recovery

Table 1 continued

Author	Year	Age/sex	First symptom	Duration (months)	Level	Axial location	Treatment	Surgical result
Andaluz et al. [4]	2000	69/M	Paraparesis	8	T3–T4	Posterior	Surgery	Recovery
Akhaddar et al. [4]	2000	47/F	Numbness in legs	36	T4–T6	Posterior	Surgery	Recovery
Akhaddar et al. [4]	2000	46/F	Paraparesis	1.5	T7–T9	Posterior	Surgery	Recovery
Akhaddar et al. [4]	2000	38/F	Numbness in legs	5	T3–T5	Posterior	Surgery	Recovery
Akhaddar et al. [4]	2000	46/F	Paraparesis	98	T3–T4	Posterior	Surgery	Recovery
Akhaddar et al. [4]	2000	12/F	Paraparesis	4	T7–T8	Posterior	Surgery	Improved
Akhaddar et al. [4]	2000	64/F	Sciatalgia	2	T2–T4	Posterolateral	Surgery	Recovery
Akhaddar et al. [4]	2000	58/M	Numbness in legs	7	T3–T6	Posterior	Surgery	Recovery
Akhaddar et al. [4]	2000	47/M	TBP	6	T3–T6	Posterior	Surgery	Improved
Amilashi et al. [3]	2001	36/F	LBP	1.5	T2–T6	Posterior	Surgery	Improved
Fourney et al. [23]	2001	46/F	Numbness in legs	5	T6–T8	Posterior	Surgery	Recovery
Gelabert et al. [26]	2002	4/M	Paraparesis	2 d	T2–T5	Posterior	Surgery	Recovery
Pinto et al. [69]	2002	85/M	Sciatica	1 d	L1–2L	Posterior	Surgery	Improved
Samdani et al. [80]	2004	49/F	LBP	3	T6–T8	Posterior	Surgery	Recovery
Aversa do Souto et al. [6]	2003	46/F	LBP	120	L4–L5	Anterior	Surgery	Recovery
Rocchi et al. [76]	2004	60/M	Sciatalgia	24	L3–L4	Anterior	Surgery	Recovery
Rocchi et al. [76]	2004	54/F	Sciatalgia	12	L3	Anterior	Surgery	Recovery
Rabin et al. [74]	2004	47/M	Numbness in legs	6	T9	Anterior-infiltrating	Embolization + Surgery	Recovery
Petrella et al. [68]	2005	16/M	LBP	12 h	T4–T8	Posterior	Surgery + abscess	Recovery
Cubillos et al. [17]	2005	40/F	Numbness in legs	1	T4–T7	Posterior	Surgery	Recovery
Konya et al. [38]	2006	60/F	LBP	6	L5	Posterolateral	Surgery	Recovery
Dogan et al. [19]	2006	50/F	Sciatalgia	24	L4–L5	Posterior	Surgery	Recovery
Dogan et al. [19]	2006	36/M	LBP	8	L2	Posterior	Surgery	Recovery

d days, F female, LBP low back pain, M male, RT radiotherapy, TBP thoracic back pain

reported details about the relevant topics were included for analysis (see Table 1). For example, a review of 48 angiolipoma cases, 8 of which were surprisingly neither described in other series nor included in the bibliography, were omitted [54].

Age and sex. Most angiolipomas occurred in adults with an age range of 1.5 to 85 years (mean 44.03 ± 5.9), and peak incidence was 46 (7 cases). A female predominance (70 females, 48 males) was observed, and mean age was 43.6 ± 5.1 for males, and 45.1 ± 6.1 for females.

Revealing symptoms. Initial symptoms were progressive or sudden weakness in 48 patients (40.6%), thoracic or lumbar pain in 27 cases (22.8%), alterations in sensation (numbness) in 24 (20.3%), sciatica uni or bilateral in 6 (5%), gait disturbance in 2 (1.7%), 1 bleeding to lumbar puncture [36], and 1 congenital [47]. In nine cases initial symptoms were not reported. Eight patients with a relapsing clinical course presented paraparesis during pregnancy or puerperium [14]. In five cases the patients were overweight and five patients were administered steroids.

The interval between the initial symptoms and tumor diagnosis ranged from 1 day to 17 years (mean interval 20.2 ± 9.6 months); in 16 cases (13.5%) the duration was not mentioned or not specified. The duration of symptoms was longer in patients with lumbar pain ($53.1 \pm$ weeks), whereas the shortest duration of symptoms was observed in four cases involving sudden motor deficit (<1 week) [5, 22, 26].

Location. Tumors were located as follows: two in the cervical region [16, 36], 93 in the thoracic region, 12 in the lumbar, 1 in the sacral [47], 5 in the cervico-thoracic region, 3 in thoracic-lumbar area, and 3 in the lumbo-sacral region. Of the 108 cases where the location was specified, 14 comprised one vertebral segment; 28 two segments, 22 three segments, 19 four segments, and 25 more than four segments.

Size. As for tumor size, data were provided for 21 cases, i.e., 10 cases described three dimensions, 5 two dimensions were reported, and only one dimension was specified in 6.

