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Intramedullary Spinal Cord Metastases

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Summary. Seven autopsy cases of intramedullary metastases, four in the cervical spinal cord, are reported and the literature reviewed. Whereas lung and breast cancer, malignant melanomas and lymphomas are reported as the most common primary tumors, the present series included three cases of breast carcinoma and two cases each of colon and oat cell carcinoma of the lung. Neither the clinical symptoms nor the neurological signs distinguished intramedullary metastases from the more common extradural deposits, but radiological evidence of vertebral metastases and myelographic stop were present in only one case each, and CSF cytology was negative. Intramedullary deposits in this series were neither associated with extradural tumor nor with spread into the subarachnoid space, while cerebral metastases were present in four cases. This favors hematogenous dissemination rather than direct transdural or perineural spread of these lesions.

Key words: Metastases – Intramedullary tumor – Myelography – CSF cytology – Differential diagnosis.

Zusammenfassung. Es wird über 7 Autopsiefälle von intramedullären Metastasen, davon 4 im Halsmark, berichtet und ein Überblick des Schrifttums gegeben. Während Bronchus- und Mammakarzinome, maligne Melanome und Lymphome die häufigsten Primärgeschwülste darstellen sollen, lagen in dieser Serie 3 Mammakarzinome und je 2 Kolon- und Haferzellkarzinome des Bronchus vor. Weder der klinische Verlauf noch die neurologische Symptomatik gestatten eine eindeutige Abgrenzung gegenüber den häufigeren extraduralen Metastasen, doch lagen röntgenologisch faßbare Wirbelmetastasen und positive Myelographie nur je einmal vor; die Liquorzytologie war negativ. Intramedulläre Metastasen unserer Serie waren weder mit extraduralen Absiedelungen noch mit Befall des Subarachnoidalraumes, in 4 Fällen aber mit Hirnmetastasen verbunden. Das spricht eher für ihre hämatogene Entstehung als ein direktes transdurales oder perineurales Einwachsen des Tumors.

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Introduction

Metastatic intramedullary spinal cord tumors are considered to be rare and may present serious diagnostic and therapeutic problems. While Guidetti and Fortuna [15] reported 16 metastases among 222 intramedullary masses found at operation, intramedullary spinal cord lesions accounted for 3.8% in an autopsy study of metastatic spinal cord tumors [33], and for 3.4% of symptomatic metastatic cord lesions [11], but were less frequent in other series [2, 8, 10, 32]. The clinical and radiological features of intramedullary spinal cord metastases were discussed by Edelson et al. [11] who reviewed 70 previously reported cases and nine of their own. Since then, a small number of intramedullary secondaries have been reported [6, 7, 8, 10, 17, 22, 25, 27]. In recent years, seven patients with metastatic intramedullary spinal cord tumors proved by postmortem examination have been observed, and will be compared with those in the literature.

Case Reports

Case 1. A man, aged 77, had had a hemicolectomy for adenocarcinoma 5 years before the development of neurological deficit. Two weeks before admission he noted nonradiating bilateral hip and leg pain. Over night he developed flaccid paraplegia, numbness of the legs and urinary retention. On neurological examination there was flaccid paraplegia and absence of deep tendon reflexes in the lower limbs without response to plantar stimulation. There was a sensory level to pinprick, touch and vibration below L_1. Spinal roentgenographs revealed spondylosis. The CSF protein content was 210 mg%. No tumor cells were identified in the CSF. On lumbar myelogram there was complete obstruction at T_{12}, suggesting spinal cord compression due to epidural metastasis, but laminectomy was not performed due to the patient's poor condition. He developed bedsores and died 3 weeks after admission. At *autopsy* the carcinoma was metastatic to the lungs, adrenals, right occipital lobe and left cerebellum, but not to the meninges. An intramedullary tumor replaced most of the spinal cord at T_{11}—L_1 and was histologically associated with subtotal transverse necrosis at these levels. There were no epidural metastases.

