Congenital Spinal Extradural Cysts:
Case Report with Review of Literature

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CONGENITAL spinal extradural cysts are unusual and relatively rare lesions. A search of the world medical literature disclosed 67 publications in which are reported 91 patients with one or more spinal extradural cysts. One case was reported but unpublished.[66] The first case (2)* was discovered at autopsy in 1898,[42] all others have been proven by operation. A total of 103 cysts have been found. Six patients with multiple isolated cysts had a combined total of 16 lesions. In 1937 the author reported two cases, one, the first recorded case in an adult and in the lumbar region (13), and the other had Schuerermann's Disease (14). Before 1937 only eight verified cases could be found in the literature, two others were questionable.[30, 54] Thirty years passed (1966) before another patient with an extradural cyst was encountered. This case was similar to the one previously reported.[5] The history, physical and x-ray findings, size and location of the cyst and results of treatment were almost identical. This interesting patient is presented, therefore, to become the 92nd reported case.

Case Report

D. K., admitted Q. H., 9/23/65 #285503, a 32-year-old Japanese carpenter, complained of weakness and decrease in size of his *right* thigh muscle for 4 years. He never had pain in his back or leg or been aware of sensory loss or change until recently. He had had no trouble with his bowels or bladder. For 3 months he had weakness in his *left* leg with intermittent tingling sensation and occasional involuntary movements of the left thigh muscles. When these symptoms appeared in his "good" leg he sought medical attention.

On examination his back was straight without scoliosis or kyphosis. Percussion over the second lumbar spinous process was painful without spread or radiation. There was marked atrophy of the right thigh which measured 20 cm. smaller than the left and the calf was 2 cm. smaller (Fig. 1). The strength of the right quadriceps muscle was less than 40% of normal. The hamstrings muscles were also weak, but all muscles below the knees were intact and symmetrical. Marked fibrillations were noted in the abductor muscles of the left thigh, but none elsewhere. Tendon reflexes were hyperactive, the right knee jerk diminished, ankle jerks symmetrical. The Babinski sign was positive on the right but equivocal on the left. Sensory examination showed slight decrease to pin prick over the anterior thigh below the groin corresponding to the second, third, fourth and part of the fifth lumbar dermatomes. All sacral areas on the right leg were hypersensitive compared to the left. The cranial nerves and upper extremities were normal.

X-rays of the thoraco-lumbar spine disclosed an extensive intraspinal mass. The right vertebral pedicle of L1 and L2 were completely destroyed with moderate bilateral erosion of the pedicles of D10, 11 and 12. The intrapedicular distance measured 33 mm. at L2 and 38 mm. at L1 compared to 25 mm. and 26 mm. at L5 and L4, respectively (Fig. 3B). In the lateral film there was deep concave erosion of the posterior surface of the vertebral bodies of T11, 12 and L1. A clinical diagnosis of a spinal extradural cyst was made from experience with a previous case.[5]

An attempt was made to insert a needle into the spinal canal at L1–2 in the hope that the cyst could be punctured and pantopaque injected into it. This was unsuccessful due to overlapping laminae, so a myelogram was performed using 6 cc. of pantopaque. The oil encountered a partial obstruction at L2, then progressed slowly to T9 beyond which it flowed freely. The return flow was held up momentarily between T9 and T10. These two

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* (2) refers to case number in Table 1 listed in order reported.

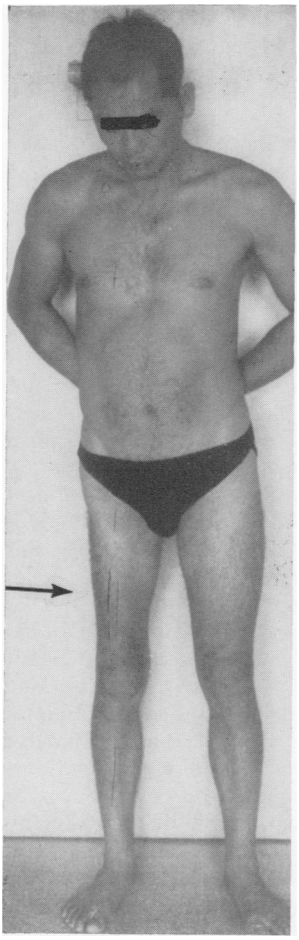

FIG. 1. Photograph of patient showing atrophy of right leg.

areas corresponded to the extremes of the pedicular erosion and were assumed to be the upper and lower ends of the lesion.

To demonstrate a communication between the subarachnoid space and the interior of the cyst, the patient was placed in a left oblique position, head down, and the oil pooled opposite T12 and L1. The oil immediately filled the dural sleeve of 12th thoracic root (Fig. 2a). At the end of this dural cuff a small round globule of oil began to form and gradually increased in size to 3 or 4 mm. in diameter (Fig. 2b). It then suddenly broke away, moved rapidly cephalad and stopped at the upper border of T10 (Fig. 2c). Another globule soon formed at the nerve root and followed the same path. It was apparent that the subarachnoid oil was passing through a defect in the sleeve of the nerve root and into the cyst. The patient was kept in the head-down position for approximately 20 minutes during which time a sufficient quantity of pantopaque entered the cyst to outline its upper end (Fig. 3a); and the lower end filled when the patient was placed upright (Fig. 3b). A cineradiographic film strip was taken to demonstrate passage of the oil from the subarachnoid space into the cyst (Fig. 2a, b, c).

Operation

Laminectomy was performed from T10 to L2. The laminae were thin, there was very little epidural fat. The lesion, a large single thin walled cyst completely filled the markedly dilated spinal canal. It ruptured easily when handled and clear spinal fluid escaped, collapsing the cyst. It was easily separated from the underlying dura, being attached only by occasional loose fibrous strands. The pedicle attachment to a single nerve root (left D12) was easily demonstrated. This was divided between two silver clips and the cyst completely removed. Its size and contour were reconstructed for photographs by filling the cyst with warm gelatin which hardened when cooled (Fig. 4). Because of the extensive laminectomy (six spinous processes and lamina) all removed bone was saved, run through a bone mill and converted into finely ground bone crumbs. These were packed over a thick pad of gelfoam in the laminar defect to protect the spinal cord and possibly produce an interlaminar fusion.