Biochemical studies. Cerebrospinal fluid samples were analyzed in 11 cases with slight protein concentrations (range 0.70–1.41 mg/dl), mean: 1.0 mg/dl, being observed in 5 cases.

Neurophysiological study. Only two neurophysiological studies were reported: the case reported by Bailey et al. [7] where the lower limb somatosensory evoked responses were normal, and the case published by Nishiura et al. [56] where the electromyogram showed a neurogenic pattern in the paravertebral muscles from T8 to T10 bilaterally.

Radiology. X-rays of the spine essentially show no bone abnormalities. The most frequent alterations were erosion of the walls of the vertebrae (bodies and pedicles) with enlargement of the interpeduncular distances [1, 57, 75]

primarily in infiltrated tumors. Other cases were associated to scoliosis [10, 56, 90], osteoporosis [84] or Klippel-Feil syndrome [15].

Myelography was performed in 43 cases, and all showed a characteristic posterior extradural spinal mass with partial or complete obstruction. In 20 cases the myelography was assisted with TC.

Angiography. Selective spinal angiography was performed in only two cases with prominent vascularity in the lateral aspect of the T9 vertebral body in one case [74], and in the T6 in the other (4). In both cases the tumors were supplied by a branch of the intercostals artery.

Computed tomography was performed in 34 cases (21 cases CT-myelography). CT scan demonstrated the presence of a space-occupying lesion in 21 cases as well as 6 CTs were normal, 7 were unspecified, and in a further 2 cases disc hernia was observed [6, 56]. Moreover, CT revealed tumor calcification in two patients [1, 73], trabeculation of the vertebral body in two cases [90, 93], erosion of the vertebral body and pedicle in another [1], a paravertebral thoracic component in two cases [1, 90] and association with vertebral hemangioma in other [1]. The tumor was hypo dense with the cord in 16 patients (between -20 to -72 HU), isodense in three patients [41, 52, 90], slight hyperdensity in three cases [63, 74, 77], and heterogeneous in only one case [1] where a reference to density was available. The epidural tumor did not show contrast enhancement in one case [41], demonstrated minimal contrast enhancement in two patients [52, 93] and sharp contrast gain in three [57, 63, 76].

MRI was performed in 47 cases (since 1988 onwards), but sufficient data were provided in only 31 cases. In the T1-weighted sequence, the tumor was as isointense as the medular conus in 7 cases, 18 were hyperintense, 5 were hypointense, and one was heterogeneous [80]. In the T2-weighted sequence, the tumor was hyperintense compared to CSF in 25 cases; two were isointense [75, 93], two hypointense [1, 17] and two mixed signal [74, 95]. After Gd injection, 13 cases presented remarkably homogeneous enhancement, 8 were heterogeneous, and in the remaining 8 cases low enhancement. Disc hernia was observed in one case [6], infiltration of the vertebral body and/or extending to the pedicle in three cases [4, 40, 75], and a paravertebral thoracic component in one case [78].

Treatment. Except for 4 cases diagnosed at autopsy, 109 patients underwent surgery and gross-total resection was carried out in 79 cases (72.4%), subtotal resection in 6 (5.5%), biopsy sampling in 1 case [95], but no mention of the extent of removal in 22 (20.1%), and no mention of surgery in 5 cases. Rabin et al. [75] performed preoperative embolization prior to T9 corpectomy and resection. Finally, radiotherapy was undertaken in only three cases [5, 15, 28].

Tumor recurrence following surgery is rare, and only two cases (1.8%) were reported: one after gross-total removal [11] and the other, an infiltrating tumor following subtotal resection [40].

Pathological features. Angiolipoma was diagnosed in all but five cases [67, 78, 93], and the most common feature was a lesion with two distinct components: mature adipocytes with peripheral, sometimes indented nuclei, and a single lipid droplet intermingled with abnormal blood vessels of varying diameter. In the case reported by Fournay et al. [23] mild atypia was observed in the endothelial cells. In two cases, the angiolipoma was associated with another tumor, osteochondroma [86], and glioblastoma [34]. Immunohistochemical assay was performed in a few cases [60, 79] and a low proliferation rate was found on Ki-67 [22, 68]. Samdami et al. [80] observed a positive stain for CD31, Factor XIIIa, and Factor VIII, and negative for Glial Fibrillar Acid Protein.

Outcome. The postoperative course was diverse ranging from 3 months to 10 years. Overall, there were two deaths (1.8%) in patients with non-infiltrating angiolipomas [11, 29] and none in the infiltrating subgroup. Though the results of surgery are difficult to assess due to the variations in terminology in the literature, most patients were reported to have improved postoperatively.