Case 2. This woman, aged 48, experienced weakness in her legs 10 months after resection of her right breast for undifferentiated carcinoma. Constipation and urinary hesitancy also occurred. Neurological examination 5 days after the onset of her symptoms revealed a Brown-Séquard syndrome at T_{10}. Within 2 days she became paraplegic and had a sensory level below T_{10}. Roentgenograms of the spine were negative, as were repeat myelograms. On lumbar puncture, CSF protein was 342 mg%; there were 19 monocytes/mm^3, but no tumor cells were identified. Necrotizing myelopathy or an intramedullary tumor were considered as the cause of her symptoms. Since a brain scan showed increased uptake in the left temporal lobe, no radiation therapy was performed. The patient was given corticosteroids but developed progressive disorders of consciousness and died 4 weeks after admission. *Autopsy* disclosed a solitary metastasis in the left cerebellum without involvement of the meninges. A circumscribed tumor mass with central hemorrhages was present in the lower thoracic cord (Fig. 3). Histologically, the tumor replaced most of the cord and showed extensive central necrosis. The tumor tissue did not invade the meninges at any place (Fig. 4).

Case 3. A woman, aged 50, had had radiotherapy for an epipharyngeal tumor 10 years prior to death, and resection of her right breast for cancer 5 years later. Four weeks before admission she experienced numbness of both feet progressing to the thighs and hips. One week later she developed weakness of the left arm and leg, and less of the right limbs. Urinary hesitancy also occurred. Neurological examination revealed tetraparesis more on the left, with right hemihypesthesia for

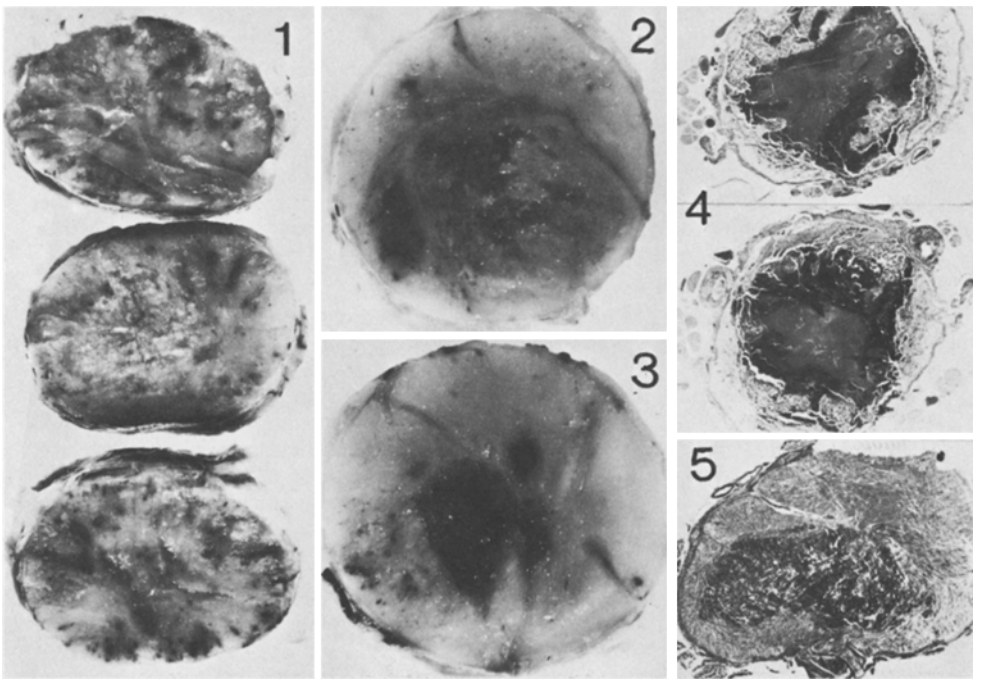

Fig. 1. Case 6. Intramedullary tumor (secondary from breast carcinoma) replacing much of cervical cord

Fig. 2. Case 4. Large metastasis from carcinoma of colon in upper cervical cord with small hemorrhages

Fig. 3. Case 2. Intramedullary hemorrhagic deposit of breast carcinoma in lower thoracic cord

Fig. 4. Case 2. Tumor replacing much of thoracic cord with large central necrosis. H-E × 5.6