Postoperative recovery was smooth and uneventful. He was discharged from the hospital on October 1, 1966 with no increase in neurological deficit. He returned to his job on November 24th and continues to work to the present. Fourteen months after operation, he had no complaints except occasional mild low back pain. He was not aware of impairment in strength of his leg muscles. The severe degree of atrophy of his right thigh muscles had improved remarkably, increasing 9 cm. The left leg measurements were the same as before operation.

Comment. There was a striking similarity between this case and the one recorded in 1937. The lesion arose from the left L1 nerve root in one patient and T12 in the other. The duration of symptoms was 3 years in the previous case and 3 to 4 years in the present one. The cysts were almost identical in size, 14 cm. versus 13½ cm. The same vertebral pedicles were destroyed and the maximum increased diameter of the spinal canal was at L1 in both cases. The myelogram demonstrated an incomplete block and the oil column deviated to the left in both, indicating the main mass of the cyst occupied the right side of the

spinal canal. No communication with the subarachnoid space was demonstrated in the first case but was in the second. Both patients were men, one 43, the other 32 years of age. Chief symptom was weakness in both legs, the right more than the left, with atrophy and fibrillations of the thigh muscles. Both patients had right sided ankle clonus and Babinski sign.

An interesting finding in the present case was the fluroscopic demonstration of communication between the cyst and the subarachnoid space through the defect in the left D12 nerve root which permitted the pantopaque to enter the cyst on myelography. Another observation was the return of strength and muscle volume in the right thigh following removal of the spinal cord pressure of long duration. This suggests that the muscle atrophy was due to compression ischemia of the motor cells causing reversible trophic changes rather than destruction.

Origin of the Extradural Cyst

These cysts are considered to be congenital and should be referred to as congenital spinal extradural cysts. They should not be confused with acquired or secondary cysts which develop from trauma to the meninges,[27, 39] or from infection (parasitic cysts). Schmidt [55] and later Elsberg *et al.*[15] considered the extradural cyst to be a "congenital diverticulum of the dura, or a protrusion of the arachnoid through a congenital defect or weak place in the dura." The dural defect is located most often in the region of the dural sleeve of the spinal nerve root. However, it may also occur in the midline. In every case reported since Elsberg's publication, the authors have attempted to verify or disprove this theory. The evidence to justify this etiology is a demonstration of a direct communication between the cyst and the subarachnoid space through a dural defect. This was first reported by Good *et al.*[19] Communication

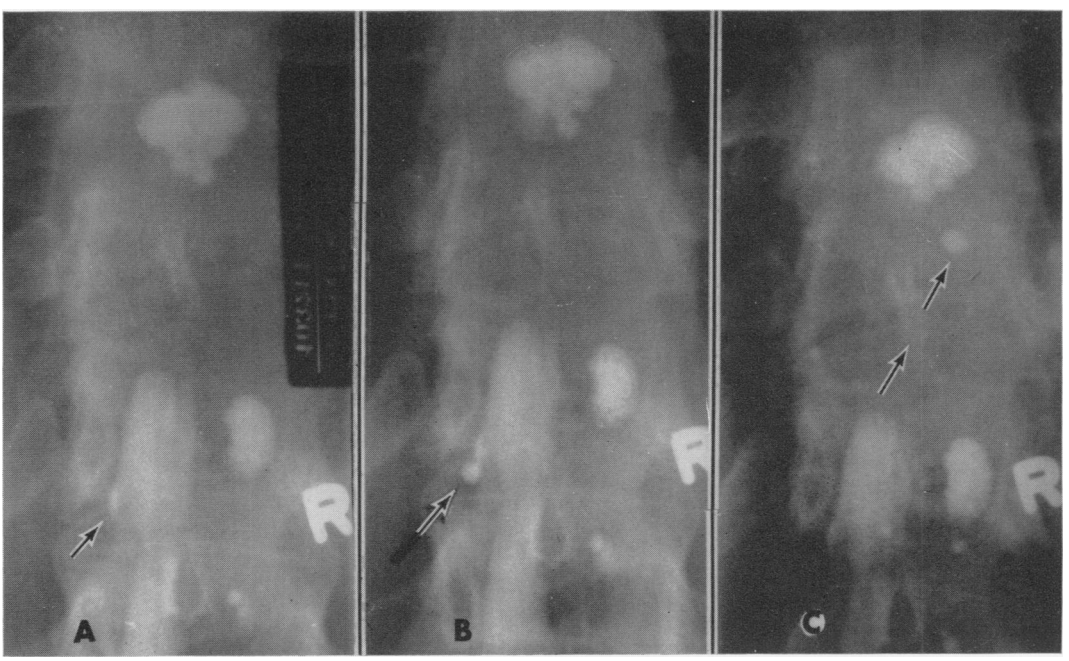

FIG. 2. Three frames from fluoroscopic cineradiography of myelogram: A. Pantopaque filling sleeve of left 12th thoracic nerve root. B. Globule of oil collecting in the dural defect demonstrating communication between the subarachnoid space and cyst. C. Globule flowing cephalad within the cyst.

TABLE 1.