Discussion

Angiolipomas are benign tumors which usually appear as painful subcutaneous nodules, particularly in the forearm, trunk, or neck but occur rarely in the spinal canal [41]. Spinal angiolipomas account for 0.14–1.2% of all spinal tumors, about 2–3% of extradural spinal tumors, and 16–35% of spinal lipomas [19, 26, 87]. Prior to the reports of five intramedullary angiolipomas by Palkovic et al. [63], Preul et al. [72], Maggi et al. [48], Klisch et al. [37] and Weill et al. [96], these were thought to occur exclusively in the epidural space. Three cases of intracranial angiolipomas have been described: two were parasellar, adherent to the dura and in intimate contact with the cavernous sinus [89, 97], and the other located in the cortico-subcortical area of the left frontal lobe [4].

The first case of spinal angiolipoma was reported by Berembruch in 1820 in a 16-year-old boy with an unsuccessfully, surgically treated thoracic mass which was confirmed at autopsy [12]. In 1901 Liebscher was the first to describe a spinal angiolipoma [43]; however, the term angiolipoma was established in 1960 by Howard and Helwig as an anatomopathological entity containing mature fat cells and proliferating vessels [35].

The histogenesis of angiolipomas is poorly understood and several theories have been advanced. Ehni and Love

postulate that, following some undefined stimulus, angiolipomas arise from pluripotential mesenchymal stem cells by divergent differentiation along both adipose tissue and angiod lines (smooth muscle and vascular endothelium) [20]. Willis [98] simply considered them to be a congenital malformation or a benign hamartoma, but most would now agree that angiolipomas and mesenchymal hamartomas arise from primitive mesenchyme [11, 42]. Hemangiomas and lipomas may represent a spectrum within which angiolipomas constitute an intermediate entity [23]. The more invasive infiltrating type of spinal angiolipoma would then represent a shift towards the hemangioma end of the spectrum [61]. Pagni and Canavero support the theory of abnormal developmental origin, based on their findings of spinal angiolipomas in patients with birth defects outside the CNS [62]. Spinal lipomas differ from angiolipomas in that they are most commonly found in the lumbosacral region, rather than mid-thoracic region, and may be associated with dysraphic abnormalities [21, 46]. Some consider spinal angiolipomas to be more aggressive than spinal lipomas [73].

Pregnancy was an aggravating factor in 11 (15.7%) of the 70 female patients reported. Termination of pregnancy, as in the case reported by Preul et al. [72] may bring about regression of symptoms [8, 18]. Pregnancy may interfere with spinal venous drainage and increase epidural venous pressure from compression of pelvic and abdominal veins; the redistribution of blood, the increase in extracellular fluid, and the forceful Valsalva maneuvers associated with vaginal delivery may acutely exacerbate symptoms [8, 73]. Other possible factors reported are vascular steal phenomenon as a cause of spinal cord ischemia [93] and pulsatile compressive effect on the adjacent cord because of its vascularity [92]. Hormonal influences and increased adiposity produced by pregnancy may also cause an increase in the size of spinal angiolipomas, much as obesity may lead to symptoms induced by increasing their fatty component as occurs in patients who receive steroid treatment or are overweight [31, 46]. Tumor thrombosis and/or hemorrhaging may cause a sudden deterioration in condition, as occurred in two cases reported by Labram et al. [42] and Anson et al. [5].

Macroscopically, the tumor is an encapsulated or unencapsulated, reddish soft mass extending into the extradural space of the spinal canal. They are subdivided into two types which should be considered and treated differently: non-infiltrating and infiltrating, the latter extending into the vertebral body [45]. In 1966, Gonzalez-Crussi [28] described the first case of vertebral “infiltrating” angiolipoma and stated that these tumors should be distinguished from angiolipomas described by Howard and Hellwig [35]. Lin and Lin [44] established infiltrating angiolipoma as a separate entity, justifying their decision on differences in clinical course, pathological picture, and

prognosis from those of “plain angioliipomas”. Histologically, they are composed of mature adipose tissue and blood vessels, features of which are described as being either normal or mimicking capillary angioma, cavernous angioma or arteriovenous malformations. The fatty tissue is of the adult type and shows no remarkable findings. The ratio of fat to vessels is variable and ranges from 1:3 to 2:3. Tumors with an abundance of smooth muscle proliferation are further subclassified as angiomyoliipomas. A thin capsule, often defective in many areas, may surround the lesion. Secretory activity has been described in one case with lipid-like material in perivascular granules [10]. In two cases described by Pearson et al. [66], additional osteoid tissue was observed. Low proliferation rates are found on Ki-67 immunostaining [69]. Neither atypia, pleomorphism or mitotic figures or karyotypical abnormalities were found [4]. Sciort et al.’s [82] analysis of the karyotype in 20 angioliipomas revealed that all were normal; the authors speculate that a normal karyotype supports the theory of angioliipomas as a reactive or hamartomatous lesion. They also suggest that the vascular proliferation could be a primary event in the development of these tumors, and the adipose tissue, a secondary, albeit prominent, component.

From a clinical point of view, extradural angioliipomas do not differ from other benign space-occupying spinal lesions. Subjective complaints are mostly of sensory disorders and motor deficits below the level of the lesion [53] that often progress to weakness in the lower limbs for long periods with sphincter dysfunction in the later stages [73].