Fig. 5. Case 7. Circumscribed intramedullary metastasis in cervical cord from oat cell carcinoma. C.V. × 6.4

pinprick and left hemihypesthesia for touch and vibration below C_5. Some days later she had paraplegia of the arms and spastic paraparesis of the legs with diminished tendon reflexes and a sensory level for all qualities at C_4. Spinal roentgenograms showed metastasis in the first lumbar vertebra. A lumbar myelogram was negative. CSF protein content was 120 mg%; there were 12 cells/mm^3, but no tumor cells were identified. In spite of corticosteroid treatment, the patient's condition deteriorated and she died of anuria one week after admission. *Autopsy* revealed metastases in the 5th cervical and 1st lumbar vertebrae, and a grayish intramedullary mass extending from the caudal oblongata to C_4. Below the tumor there was a cone-shaped central necrosis in the cervical and upper thoracic cord. Microscopically there were circumscribed tumor masses of solid carcinoma in the central areas of the caudal oblongata (level of the pyramidal decussation) and upper cervical cord. Below the tumor, a pencil-shaped necrosis involving the ventral areas of the posterior columns extended to the T_2 level. No tumor was present in the spinal meninges, nor in the spinal epidural and intracranial areas.

Case 4. A woman, aged 74, noted severe local pain in the neck about 6 years after resection of an adenocarcinoma of the colon. Three weeks later she developed weakness of her legs and urinary hesitancy. Neurological findings included tetraparesis with hyperactive tendon reflexes, more prominent on the right than on the left, bilateral Babinski reflexes, and hypesthesia to prinpick and

temperature on the left and for vibration on the right below C_{3-4}. Within the next few days she developed a sensory level for all qualities below C_3, and confusional states. Roentgenograms of the spine were negative. CSF protein was 160 mg%; there were 14 cells/mm^3, but no malignant cells were detected. Myelogram was not performed. The patient died 9 days after admission. It was believed that she had either myelomalacia or an intramedullary tumor at the upper cervical level. *Autopsy* revealed a small metastasis in the left frontal lobe and an intramedullary tumor in the upper cervical cord with small hemorrhages (Fig. 2). Histologically, the cone-shaped tumor tissue extended from C_2 to C_4 and was associated with necrosis in the posterior columns. Neither meningeal nor extradural neoplastic deposits were seen. There were no extraneural secondaries.

Case 5. This woman, aged 69 years, developed pain in the neck radiating into both arms 6 weeks prior to death. Over the next 4 weeks weakness of her legs and left arm developed. Neurological examination disclosed a Brown-Séquard syndrome at C_3 with increased tendon reflexes except for decreased left biceps reflex, bilateral pyramidal signs, and 3 days later, a sensory level below C_3. Spinal roentgenograms were negative. CSF protein was 190 mg%. CSF cytology and a myelogram were not performed. Thoracic roentgenograms disclosed a central carcinoma of the left lung. The patient's condition deteriorated and she died within 2 weeks. At *autopsy,* there were widespread metastases of oat cell carcinoma sparing the brain and spinal dura. The cervical cord was enlarged from C_5 to C_8 by an intramedullary tumor involving much of the cord. Histologically, the tumor cells appeared to have gained entrance at one level into the adjacent meninges, but most of the subarachnoid space was spared. There was edema and demyelination of the upper thoratic cord.

Case 6. A woman, aged 67 years, had had resection of her left breast for undifferentiated carcinoma 9 months prior to death. Postsurgical radiotherapy was given. Six months before admission, she noted back pain and weakness of the right arm and leg. About 4 weeks later she developed tetraparesis, numbness below the umbilical level, and dysfunctions of bladder and bowel. Neurological findings included tetraparesis more prominent on the right, with positive Babinski signs, and a sensory level below T_4, with sensory loss for pinprick on the left and for touch more on the right. Two days later she had tetraparesis and a complete sensory level below C_6. Left carotid angiography, skull and spine roentgenographs, and a myelogram were negative. Lumbar puncture revealed 178 mg% protein and 12 monocytes/mm^3, but no tumor cells. The patient died one week after admission. At *autopsy* carcinoma was metastatic to cervical lymph nodes and lungs, but not to the brain and meninges. Within the cervical spinal cord, tumor masses extended from C_4 to C_6 (Fig. 1). Histologically they were located in the central areas of the cord and did not invade the meninges. Below the tumor, a cone-shaped necrosis in the posterior columns extended to the T_{3-4} level.