Case No.	Date	Author (Bibliog.)	Age	Sex	Symptoms	Location	Commun.	Kyphosis	Comment
1	1904	Schmidt[55]	16	M	18 mo	D4–D8			
2	1908	Nonne[42]	26	M	2–4 yrs	D7–D10			"Sausage" cyst
3	1915	Collins & Marks[8]	15	M	9 mo	D4–D9		Yes	bedridden 4 yrs
4	1926	Dandy[10]	27	M	7 yrs	D7–D8			
5	1932	Mixter[31]	26	M	13 yrs	D3–D7		Yes	Multioccular
6	1933	Rogers[50]	25	F	2 yrs	D6–D8	No		
7	1934	Elsberg *et al.*[15]	15	F	2 yrs	D3–D6			
8	1934	Elsberg *et al.*	12	M	3½ yrs	D5–D12	No	No	
9	1934	Elsberg *et al.*	15	M	3 mo	D6–D9		Yes	
10	1935	Lehman[32]	12	M	3½ mo	D6–D8	Yes	Yes	
11	1936	Lehman	17	M	9 mo	D6–D10		Yes	
12	1936	Peet & Kahn[47]	12	F	2 mo	D9–L4		Yes	Vasomotor changes
13	1937	Cloward[5]	43	M	3 yrs	D10–L4	No	No	
14	1937	Cloward & Bucy[6]	20	M	4 yrs	D7–D9	No	Yes	
15	1937	Kelly[29]	15	M	5 mo	D5–D8	Yes	Yes	
16	1938	Haffner[24]	19	M	10 mo	D6–D10	No	Yes	Paraplegic
17	1939	Robertson & Graham[49]	14	M	6 mo	D8–D11		Yes	
18	1939	Turnbull[68]	14	F	3 mo	D6–D11	Yes	Yes	
19	1941	Adelstein[1]	15	F	5 mo	D5–D7		Yes	
20	1942	Gross[23]	14	M	1 mo	D4–D9			
21	1942	Hartmann[26]	14	M	1½ yrs	D6–D9	Yes		
22	1942	Mayfield & Grantham[39]	16	M	3 yrs	D6–D9	Yes	Yes	
23	1942	Mayfield & Grantham	26	M	12 yrs	D5–D8	No	Yes	
24	1944	Good *et al.*[19]	42	M	8 yrs	D8–D1	Yes	Yes	
25	1945	Cohen[7]	48	F	3 mo	D8		No	
26	1945	Shenkin *et al.* [58]	51	F	3 mo	D7–D9	No	No	
27	1945	Shenkin *et al.*	13	F	2 yrs	D6–D8	No	No	
28	1946	Hyndman & Gerber[27]	13	M	8 mo	D4–D10	No	Yes	
29	1947	Swanson & Fincher[63]	43	F	—	D11–L2		No	
30	1947	Swanson & Fincher	20 mo	F	4 mo	L2–S1	No	No	Large midline cyst
31	1947	Turner[69]	11	M	5 mo	D6–D11	Yes	Yes	Improved after lumbar punct.
32	1948	Olsson[45]	13	F	1 yr	D9–D11	No	Yes	
33	1948	Roques *et al.*[51]	14	M	11 mo	D6–D9	No	Yes	
34	1948	Dutoit[14]	42	F	15 yrs	L1–2–3	Yes	No	3 separate cysts
35	1949	Decker& Livingston[13]	31	F	6 yrs	L3	Yes	No	
36	1949	Davis[12]	18	M	9 mo	D6–D8	Yes	No	
37	1949	List[34]	12	F	1 yr	D4–D7	Yes	No	Negro
38	1949	Hamlin[25]	25	M	2 yrs	D12–L2		No	
39	1950	Schreiber & Nielson[56]	27	M	2½ yrs	L2–L3	Yes	No	
40	1951	Zolton & Farao[71]	6	F	1 yr	D4–D6	No	Yes	
41	1953	O'Connell[46]	46	F	6mo	L5–S1	Yes	No	
42	1954	Jacobs *et al.*[28]	32	M	9yrs	D6–D8	Yes	Yes	Filled with myelogram
43	1954	Strully & Heiser[61]	37	M	2 yrs	S2	Yes	No	
44	1954	Strully & Heiser	43	F	—	S3	Yes	No	
45	1954	Strully & Heiser	57	F	Short	L5–S1–2–3	Yes	No	
46	1955	Wise & Foster[70]	29	M	16 yrs	D11–L2	No	No	
47	1955	Troupp[67]	42	M	11 yrs	D7–D10	Yes	No	Large comm. of root
48	1955	Schurr[57]	31	M	4 yrs	L5–S1–2–3	Yes	No	
49	1955	Luyendijk[37]	14	F	4 mo	D5–D7	Yes	Yes	Oil entered cyst
50	1955	Luyendijk	9	M	short	L1–L4	Yes	Yes	
51	1956	Cuneo[9]	48	M	2 yrs	D5–D8	Yes	No	
52	1956	Marques *et al.*[38]	10	M	2 mo	D5–D8	Yes	No	
53	1956	Lombardi & Passerini[35]	15	M	15 yrs	D6–9 & D11	Yes	No	Double cyst

TABLE 1.—(*Continued*)