In most instances spinal radiographs were normal with only a few reports of bone involvement, i.e., bone erosion had a coarsened trabecular pattern more typical of vertebral hemangioma in two infiltrating angioliipomas [41, 93], but no calcifications were reported. Myelography was the most common radiologic technique for identifying the tumor prior to 1988; the findings, however, were not specific and showed partial or total blocking of the contrast column that was also characteristic of other extradural lesions compressing the spinal cord [77]. Spinal angiography was undertaken in two cases revealing prominent vascularity in the vertebral body [4] or the lateral aspect of the T9 vertebra in the other [75], both of which were supplied by a branch of the intercostals artery.

Rubin et al. [77] and Weil et al. [96], report that CT scan revealed dense fatty tissue in most of the extradural angioliipomas. Though CT scan usually demonstrates a hypodense lesion, some tumors are isodense [52] or hyperdense depending on the extent of the vascular component or the presence of calcification. Pagni and Canavero [62] suggest that CT is not specific for spinal epidural angioliipomas and could be misleading.

MRI is the most valuable radiological technique for diagnosing spinal angioliipomas [38], which are typically hyperintense on non-contrast T1-weighted images owing to their fatty content [95]. Provenzale and McLendon [73] showed that large hypointense foci observed within spinal angioliipomas on non-contrast T1-weighted images are correlated with increased vascularity and most lesions enhance with gadolinium administration whereas T2-weighted imaging can be variable, but are usually hyperintense [52, 63, 71, 90]. Non-infiltrating angioliipomas are generally located in the posterior portion of the epidural space and are well delimited from the surrounding tissue and can usually be removed easily by laminectomy.

Total removal of infiltrating angioliipomas, that often involve the body of the vertebral than the posterior arch, has been recommended using the anterolateral approach [93] and stabilization of the affected vertebrae is desirable. Some authors believe that the tumor-invaded vertebral body should be preserved, because analogous to vertebral hemangiomas, spinal angioliipomas may not enlarge [4].

In three cases, postoperative radiotherapy was administered following a partial excision owing to concerns of potential malignancy [5, 15, 28]. No adjuvant radiation should be applied to patients with this benign pathological entity, since even in the infiltrating group, the prognosis is very good. Most authors report good outcomes after surgical excision of spinal angioliipomas in spite of severe preoperative neurological deterioration [4]. Only one case of recurrence of an angioliipoma has ever been reported with successful surgery 12 years after the first intervention [11] and none has been reported in all the other cases of infiltrating angioliipomas, even in cases in which a complete removal could not be attained [73]. A total of six deaths have been reported, three of which date to the late 1800s or early 1900s with no clear indication as to the cause of death [8, 12, 36, 44]. More recently, one patient who had never undergone surgery died from a cervical glioblastoma [34] and the other from a respiratory infection [29].

Conclusions

Spinal angioliipomas are rare benign tumors, of angiomatous and lipomatous tissue, predominantly located in the mid-thoracic region. Although the etiology of angioliipomas remains unknown, they may result from the abnormal development of the primitive, pluripotential mesenchyme from which adipose tissue, smooth muscle, and vascular endothelium arise, or they may be hamartomatous in nature.

All angioliipomas show iso- or hyperintensity on T1-weighted images and hyperintensity on T2-weighted

images and most lesions enhance with gadolinium administration. The treatment for spinal extradural angioliipomas is total surgical resection and no adjuvant radiation therapy should be administered to patients with this pathological entity since prognosis, even if infiltrating, is excellent.