Case 7. A man, aged 78 years, noted weakness and numbness of the legs 2 months prior to death. Over the next week he developed paraplegia of the legs, constipation and bladder dysfunction. Neurological examination disclosed flaccid paraplegia of the legs with bilateral areflexia and a sensory level below L_2. Roentgenographs of the lungs, skull and spine, and repeat myelograms were negative. CSF protein was 200 mg% with 14 monocytes/mm^3, but no tumor cells were seen. Despite corticoid treatment the patient's condition deteriorated, and he died in coma. *Autopsy* revealed oat cell carcinoma of the lung metastatic to the adrenals, left cerebellum and right thalamus, and a 1.5 cm cone-shaped intramedullary tumor in the conus at L_{2-4}. Histologically much of the cord was replaced by the circumscribed tumor that was growing to the surface of the cord and into the posterior roots, but did not invade the spinal meninges (Fig. 5).

Discussion

1. Incidence of Spinal Cord Metastases. In autopsy series of secondary blastomas of the CNS the incidence of intramedullary deposits ranges from 0.9% [24] to 5% [9], while Jänisch et al. [18], reviewing 783 autopsy cases of extraneural tumors, encountered not a single case of exclusively intramedullary metastasis. Four of

Table 1. Primary tumor in patients with intramedullary metastases

Primary	Intramedullary secondaries		
	Literature[a]	Present series	Total
Lung cancer	39	2	41
Breast cancer	14	3	17
Malignant melanoma	7	0	7
Malignant lymphoma	7	0	7
Kidney blastoma	6	0	6
Colon-rectum cancer	5	2	7
Adrenal tumor	3	0	3
Chorionepithelioma	1	0	1
Sarcoma of testicle	1	0	1
Olfact. neuroblastoma	1	0	1
Cervix carcinoma	1	0	1
Nasopharynx carcinoma	1	0	1
Stomach carcinoma	1	0	1
Unknown tumor	6	0	6
Total	93	7	100

[a] References 1, 4, 6, 7, 8, 10, 11, 17, 22, 25, 27

Table 2. Site of intramedullary metastases

Site	Literature[a]	Present series	Total
Cervical	19	4	23
Cervicothoracic	1	0	1
Thoracic	24	1	25
Thoracolumbar	8	1	9
Lumbar	11	1	12
Unknown/multiple	39	0	30
Total	93	7	100

[a] See Table 1

the seven cases reported here have been observed in a large general hospital during a 2 year period. The other three cases derived from other sources were seen in an autopsy series of 260 consecutive secondary blastomas of the CNS; an additional case of intramedullary melanoma was observed among 110 consecutive cases of neurosurgically proven metastatic cord lesions [20]. More than 40% of the reported intramedullary secondaries originate from the lung, and more rarely from the breast, kidney and colon (Table 1). Malignant melanomas and lymphomas representing other common causes of intramedullary deposits [11], have not been observed in our series. No intramedullary lesion was found in a large autopsy series of malignant lymphomas [21].

Table 3. Clinicopathological features of 7 patients with intramedullary metastases

Patient	W.J.	K.E.	M.T.
Age, sex	77 m	48 f	50 f
Primary	colon	breast	breast
Interval of surgery	5 years	10 months	5 years
Onset to full neur. deficit	1 week	1 week	4 weeks
Duration of neur. disease	3 weeks	4 weeks	5 weeks
Initial symptoms	pain in both legs	weakness legs	numbness legs, left hemiparesis
Clinical course	acute paraplegia, BBD, SL L_1	BSS T_{10}, paraparesis legs, SL T_{10}, somnolence	BSS C_4, tetraparesis BBD, incompl. transv. l. C_4
Spinal X-ray (metastasis)	neg	neg	metastasis L_1 vert
Myelogram	block T_{12}	neg	neg
CSF cells	17	19	17
protein	210 mg%	342 mg%	120 mg%
tumor cells	0	0	0
Primary	adenocarcinoma	undiff. carcinoma	solid. med. carcinoma
Extraneur. secondaries	lungs, adrenals	none	L_1 vertebra
Intramed. secondary	T_{11}–L_1	T_{9-11}	caud. oblong.–C_4
Other cord lesions	necrosis T_{10}–L_1	ascending tract deg.	cent. necrosis C_6–T_2
Meningeal invasion	0	0	0
Brain metastasis	rt occipit. cerebellum	lt cerebellum	0

BSS = Brown-Séquard syndrome, BBD = bladder and bowl dysfunction, SL = sensory level, rt = right,

Whereas in general, there is almost equal distribution of intramedullary secondaries to the cervical, thoracic and lumbar spinal cord, in our series the cervical region was most often involved (Table 2). Some of the patients had multiple lesions [11, 27].