Case No.	Date	Author (Bibliog.)	Age	Sex	Symptoms	Location	Commun.	Kyphosis	Comment
54	1956	Lerman[32]	17	M	3 yrs	D8–D12			
55	1956	Lerman	23	M	1 yr	D9–D11			
56	1957	Myslivy & Klavis[41]	50	F	2 mo	D5–D6			Small cyst
57	1958	Balestrieri[2]	19	F	1 yr	D1–D10	Yes	Yes	
58	1958	Garcin *et al.*[17]	6	M	1 yr	L1–L4	No	No	
59	1958	Norman *et al.*[43]	46	F		D5–D9	No	No	Negro
60	1958	Smith & Chavez[59]	41	F	5 mo	D12–L3	No	No	
61	1959	Nugent *et al.*[44]	29	F	4 yrs	L1–L4	Yes	No	3 midline cysts
62	1959	Nugent *et al.*	43	F	7 yrs	D6–D10	No	Yes	
63	1959	Nugent *et al.*	50	M	28 yrs	C7–D1	No	No	
64	1959	Nugent *et al.*	13	M	3 yrs	D8–D9	No	Yes	
65	1959	Nugent *et al.*	28	M	7 yrs	D8–D10	No	No	
66	1959	Nugent *et al.*	46	M	5 yrs	D8–D10	No	No	
67	1959	Nugent *et al.*	14	M	2 yr	D6–D8	Yes	Yes	
68	1960	Svehla *et al.*[62]	22	F	8 yrs	D11–L2			8 yr remission sudden paraplegia after fall
69	1960	Brungraber[4]	7	M		L1–L2	No		
70	1961	Strang & Tovi[60]	13	F	14 mo	D6–D12 & D10–D11	No	Yes	Double cyst midline
71	1961	Gosinki[22]	50	F	6 yrs	D10–D11	No	Yes	
72	1961	Gosinki	14	F	2 yrs	D6–D8 D8–D10	Yes No	Yes	
73	1963	Gortvai[20]	53	M	7 mo	C3–C5	Yes	No	
74	1963	Gortvai	23	F	2 yrs	C1–C4	Yes	No	
75	1963	Gortvai	40	M	2 yrs	S2–S4	Yes		Midline
76	1963	Gortvai	64	M	10 yrs	D10–L2	Yes		
77	1963	Gortvai	33	M	3 yrs	S2–S3	Yes		
78	1963	Dastur[11]	15	M	6 wks	D7–D11	Yes	Yes	Double cyst
79	1963	Dastur	40	M	8 yrs	D11–L3	Yes	No	Filled with myelogram
80	1963	Dastur	19	M	14 yrs	D6–D10	Yes	Yes	
81	1963	Rosenblum[52]	45	M	2 yrs	D12–L1	Yes	No	
82	1963	Lombardi & Morello[36]	15	M	2 mo	D6–D9	No	No	
83	1963	Lombardi & Morello	58	F	2 yrs	S3	Yes	No	
84	1963	Lombardi & Morello	32	M	1 yr	S1–S2	Yes		
85	1964	Fried *et al.*[16]	32	F	9 yrs	L1–2	No	No	
86	1964	Fried *et al.*	17	M	11 yrs	D3–D6	Yes	Yes	Symptom improved after L.P.
87	1964	Fried *et al.*	16	F	2 yrs	D7–D10	No	No	
88	1964	Fried *et al.*	12	M	3 yrs	D4–D7	No		
89	1964	Glasauer[18]	71	F	2 yrs	L1–L2	No	No	10 yr x-ray
90	1967	Kronborg[31]	13	F	2 mo	D4–D12	Yes	Yes	4 cysts
91	1967	Gortvai[21]	21	F	10 yrs	D2–D12	Yes		2 operations
92	1968	Cloward	32	M	4 yrs	D10–L2	Yes	No	

was mentioned and considered to be present in 43 cases. In 46%, therefore, communication was verified by opaque oil entering the cyst on myelography, as in the present case and cases 26, 42, 44, 49, 51, 78 and 80 or demonstrated at operation (cases 47 and 49).

The congenital dural defect is located in most patients at the termination of the dural sleeve of the nerve root at its exit from the intervertebral foramen, or at the junction of the sleeve and the dural sac. The work of Rexed[48] indicates that these areas are common sites for developmental anomalies. A study of microscopic sections of human spinal nerves and nerve roots

demonstrated "proliferation of the root arachnoidea connected to small cysts in the roots themselves, and also proliferative changes in the dense dural sheath surrounding the root." Cases of multiple cysts have been reported in which small cysts were found within the sheath of a nerve root at the level of the larger lesion (34 and 80) or a second cyst removed from it (53).

Eight cases have been reported in which the cyst arose from a defect in the dural sac in the midline (30, 50, 52, 61, 70, 74, 75, 78). It is reasonable to assume that the midline cysts are also congenital in origin, since they all communicate with the subarachnoid space. Most of these are located in the lumbar or sacral region (50, 61, 75), but have been found in the cervical (74) and thoracic dura (52). An incomplete midline fusion of the embryonic mesenchymal structures surrounding the neural tube, sometimes referred to as *dysraphysm,* may leave a weakened dura and predispose to future cyst formation. A single midline opening may permit herniation of thin arachnoid which forms the wall of a cystic sac (61, 75) and may be mistaken for a meningocele. However, it is not difficult to differentiate anatomically between these two lesions. As in Case 30 Swanson and Fincher's [63] 20-month-old patient, the large midline lumbar cyst was not associated with spinal bifida, lipoma, anomalies of the cauda equina, or other lesions found with a meningocele. Other congenital anomalies in patients with extradural cyst have been reported. Case 57 had atresia of foramen of Luscha and other bony malformations, Case 74 whose cyst was at C1-C5 had anomalous odontoid. And case 70 with a lumbar cyst had an absent arch of Atlas.

Mechanism of Formation and Enlargement of the Cyst

If these lesions are congenital, they must appear early in life as a small diverticulum of the arachnoid. The rate of growth and enlargement of the cyst varies considerably. They may grow very large in a short time causing symptoms early in life or may not appear until late in life. Whether the lesion has been present for 40 years, asymptomatic (88), or whether the cyst formed initially late in life cannot be determined. The latter is suggested by Case 56 a small cyst with symptoms of 2 months duration was removed from a 50-year-old woman.

There is marked variation in the rate of progress of symptoms once they appear. This has led to speculation on the mechanism of enlargement. Three factors are thought responsible: (1) hydrostatic pressure of the cerebral spinal fluid; (2) osmosis of water into the cyst and (3) secretion by the cyst wall.

1. **Hydrostatic Pressure.** As long as there is free communication between the cyst and the subarachnoid space the pressure within the cyst cannot rise above the pressure of the cerebral spinal fluid. This was pointed out long ago by Schmidt.[55] The hydrostatic pressure of the cerebrospinal fluid would not alter these relationships providing there is no interference with free circulation of spinal fluid from compression of the subarachnoid space by the cyst. Should this occur and the communication between the cyst and the subarachnoid space become narrow or partially obliterated, the velocity of flow at the constriction would increase in the direction of the subarachnoid space similar to the common suction pump connected to a running water tap.[20] This dynamic pressure of circulating spinal fluid would tend to empty the cyst rather than inflate it.