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References

1. Akhaddar A, Gazzaz M, Derraz S, Rifi L, Amarti A, Aghzadi A, El Ouahabi A, El Khamlich A (2000) Spinal epidural angioliipomas: a rare cause of spinal cord compression. A report of 8 cases and review of the literature. *Neurochirurgie* 46:523–533
2. Al-Anazi A, Ammar A, Shannon P, Al-Mulhim F (2000) Spinal extradural angioliipoma. *Br J Neurosurg* 14:471–472. doi:10.1080/02688690050175319
3. Amlashi SF, Morandi X, Chabert E, Riffaud L, Haegelen C, Rolland Y (2001) Spinal epidural angioliipoma. *J Neuroradiol* 28:253–256
4. Andaluz N, Balko G, Bui H, Zuccarello M (2000) Angioliipomas of the central nervous system. *J Neurooncology* 49:219–230. doi:10.1023/A:1006492629326
5. Anson JA, Cybulski GR, Reyes M (1990) Spinal extradural angioliipoma: a report of two cases and review of the literature. *Surg Neurol* 34:173–178. doi:10.1016/0090-3019(90)90069-2
6. Aversa do Souto A, Domingues FS, Chimelli L, Lemos AM (2003) Lumbosacral angioliipoma: case report. *Arq Neuropsiquiatr* 61:269–273
7. Bailey D, Andrews J, Mazur E, Cleary J (2000) Dorsal thoracic cord compression from a spinal angioliipoma: case report and brief comment. *Conn Med* 64:267–269
8. Balado M, Morea R (1928) Extramedular haemangioma producing paraplegia during pregnancy. *Arch Argent Neurol* 1:345–351
9. Balbo RJ, Araujo JF, Melro CA, Infigliola MG, Valvassore FR (1995) Thoracic epidural angioliipoma. Report of a case. *Arq Neuropsiquiatr* 53:659–661
10. Bardosi S, Schaale TH, Friede RL, Roessmann U (1985) Extradural spinal angioliipoma with secretory activity. An ultrastructural, clinicopathological study. *Virchows Arch* 406:253–256. doi:10.1007/BF00737090
11. Bender JL, Van Landingham JH, Manno NJ (1974) Epidural lipoma producing spinal cord compression. *J Neurosurg* 41:100–103
12. Berenbruch K (1890) Ein fall von multiplen angioliipomen kombiniert mit eniem angiom des ruckenmarks. *Tubingen*
13. Boockvar JA, Black K, Malik S, Stanek A, Tracey KJ (1997) Subacute paraparesis induced by venous thrombosis of a spinal angioliipoma: a case report. *Spine* 22:2304–2308. doi:10.1097/00007632-199710010-00022
14. Bouramas D, Korres DS, Roussos L, Mantzilas T, Anagnostopoulos D (1995) Spinal extradural angioliipoma. *J Spinal Disord* 8:324–327
15. Bucy PC, Ritchey H (1947) Klippel–Feil syndrome associated with compression of spinal cord by an extradural hemangioliipoma. *J Neurosurg* 4:476–481
16. Butti G, Gaetani P, Scelsi M, Pezzotta S (1984) Extradural spinal lipomas. Report of two cases and review of the literature. *Neurochirurgia (Stuttgart)* 27:28–30
17. Cubillos A, Acevedo H, Castillo M, Ramis C, Gutiérrez J, Trewhela R, Olivares O (2005) Spinal extradural angioliipoma. Report of one case presenting during pregnancy. *Rev Chil Neurocirug* 24:42–46
18. Cull DJ, Erdohazi M, Symon L (1978) Extradural haemangioliipoma of the spinal canal: two cases presenting during pregnancy. *Acta Neurochir (Wien)* 45:187–193. doi:10.1007/BF01774393
19. Dogan S, Arslan E, Sahin S, Aksoy K, Aker S (2006) Lumbar spinal extradural angioliipomas. Two cases reports. *Neurol Med Chir (Tokyo)* 46:157–160. doi:10.2176/nmc.46.157
20. Ehni G, Love JG (1945) Intraspinal lipomas: report of cases, review of the literature, and clinical and pathological study. *Arch Neurol Psychiatry* 53:1–28
21. El Abbadi N, El Mostarchid B, Bellakhdar F (1999) Thoracic epidural spinal angioliipoma Case report. *Pan Arab J Neurosurg* 3:49–51
22. Fernández JJ, Abad RM, Ribas T, García CPJ, Mostaza SAL, Vihuela LJ, Mazabel M (1994) Spinal angioliipoma causing acute paraplegia. Report of two cases. *Neurocirugia* 5:242–245
23. Fournay DR, Tong KA, Macaulay RJ, Griebel RW (2001) Spinal angioliipoma. *Can J Neurol Sci* 28:82–88
24. Frazier CH, Allen AR (1918) *Surgery of the spine and spinal cord*. Appleton, New York
25. Gagliardi FM, Gambacorta D (1968) Extradural thoracic angioliipoma. *Gazz Int Med Chir* 73:5602
26. Gelabert-González M, Agulleiro-Díaz J, Reyes-Santías RM (2002) Spinal extradural angioliipoma with a literatura review. *Childs Nerv Syst* 18:725–728. doi:10.1007/s00381-002-0653-5
27. Giuffré R (1976) Spinal lipomas. In: Vinken AJ, Bruyn GN (eds) *Handbook of clinical neurology*, vol 20. North-Holland, Amsterdam, pp 389–414
28. Gonzalez-Crussi F, Enneking WF, Arian VM (1966) Infiltrating angioliipoma. *J Bone Joint Surg Am* 48:1111–1124
29. Goyal RN (1980) Epidural lipoma causing compression of the spinal cord. *Surg Neurol* 14:77–79
30. Griebel RW, Khan M, Rozdilsky B (1986) Spinal Extradural angioliipoma. A case report and literature review. *Spine* 11:47–48. doi:10.1097/00007632-198601000-00012
31. Guegan Y, Fardoun R, Launois B, Pecker J (1982) Spinal cord compression by extradural fat after prolonged corticosteroid therapy. *J Neurosurg* 56:267–269
32. Haddad FS, Abba A, Allam CK (1986) Extradural spinal angioliipoma. *Surg Neurol* 26:473–486. doi:10.1016/0090-3019(86)90261-2
33. Hanakita J, Koyama T (1982) Spinal epidural hemangioliipoma. A case report. *No Shinkei Geka* 10:313–316 in Japanese
34. Henry P, Vital C, Vallat JM et al (1971) Anatomoclinical study of an extradural angioliipoma associated with intramedullary glioblastoma. *Rev Neurol (Paris)* 124:385–391
35. Howard WR, Helwig EB (1960) Angioliipoma. *Arch Dermatol* 82:924–931
36. Kasper JA, Cowan A (1929) Extradural lipoma of the spinal canal. *Arch Pathol (Chic)* 8:800–802
37. Klisch J, Spreer J, Bloss HG, Baborie A, Hubbe U (1999) Radiological and histological findings in spinal intramedullary angioliipoma. *Neuroradiology* 41:584–587. doi:10.1007/s002340050811
38. Konya D, Ozgen S, Kurtkaya O, Pamir NM (2006) Lumbar spinal angioliipoma: case report and review of the literature. *Eur Spine J* 15:1025–1028. doi:10.1007/s00586-005-1028-x
39. Krishnan VB, Law AJ, Kenwright DN (1996) Spinal extradural angioliipoma. *Aust N Z J Surg* 66:194–196. doi:10.1111/j.1445-2197.1996.tb01159.x
40. Kujas M, Lopes M, Lalam TF, Fohanno D, Poirier J (1999) Infiltrating extradural spinal angioliipoma. *Clin Neuropathol* 18:93–98
41. Kuroda S, Abe H, Akino M, Iwasaki Y, Nagashima K (1990) Infiltrating spinal angioliipoma causing myelopathy: case report.