2. Clinical Features (Tables 3 and 4). Five of our patients had had resection of carcinoma 9 months to 6 years before the onset of neurological symptoms. In one individual (Case 5), a lung carcinoma was detected radiologically, while in Case 7 no primary malignancy was known. A striking clinical feature was the rapid course of the neurological disorder. Our patients progressed from the initial symptoms to full development of neurological deficit between 1 and 5 weeks, which was considerably shorter than in many other cases (Table 4). The survival time after the appearance of spinal cord symptoms ranged from 3 weeks to 2 months. Death was usually the result of widespread metastatic disease with intracranial lesions.

Among the clinical symptoms initial pain was reported in four of our seven patients and in the majority of the previously recorded cases. The pain was usually in the midback initially, but often became radicular. Weakness was present in all our cases and about 60% had spasticity. In two patients, weakness was a presenting symptome. Paresthesias, reported in about half of the cases in

M.F.	R.M.	H.E.	L.W.
74 f	69 f	67 f	78 m
colon	lung	breast	?
6 years	0	9 months	0
3 weeks	4 weeks	2 weeks	1 week
5 weeks	6 weeks	6 weeks	2 months
cervical pain, weakness legs	radicul. cerv. pain, weakness legs	cerv. pain, rt hemiparesis	weakness legs
BSS C_3, tetraparesis, BBD, SL C_3 and rt hemiparesis	BSSC_3–T_1→ tetraparesis, BBD, SL C_3	BSS T_4, paraparesis legs, BBD, SL T_4	flaccid paraplegia, BBD, SL L_2, somnolence
neg	neg	neg	neg
n.e.	n.e.	neg	neg
14		12	14
160 mg%	190 mg%	178 mg%	200 mg%
0	n.e.	0	0
not detected	oat cell carcinoma	undiff. carcinoma	oat cell carcinoma
non	lymph nodes, adrenals	lymph nodes, lungs	lymph nodes, adrenals
C_{2-4}	C_{5-8}	C_{4-6}	L_{2-4}
necrosis vent. post. c.	edema T_{1-2}	cent. necrosis C_8–T_3	0
0	local	0	0
lt front.	0	0	rt thalamus, lt cerebell.

lt = left; n.e. = not examined

Table 4. Signs and symptoms of intramedullary metastases

Symptoms	Literature[a]	Present series	Total
Pain	34	4	38
Local	16	3	19
Radicular	18	1	19
Paresthesias	22	1	23
Bladder or bowel	26	6	32
Sensory level	29	7	36
Brown-Séquard s.	6	(5)	11
Spastic pareses	26	4	30
Time course (onset to full neurological deficit)			
less than 1 week	4	5	9
less than 1 month	18	2	20
less than 6 months	18	0	18

[a] Based on 43 previous case reports [1, 7, 10, 11, 22, 27]. In some cases complete data were not included by the authors

the literature, were observed in only one patient. All our cases had a sensory level which in five developed from an initial Brown-Séquard syndrome. The sensory level began caudally and rose toward the lesion in only one of them as rather often reported in the literature [11]. Bowel and bladder difficulties were present in six of our cases and in 60% of those in the literature.

CSF changes included considerable elevation of protein levels with normal cell counts, and no malignant cells were identified by cytological examination of the CSF. Normal myelograms were obtained in four of our cases, while one showed complete obstruction of the flow of contrast material suggesting extramedullary compression. In only one of our seven cases was there radiological and autopsy evidence of vertebral metastases. These findings as well as those reported in the literature [11] emphasize the clinical and radiological difficulties in distinguishing intramedullary metastases from extradural spinal cord compression [1, 2, 8, 11, 12, 13, 15, 16, 31].