O'Connell [46] on the other hand ascribes the distention of the cyst to the pulsating nature of cerebral spinal fluid. He believes that communication between the cyst and the subarachnoid space may be valvular rather than a simple opening. In the erect position fluid enters the cyst under greater hydrostatic pressure. When the patient is

recumbent, the inflated cyst may cause some degree of spinal compression. Temporary increase in spinal fluid pressure by coughing and straining may fill the cyst with fluid which becomes trapped by the valvular opening. The pressure within the cyst would rise to the sum of the static pressure and the hydrodynamic pressure, and result in progressive enlargement or rapid distention of the cyst with cord compression. In Case 38 an airplane pilot would develop transient paraplegia each time he would pull his plane out of a dive. In Case 4, the patient had intermittent attacks of paraparesis always brought on by straining at stool. In Case 49, the cyst filled with oil during myelography but after the pedicle was cut and the cyst removed, its fluid content could not be evacuated by compression, indicating a valve-like action which permitted filling but not emptying. This was suggested as a cause of the rapid onset of paraplegia in this patient. The ball-valve mechanism has been referred to as an *internal meningocele.*[34] The cyst remains patent if greater hydrostatic pressure is applied to the communication between the cyst and the subarachnoid space. This explains why lumbar and sacral cysts attain larger size and are present for a longer period of time before producing symptoms. In the thoracic region where spinal fluid pressure in the upright position approaches zero, the channel of communication probably closes early by proliferation of arachnoidal cells. Then by osmosis, pressure within the cyst increases causing early spinal cord compression.

2. **Osmosis.** Osmosis of fluid through the semi-permiable membrane of the cyst wall may act as an adjunct to the enlargement of the cyst. Most authors indicate the fluid content of the cyst to be clear and colorless, the same as spinal fluid. Turner [69] record laboratory studies on the cystic fluid compared to cerebral spinal fluid and found nitrogen, potassium and calcium almost the same. Other authors indicate finding xanthochromic fluid in the cyst with increased protein content. In one case (75), the protein content was 750 mg./100 ml. In this case a valvular opening in the cyst was demonstrated at operation. The osmotic action of passage of fluid from the lesser to the greater constentation would be apparent.

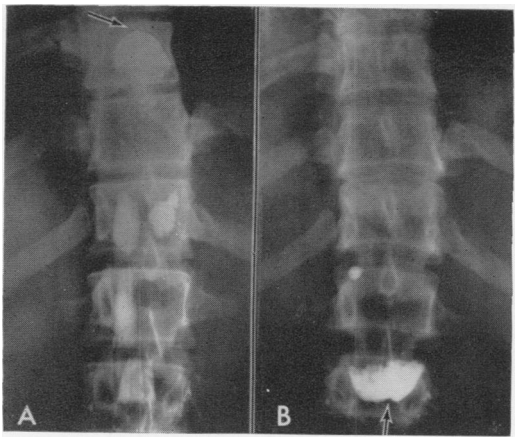

FIG. 3. After myelogram, opaque oil in the cyst outlines the upper pole (A) and lower pole (B). Note in B severe pedicle atrophy from D10 to L2.

3. **Secretion of the Wall.** The wall of the cyst in most cases is composed of acellular fibers of connective tissue and have a lining of a single layer of flat cells resembling the endothelium of the arachnoid membrane. It is conceivable and has been suggested that these cells have secretory powers. This mechanism of enlargement of the cyst cannot be proven or disproved from existing data. One cyst (75) was not removed at operation, only the pedicle ligated. Clinical recovery without recurrence in this case indicates no refilling of the cyst from internal secretion.

Discussion

In the 64 years since Schmidt's case 64 authors have disclosed etiological, pathological and clinical information sufficient to establish this lesion as an entity.

Age, Sex, and Race. Genetic factors associated with these congenital lesions have not been determined, but that cysts occur

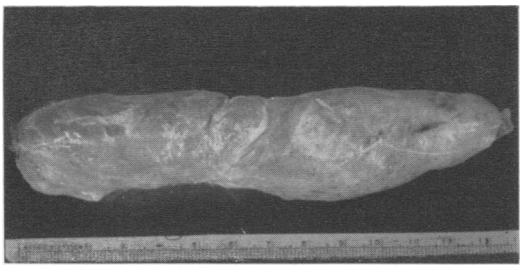

FIG. 4. Large extradural cyst, wall composed of thin arachnoid-like membrane. Note silver clip applied to pedicle of cyst on nerve root. (Size and shape of cyst reconstructed by injecting warm gelatin.)

almost twice as often in males than females may have some significance. There were 56 males and 36 females. The ages of patients are recorded as of the time when the lesion was first recognized or operated upon. The extremes are 20 months (30) and 71 years (89) and cysts have been found in every decade. Adolescents are most frequently afflicted: 44 (48%) were under 20; the second and fourth decades had about equal numbers, 15 (16%) (Fig. 5).

The race reported in six cases (37, 59), was both young Negro females and (18, 79, 80) Indians, and present case was Japanese (92). All others, assumed to be Caucasian are reported by Slavic,[33, 41, 71] Northern [23, 45] and Southern European [88] countries as well as English. This indicates that the lesion occurs in all races.

Location of the Cyst. In early reports the lesions were generally thought to be confined to the mid-thoracic lesion and to occur primarily in adolescence.[14] The finding of a large lumbar cyst in an adult [5] indicated that the lesion can occur elsewhere. Subsequent reports verified the fact that these cysts occur at all levels of the spinal axis from the first cervical to the fifth sacral: 60 (65%) were located in the thoracic region; 11 (12%) were thoracolumbar; 12 (13%) lumbar and lumbosacral; 6 cases (6.6%) sacral and three cases (3.3%) were located in the cervical canal (Fig. 6).