- Neurosurgery 27:315–318. doi:[10.1097/00006123-199008000-00027](https://doi.org/10.1097/00006123-199008000-00027)
42. Labram EK, El-Shunnar K, Hilton DA, Robertson NJ (1999) Revisited: spinal angioliopoma, three additional cases. *Br J Neurosurg* 13:25–29. doi:[10.1080/02688699944140](https://doi.org/10.1080/02688699944140)
 43. Liebscher C (1901) Angiolipom des Wirbelkanals mit Kompression des Rückenmarks. *Pag Med Wochenschr* 26:189–191
 44. Lin JJ, Lin F (1974) Two entities in angioliopomas. A study of 459 cases of lipoma with review of literature on infiltrating angioliopoma. *Cancer* 34:720–727. doi:[10.1002/1097-0142\(197409\)34:3<720::AID-CNCR2820340331>3.0.CO;2-K](https://doi.org/10.1002/1097-0142(197409)34:3<720::AID-CNCR2820340331>3.0.CO;2-K)
 45. Lipson SJ, Naheedy MH, Kaplan MM, Bienfang DC (1980) Spinal stenosis caused by epidural lipomatosis in Cushing's syndrome. *N Engl J Med* 302:336
 46. Llewellyn CG (1996) Spinal angioliopoma mimicking extradural lipomatosis. *Can Assoc Radiol J* 47:51–53
 47. Lo Re F, Michelacci M (1969) Clinical and surgical findings on various lumbo-sacral abnormalities associated with angioliopoma. *Arch Putti Chir Organi Mov* 24:70–85
 48. Maggi G, Aliberti F, Colucci MR, Petrone G, Dorato P, De Giorgi AM (1996) Spinal intramedullary angioliopoma. *Childs Nerv Syst* 12:346–349. doi:[10.1007/BF00301027](https://doi.org/10.1007/BF00301027)
 49. Maier HC (1962) Extradural and intrathoracic lipoma causing spinal cord compression: successful treatment by surgical excision. *JAMA* 181:610–612
 50. Mascalchi M, Arnetoli G, Dal Pozzo G, Canavero S, Pagni CA (1991) Spinal epidural angioliopoma: MR findings. *AJNR Am J Neuroradiol* 12:744–745
 51. Matsushima K, Shinohara Y, Yamamoto M et al (1987) Spinal extradural angioliopoma: MR and CT diagnosis. *J Comput Assist Tomogr* 11:1104–1106
 52. Michilli R, Tzonos P, Iglesias-Rozas JR (1993) Spinal extradural angioliopoma: case report and literature review. *Neurochirurgia (Stuttg)* 36:63–65
 53. Miki T, Oka M, Shima M, Hirofujii E, Tanaka S (1981) Spinal angioliopoma. A case report. *Acta Neurochir (Wien)* 58:115–119. doi:[10.1007/BF01401689](https://doi.org/10.1007/BF01401689)
 54. Mimata C, Wada H, Sano Y, Saitou Y, Kitamura I, Ushio Y (1992) Spinal extradural angioliopoma: a case report. *No Shinkei Geka* 20:1085–1089
 55. Moscatelli G, Merigliano D (1958) Considerazioni istogenetiche e nosografiche sui lipoma extradurali Contributo casistico. *Riv Neurol* 28:667–668
 56. Nishiura I, Kubo Y, Koyama T (1986) Spinal haemangioliopoma: three case reports. *Neurochirurgia (Stuttg)* 29:63–66
 57. Obrador S, Villarejo F, Deblas A (1977) Extradural angioliopoma: a rare cause of spinal cord compression. *Rev Clin Esp* 146:395–396
 58. Occhiogrosso M, Vailati G (1977) Medullar compression due to epidural angioliopoma. Report of a case. *Acta Neurol (Napoli)* 32:850–853
 59. O'Donovan NA, Naik K, Maloney WJ, Llewellyn CG (1996) Spinal angioliopoma mimicking extradural lipomatosis. *Can Assoc Radiol J* 47:51–53
 60. Oge HK, Söylemezoglu F, Rousan N, Ozcan OE (1999) Spinal angioliopoma: case report and review of literature. *Spinal Disord* 12:353–356
 61. Padovani R, Tognetti F, Speranza S, Pozzati E (1982) Spinal extrathecal hemangioliopomas: report of two cases and review of the literature. *Neurosurgery* 11:674–677. doi:[10.1097/00006123-198211000-00010](https://doi.org/10.1097/00006123-198211000-00010)
 62. Pagni CA, Canavero S (1992) Spinal epidural angioliopoma: rare or unreported? *Neurosurgery* 31:758–764
 63. Palkkovic S, Wassman H, Bonse R, Kashab M (1988) Angioliopoma of the spinal cord magnetic resonance imaging and microsurgical management. *Surg Neurol* 29:243–245. doi:[10.1016/0090-3019\(88\)90014-6](https://doi.org/10.1016/0090-3019(88)90014-6)