3. Differential Diagnosis. Four symptoms characterizing the clinical features of cord compression, i.e. pain, weakness, autonomic dysfunction and sensory loss [13], are also present in most of the cases with intramedullary secondaries. Both local and radicular pain, produced and exacerbated by straight leg rising or neck flexion which are said to suggest an extramedullary lesion, were often seen with intramedullary metastases. Weakness is a frequent symptom with both extradural and intramedullary lesions, whereas muscular atrophy and fasciculations, considered typical for intramedullary lesions [15, 16], were rarely observed with intramedullary secondaries. Paresthesias and dissociated sensory loss, said to be characteristic intramedullary signs [15, 16], occurred only in part of the cases with intramedullary secondaries, which may be due to the rapid progression of the neurological deficit. However, initial asymmetrical cord signs, suggesting the Brown-Séquard syndrome, later progressing to a complete transverse lesion in four of our patients, suggested intramedullary lesions. Autonomic dysfunction, including bladder and bowel disorders, which are considered to occur earlier with intramedullary than with extramedullary lesions [11, 15, 16], were almost never initial symptoms in cases with intramedullary secondaries.

Thus, there are hardly any clinical signs or symptoms that enable differentiation between intramedullary and extradural spinal cord metastatic lesions. CSF abnormalities do not distinguish the two processes and, although myelography usually establishes the presence of an extramedullary lesion, a normal myelogram does not exclude an intramedullary process. If the myelogram is nondiagnostic, radiation myelopathy, paraneoplastic necrotizing myelopathy, vascular disorders of the spinal cord including vascular malformations, syringomyelia, multiple sclerosis, and seeding of the meninges with tumor must be considered in the differential diagnosis; the latter can be excluded by CSF cytology.

4. Neuropathology. Negative CSF cytology in the majority of intramedullary metastases of the spinal cord is in accordance with the autopsy findings that usually show a well circumscribed tumor mass extending over one or more segments. Although often much of the cord is replaced by tumor which may infiltrate the posterior roots, only rarely is there evidence of invasion or seeding into the subarachnoid space. Hemorrhage and necrosis may complicate intramedullary deposits of carcinoma [22, 30]. In four cases we saw a cone-shaped necrosis in the central areas of the posterior columns above and/or below the tumor. This central fusiform necrosis seen in a variety of spinal cord lesions

including compression, myelitis, myelomalacia, and trauma, is considered to arise either from mechanical or vascular disorders or both [19, 34]. It may explain segmental differences between the clinical deficit and the anatomical site of the tumor.

5. The pathogenesis of tumor spread into the spinal cord is controversial. Several mechanisms have been suggested:

a) Hematogenous dissemination via the arterial system which is believed to be operative in CNS leukemias and lymphomas [21, 28], meningeal carcinosis [26] and metastatic brain disease [29].
b) Tumor spread via the vertebral venous system [3] extending from the pelvis to the cranial venous sinuses which drain the CNS blood to the systemic venous circulation and enable retrograde transport to the spinal cord, as seen with cartilaginous emboli [25].
c) Spread to spinal cord by direct extension from nerve roots or CSF via intraspinal perineural sheaths as in carcinomatous meningitis [14] or direct spread from the bone along the perforating veins and nerves or perineural lymphatic channels through the dura and into the arachnoid space, as suggested in leukemia and lymphomas [5]. This latter type of direct extension may be operative in rare cases with both extradural and intramedullary lesions [11, 18], as well as in the rather frequent spread of tumor to the conus-cauda region and lumbar nerve roots [26].

In the present series of cases the intramedullary deposits were frequently combined with cerebral or extraneural metastases, but neither with extradural or local vertebral deposits nor with invasion of the subarachnoid space, which does *not* favor the assumption of direct transdural or perineural spread of tumor. It is rather suggested that most of the intramedullary secondaries may result from hematogenous dissemination. Whether this occurs via the arterial and/or venous systems remains open for discussion.

6. Treatment. Metastatic intramedullary disease is inexorably progressive, although in some instances it responded to aggressive radiation [11] which is considered by some authors as the treatment of choice for both intramedullary deposits and extradural spinal cord compression from metastases [11, 13], whereas the value of decompressive surgery is a matter of controversy [2, 8, 10, 11, 13]. In view of the rapid unfavorable course, radiotherapy was not given to any of our patients; steroid treatment of intramedullary metastases is considered to be of little value [11]. However, early diagnosis and aggressive radiation therapy in patients with radiosensitive intramedullary deposits, e.g. malignant lymphomas, may provide a more favorable outcome.

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