The location of the cyst in relation to the dural attachment indicated that the majority of lesions arise in a nerve root and are either lateral, posterior-lateral or posterior. Only one cyst originating from the nerve root had expanded anterior to the dural sac (72). There were eight in which the cyst had its origin in the midline.

The size of the cyst varies. Some were reported by fluid content, ranging from a few cc. to 300 cc. Measurements of width and length of the lesion were not recorded in most cases, the lesion only being referred to as small or large. The size of the cyst could not be determined by the number of segments of the spinal canal which it occupied because of variation in size of the spine due to age. One lesion (88) extended four segments of the spine (D4 to D7), and was reported as *thumb size* but it was in a boy 12-years-old. The present case also occupied four segments of the thoracolumbar spine in an adult and was 14 cm. long.

Multiple Lesions. In six cases more than one cyst was demonstrated by myelography or identified at operation. Three were reported to have double lesions. In Case 53

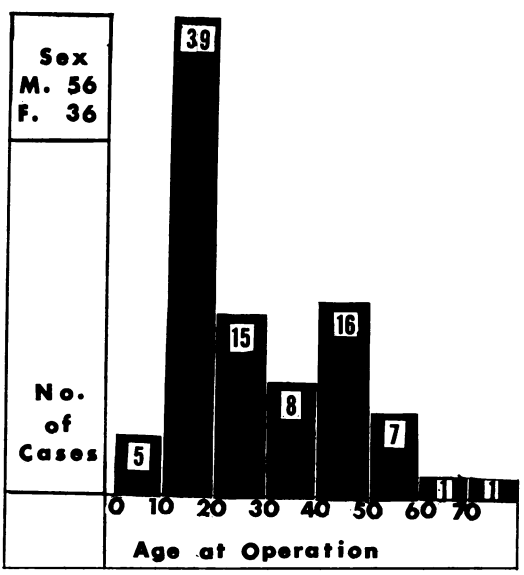

FIG. 5. Chart to show age distribution of 92 cases.

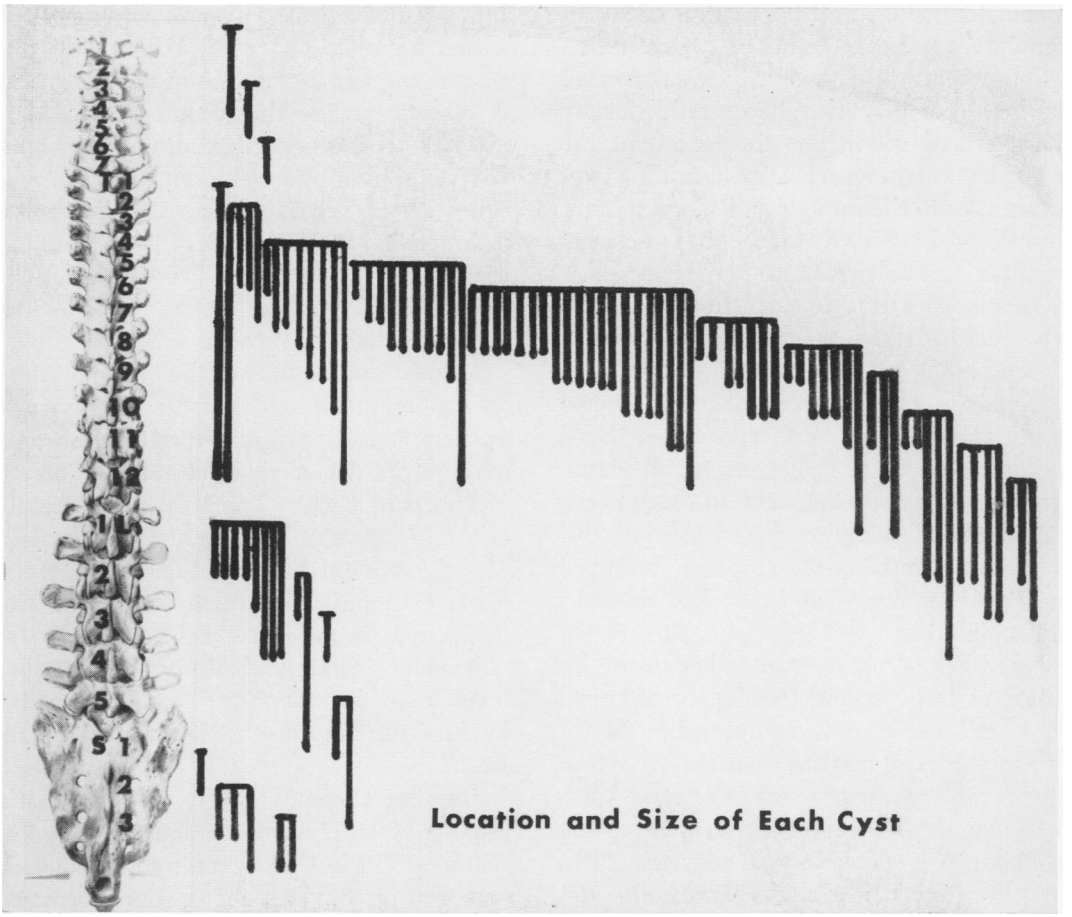

FIG. 6. Each reported cyst is represented by a black line showing size and location in spinal canal.

there was a large cyst at the D7, 8, 9 and a second small one protruding from the nerve root of D11 enlarging the foramen and eroding the vertebral pedicle. Case 78 had a double thoracic cyst and Case 70 had two large cysts superimposed. The larger one, D6 to D12, was collapsed and compressed by the smaller D9, 10 and 11 lesion which was distended and dorsally situated.

Two cases were reported with three cysts: Case 34, three lesions arose from three lumbar nerve roots each protruding through the intervertebral foramen, all visualized by myelogram. In Case 61, 3 small midline cysts at successive levels were found each being 1 to 2 cm. in diameter.