 64. Parizel PM, Balériaux D, Rodesch G, Segebarth C, Lalmand B, Christophe C et al (1989) Gd-DTPA enhanced MR imaging of spinal tumors. *AJR Am J Roentgenol* 152:1087–1096
 65. Pasquier B, Vasdev A, Gasnier F, Chirossel JP, Kedani E, Pasquier D, Crouzet G, Couderc P (1984) Epidural angioliopoma: a rare and curable cause of spinal cord compression. *Ann Pathol* 4:365–369
 66. Pearson J, Stellar S, Feigin I (1970) Angiomyoliopoma: long term cure following radical approach to malignant-appearing benign intraspinal tumor-report of 3 cases. *J Neurosurg* 33:466–470
 67. Petit-Dutailis D, Christoph J (1931) Spinal cord compression due to a giant extradural angioliopoma. Surgical removal. *Rev Neurol (Paris)* 2:824–827
 68. Petrella G, Tamburrini G, Lauriola L, Di Rocco C (2005) Spinal epidural angioliopoma complicated by an intratumoral abscess. Case report. *J Neurosurg* 103(2 suppl):166–169
 69. Pinto-Rafael JJ, Vázquez-Barquero A, Martín-Laez R, García-Valtuille R, Sanz-Alonso F, Figols-Guevara FJ, MC Pérez-Castro, Abascal F, Cereza L (2002) Spinal angioliopoma. Report of one case. *Neurocirugia* 13:321–325
 70. Poon TP, Beban M, Matoso IM, Katz MA, Pearl M (1988) Epidural angioliopoma with spinal cord compression. *J Natl Med Assoc* 80:347–349
 71. Preul MC, Leblanc R (1993) MRI in the diagnosis of spinal extradural angioliopoma. *Br J Neurosurg* 7:32–38. doi:[10.3109/02688699309023822](https://doi.org/10.3109/02688699309023822)
 72. Preul MC, Leblanc R, Pampieri D, Robitaille Y, Pokrupa R (1993) Spinal angioliopomas. Report of three cases. *J Neurosurg* 78:280–286
 73. Provenzale JM, McLendon RE (1996) Spinal angioliopomas: MR features. *AJNR Am J Neuroradiol* 17:713–719
 74. Rabin D, Hon BA, Pelz DM, Ang LC, Lee DH, Duggal N (2004) Infiltrating spinal angioliopoma. A case report and review of the literature. *J Spinal Disord Tech* 17:456–461. doi:[10.1097/01.bsd.0000109834.59382.d5](https://doi.org/10.1097/01.bsd.0000109834.59382.d5)
 75. Rivkind A, Margulies JY, Lebenstart P, Sherman Y, Robin GC (1986) Anterior approach for removal of spinal angioliopoma: a case report. *Spine* 11:623–625. doi:[10.1097/00007632-198607000-00016](https://doi.org/10.1097/00007632-198607000-00016)
 76. Rocchi G, Caroli E, Frati A, Cimatti M, Savlati M (2004) Lumbar spinal angioliopomas: report of two cases and review of the literature. *Spinal Cord* 42:313–316. doi:[10.1038/sj.sc.3101535](https://doi.org/10.1038/sj.sc.3101535)
 77. Rubin G, Gornish M, Sandbank J, Shevach I, Rappaport ZH (1992) Spinal extradural angioliopoma. Case report and review of the literature. *Spine* 17:719–724. doi:[10.1097/00007632-199206000-00013](https://doi.org/10.1097/00007632-199206000-00013)
 78. Sakaida H, Waga S, Kojima T, Kubo Y, Matsubara T, Yamamoto J (1998) Thoracic spinal angioliopoma with extracanal extension to the thoracic cavity: a case report. *Spine* 23:391–394. doi:[10.1097/00007632-199802010-00022](https://doi.org/10.1097/00007632-199802010-00022)
 79. Sakaki T, Fujita T, Yoshimine T, Taneda M, Hayakawa T (1993) Spinal extradural angioliopoma: a case report. *No Shinkei Geka* 21:959–962
 80. Samdani AF, Garonzik IM, Jallo G, Eberhart CG, Zaos P (2004) Spinal angioliopoma: case report and review of the literature. *Acta Neurochir (Wien)* 146:299–302. doi:[10.1007/s00701-003-0196-x](https://doi.org/10.1007/s00701-003-0196-x)
 81. Scanarini M, Carteri A (1974) Intrarachidian lipomas. Observation on 4 cases and pathogenetic considerations. *J Neurosurg Sci* 18:136–141
 82. Sciort R, Akerman M, Dal Cin P, De Wever I, Fletcher CD, Mandahl N, Mertens F, Mitelman F et al (1997) Cytogenetic analysis of subcutaneous angioliopoma: Further evidence supporting its difference from ordinary pure lipomas. A report of the