In Case 90, 4 cysts were encountered, at D4 to D12, D4 to D8, D7 left and D10 right. All cysts were attached to nerve roots and all communicated with the subarachnoid space. In Case 5 the cyst was reported as multioccular.

Clinical Features. The history of symptoms may be of short duration or may cover a period of several years. The shortest history was one month (20). The longest 28 years (63), the average about 3½ years. In the thoracic region symptoms were present in most patients for only a few months, however, several had prolonged symptoms (23, 76, 80, 86). The average period in 60 thoracic cases was 16 months. In those with lumbar cysts the period of symptoms averaged nearly 5 years and in sacral lesions 2

years. Remissions and fluctuation of symptoms, were recorded in about 30% of cases.

Symptoms were primarily weakness of the legs, difficulty in walking, mild sensory changes and often paresthesias about the trunk, back pain with root pain in the lower extremities in the lumbar and sacral lesions. Bowel and bladder disturbances were most frequent in these patients but was present in nearly 20% of all cases. Impairment of bowel and bladder function was frequently absent even in large lesions. Periods of temporary bladder paralysis and loss of libido and potency were reported in sacral lesions. Pain is a symptom in all cases, ranging from dull back ache to sharp nerve root pain radiating over a segmental distribution. Remission of symptoms occurred in approximately ⅓ of cases, but this was not a significant feature.

Neurological changes varied with the location of the cyst and the degree of spinal cord and nerve root compression. Motor signs were more marked than sensory. Paraplegia and paraparesis with marked spasticity clonus and Babinski sign was present in about 70% of cases and monoplegia in 10%. Sensory disturbances were mild. Reduction of pain and temperature sensation was present in 75% and vibratory and position sense was impaired in 35%.

Cervical Cysts. Cervical cysts are rare, only three reported.[49] The *first recorded cervical cyst,* Case 63 occurred in a man aged 50 with a 28-year history of progressive weakness and numbness, and finally a claw hand. Extensive arthritic changes in the lower cervical vertebra and a positive spinal fluid Wassermann test delayed the correct diagnosis for over 5 years. The 4 × 1 cm. cyst extended from C7 to T1. *The second cervical* case (73) a 53-year-old woman with symptoms of weakness and stiffness of the legs for 7 months had a multioccular extradural cyst from C3 to C5 with the inner wall firmly adherent to the dura. Only the outer wall was removed. The patient made an excellent recovery. *The third case* was a woman aged 23 (74) with a 2-year history of weakness and stiffness of the left extremities and impairment of sensation up to the second cervical dermatome. A large cyst extending from C1 to C5 was shown by myelogram to communicate with the subarachnoid space. Extensive destruction of bone by the expanding lesion and laminectomy required subsequent cranio-cervical fusion. The patient made an excellent recovery.

The clinical features of the cervical lesion are upper motor and sensory involvement of the upper extremities. Horner's syndrome may be present in lower cervical lesions.

Thoracic Cysts. Thoracic cysts usually occur in young adolescents who complain of progressive weakness and stiffness in the lower extremities. Pain is not common, and if present is mild backache. Sensory involvement is minimal although a sensory level may be demonstrated. Progressive kyphosis of the mid-thoracic spine is common.

Lumbar Cysts. The thoraco-lumbar and lumbar cysts [59] have characteristic clinical features. The lesion occurs in fourth decade and symptoms last for years. Localized weakness and atrophy of lower motor neuron type occurs in the lower extremities. In upper lumbar lesions, pain is not a prominent symptom but the lower lumbar cysts cause nerve root pain with paresthesias and dermatome sensory loss. These symptoms may lead to a mistaken diagnosis of a ruptured lumbar intravertebral disc. Relief of pain and reversal of neurological changes follows removal of the cyst.

Sacral Cysts. The clinical feature of extradural cyst located in the lumbosacral and sacral canal is primarily pain, located in the lower back and radiating along the course of the sciatic nerve. This cannot be differentiated from pain associated with lumbar disc lesions, cauda equina tumors, spondylolisthesis and tuberculosis of the sacroiliac region. Disturbance of peroneal sensation and micturition associated with

the sciatic syndrome may suggest this lesion. Erosion of bone seen in plain roentgenograms rules out disc-protrusion alone as unlikely. A myelogram demonstrates the cystic nature of the lesion as all sacral cysts communicate with the subarachnoid space and may be mistaken for meningoceles.

Perineural cyst described by Torlov [64] must be included in the differential diagnosis of sacral extradural cysts. These lesions arise in the space between the perineurium and endoneurium at the junction of the posterior nerve root with the dorsal root ganglion. The cyst surrounds the nerve root and always contains nerve fibers and ganglion cells. Communication with the subarachnoid space is never free and is usually not present. The cysts are often multiple and erosion of bone rarely occurs. They are frequently asymptomatic and a coincidental finding on myelogram and autopsy. When symptomatic, they produce the same pain as the extradural cyst.[65]

Changes in the Spine Caused by the Cyst. Bony erosion in the spine in all cases is caused by intermittent hydrostatic pressure within cysts of long standing and slow expansion. When the cyst attains a large size the expanding pressure is transmitted equally in all directions eroding the lamina posteriorly, the vertebral pedicles laterally and the posterior surface of the vertebral body anteriorly. In smaller lesions which arise laterally in the spinal canal, erosion of one or two pedicles unilaterally may be the only bone change. The rate of growth of the cyst is demonstrated in Case 89. Two x-rays of the lumbar spine taken ten years apart showed progressive destructive changes in only one vertebral pedicle. Pedicle erosion is the most common finding. In the antero-posterior x-ray they may be completely destroyed or appear as a narrow strip of bone. The interpedicular distances may be as much as 10 to 12 mm. wider than normal. Maximum bony erosion is always at the approximate center of the cyst where pressure has been present for a longer time. The number of eroded pedicles is not always an indication of the size of the cyst as the tapered ends of the lesion may extend 2 or 3 segments above and below without eroding bone (28, 31, 88).