- CHAMP Study Group. *Am J Surg Pathol* 21:441–444. doi: [10.1097/00000478-199704000-00010](https://doi.org/10.1097/00000478-199704000-00010)
83. Shibata Y, Sugimoto K, Matsuki T, Nose T (1993) Thoracic epidural angioliipoma: case report. *Neurol Med Chir (Tokyo)* 33:316–319. doi: [10.2176/nmc.33.316](https://doi.org/10.2176/nmc.33.316)
 84. Shiffer J, Giboa Y, Ariazoroff A (1980) Epidural angioliipoma producing compression of the cauda equina. *Neurochirurgia (Stuttg)* 23:117–120
 85. Shuangshoti S, Hongsaprabhas C (1979) Intraspinal epidural angioliipoma. *J Med Assoc Thai* 62:457–460
 86. Shuangshoti S, Lerdlum S (1997) Concurrent occurrence of solitary spinal epidural osteochondroma and angioliipoma. *Clin Neuropathol* 16:107–110
 87. Stookey B (1927) Intradural spinal angioliipoma: report of a case and symptoms for 10 years in child aged 11: review of literature. *Arch Neurol Psychiatry* 18:16–43
 88. Stranjalis G, Jamjoom A, Torrens MJ (1992) MRI in the diagnosis of spinal extradural angioliipoma. *Br J Neurosurg* 6:481–483. doi: [10.3109/02688699208995039](https://doi.org/10.3109/02688699208995039)
 89. Takeuchi J, Handa H, Keyaki A, Haibara H, Ozaki S (1981) Intracranial angioliipoma. *Surg Neurol* 15:110–113. doi: [10.1016/0090-3019\(81\)90024-0](https://doi.org/10.1016/0090-3019(81)90024-0)
 90. Taylor J, Harries BJ, Schurr PH (1951) Extrathecal hemangioliipomas of the spinal canal. *Br J Surg* 39:1–7. doi: [10.1002/bjs.18003915302](https://doi.org/10.1002/bjs.18003915302)
 91. Trabulo A, Cerqueira L, Monteiro J, Roque P, Reis FC, Coelho MR (1996) Spinal angioliipomas revisited: two case reports. *Acta Neurochir (Wien)* 138:1311–1319. doi: [10.1007/BF01411061](https://doi.org/10.1007/BF01411061)
 92. Turanzas FS, Domínguez J, Eyerbe P, Dhimes P (1994) Infiltrating spinal angioliipoma. Case report. *Neurocirugia* 5:246–249
 93. Turgut M (1999) Spinal angioliipomas: report of a case and review of the cases published since the discovery of the tumour in 1890. *Br J Neurosurg* 13:30–40. doi: [10.1080/02688699944159](https://doi.org/10.1080/02688699944159)
 94. von Hanwehr R, Apuzzo MLJ, Ahmadi J, Chandrasoma P (1985) Thoracic spinal angiomyoliipoma: case report and literature review. *Neurosurgery* 16:406–411. doi: [10.1097/00006123-198503000-00023](https://doi.org/10.1097/00006123-198503000-00023)
 95. Warot P, Petit H (1969) Extra-dural dorsal angioliipoma: unusual case of medullary compression. *Lille Med* 14:311–315
 96. Weill A, del Carpio-ODonovan R, Tampieri D, Melanson D, Ethier R (1991) Spinal angioliipomas: CT and MR aspects. *J Comput Assist Tomogr* 15:83–85. doi: [10.1097/00004728-199101000-00011](https://doi.org/10.1097/00004728-199101000-00011)
 97. Wilkins PR, Hoddinott C, Hourihan MD et al (1987) Intracranial angioliipoma. *J Neurol Neurosurg Psychiatry* 50:1057–1059
 98. Willis RA (1948) Pathology of tumors. Butterworths, London, p 700
 99. Yamashita K, Fuji T, Nakai T, Hamada H, Kotoh K (1993) Extradural spinal angioliipoma: report of a case studied with MRI. *Surg Neurol* 39:49–52. doi: [10.1016/0090-3019\(93\)90110-M](https://doi.org/10.1016/0090-3019(93)90110-M)