In lateral x-rays the antero-posterior diameter of the spinal canal is increased due to atrophy of the laminae and vertebral bodies. To quote from operative report of the author's original case, "It was very interesting to look into this huge cavity and see how the bodies of the vertebra had been eroded so that the intervertebral discs stood out like ridges between them." The bone is worn away but the resilient intervertebral disc is unaffected.

Sacral extradural cysts are usually small and cause no bone erosion. In large lesions a deep concave erosion of the posterior surface of the vertebral bodies of the sacrum and 5th lumbar may occur (48).

Kyphosis. Bony changes in the anterior one-third of the vertebral bodies opposite the cystic lesion was first described by Lehman.[31] Erosion of the vertebral end plate and subcortical bone gave the appearance of vertebral epiphysitis described by Schuerermann [53] and Blum.[31] Cloward and Bucy [6] believed that these bony vertebral changes which lead to kyphosis was due to venous stasis caused by pressure of the cyst on the intraspinal epidural veins. Postoperative progression of kyphosis is due to softening and collapse of the anterior third of the vertebral body. Also following laminectomy, lessened resistance to movement may occur in the spine. This is frequently encountered in the cervical region and according to Shankin, Horn and Grant [58] postoperative kyphosis is not rare in young persons after dorsal laminectomy for any cause.

Most authors recorded the presence of kyphosis in patients with extradural cysts. Of 71 thoracic cysts, 34 (48%) were reported as causing dorsal kyphosis. In few of these, however, were the bony changes described as characteristic of vertebral

epiphysitis. Dorsal kyphosis was reported in one lumbar case (50). It is not certain, therefore, whether the high percentage of reported kyphosis dorsalis (nearly 50%) was true kyphosis dorsalis juvenilis or postural changes following laminectomy.

Since 1937 [5] every patient the author has operated upon for an expanding intraspinal lesion was studied to see if vertebral epiphysitis was associated with lesions other than extradural cysts. No case has been found. This indicates that vertebral changes are peculiar to the extradural cyst and occur only in the thoracic region in the vertebra opposite the cyst. Epidural veins here are more easily compressed due to lower venous pressure and the small spinal canal. This impairs the circulation and nutrition to the vertebral epiphyseal cartilage of the adolescent before growth of the vertebral body is complete. In the lumbar spine, vertebral body lesions are not found because the canal is large and the cauda equina is easily compressed so the circulation to the vertebral body is not impaired.

Myelograms on 51 patients, in 26 showed complete block, and in 25 showed deformities in the oil column. Direct filling of the cyst with the oil was demonstrated in 15 (79, 78, 51). Only one (49) report of "seeing the oil drip into the cyst," visualized the exact location of the communication.

Differential Diagnosis. In addition to other space taking lesions, multiple sclerosis must be considered in the differential diagnosis, particularly, in cases with remissions. This mistaken diagnosis was made in Case 68. The patient had a complete remission for 6 years delaying operative removal of the lesion.

Treatment. The only treatment is surgical removal of the cyst by laminectomy. In most cases the cyst was totally removed in a single operation. In four cases a second operation was required before the cyst was located. One patient with multiple cysts relapsed and required four operations. In one patient, the cyst was so large its removal required a two-stage procedure (91). In another (73) the inner wall of the cyst was adherent to the dura and only the posterior wall was removed. In a patient (75) with a large sacral lesion, the communication with the subarachnoid space was closed and the lesion left intact, and not removed.

Large lesions extending over five or more vertebral segments (8, 13, 20, 28, 31) require extensive laminectomy particularly in the cervical and thoracic region and may result in anterior subluxation or kyphosis of the spine (58). To obviate these postoperative changes, the length of laminectomy should be kept at a minimum. It may not be necessary to remove the laminae until the upper and lower end of the cyst is exposed. The loose fibrous strands attached to the dura can be dissected free and the end withdrawn from beneath a lamina. In this manner one or two laminae may be saved. With extensive bone removal, a posterior spinal fusion should be included in the operation, or a secondary fusion procedure may be required (72, 68). A hyperextension brace is recommended after removal of large thoracic extradural cysts in adolescents.[13, 25] If changes of epiphysitis are present in the thoracic vertebra, the brace should be worn until spinal lesions are healed or the vertebral body growth is complete.

Summary and Conclusions

A case of a large congenital extradural cyst in the thoraco-lumbar region is reported, and demonstrates unusual clinical and x-ray findings. A review of the world literature for the past 64 years disclosed 103 cysts in 91 cases reported by 64 authors. A study of these cases shows that this lesion is a definite entity with characteristic, clinical findings and roentgenographic changes in the spine.

The lesion develops from an arachnoidal herniation through a congenital defect in the spinal dura at or near a nerve root or

in the midline. Progressive enlargement of the cyst is due to communication with the subarachnoid space, which is demonstrated by myelography or at operation in 50% of cases. Cysts occur at all levels of the spine. Seventy-seven per cent are in the thoracic region. Symptoms are of short duration in thoracic lesions and prolonged in other areas. Clinical features result from nerve root or spinal cord compression and vary with the location of the cyst. Changes in bone consist of erosion of the walls of the spinal canal by the expanding lesion; thin laminae, narrow pedicles and concave vertebral bodies. Epiphysitis from ischemia may occur in the anterior third of the bodies of the thoracic vertebra opposite the lesion, producing associated kyphosis dorsalis. Laminectomy and removal of the cyst is the only treatment.

Addendum

Since this paper was submitted for publication, Bergland (J. Neurosurg., **28**:495, 1968) has reported 3 cases of extradural cysts in one family, suggesting the lesions to be hereditary and familial, as well as congenital. This increases the total of reported cysts to 106 in 94 cases.

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