Respiratory Arrest and Cervical Spinal Cord Infarction Following Lumbar Puncture in Meningitis

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SUMMARY: *A 6-year-old child with meningitis had a respiratory arrest 20 minutes after a lumbar puncture. Thereafter she required maintenance on a ventilator, had a flaccid quadriplegia, and died 12 days later. Necropsy showed infarction of the central portion of the cord at the level of the decussation of the pyramids. The suggested mechanism of damage is compression of the arterial supply to the cord at the level of the foramen magnum by herniated cerebellar tonsils; concomitant hypotension may have contributed to production of the damage. Four similar cases, who survived with residual deficit, have also been reported. Other separate mechanisms by which the cord can be damaged in meningitis are vasculitis, thrombosis and arachnoiditis.*

RÉSUMÉ: *Un enfant de 6 ans souffrant de méningite fut victime d'un arrêt respiratoire 20 minutes après une ponction lombaire. Le tout fut suivi d'une quadriplégie flasque nécessitant un respirateur, mais l'enfant mourut 12 jours plus tard. L'autopsie montra un infarcissement de la portion centrale de la moelle au niveau de la décussation des pyramides. Le mécanisme pathogénique semble être une compression de l'apport artériel à la moelle au niveau du foramen magnum par des amygdales cérébelleuses herniées; une hypotension concomittante peut avoir contribué à la production de la lésion. Quatre ces semblables ayant survécu, mais avec un déficit résiduel, furent rapportés. D'autres mécanismes existent qui pourraient expliquer une telle lésion de la moelle dans la méningite; ce sont les vasculites, les thromboses et les arachnoidites.*

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Spinal cord damage is a rare complication of bacterial meningitis. This case is being reported to draw attention to the fact that infarction of the cervical spinal cord can occur after respiratory arrest following lumbar puncture in meningitis.

CASE REPORT

This six year old girl was well until the night prior to admission when she developed headache and fever. The next day she vomited and was noted to be confused. By the time she was seen in her local hospital she was unresponsive and had a stiff neck. A lumbar puncture showed turbid cerebrospinal fluid. The CSF contained 21,400 leukocytes per cubic mm. and a smear showed gram negative rods. Treatment was commenced with ampicillin and chloramphenicol. Twenty minutes following the lumbar puncture she had a respiratory arrest. She was intubated and ventilated. Blood gases at this time were as follows: 7.37 pH, pCO_2 35 mm.Hg, pO_2 136 mm. Hg.

Three and one-half hours later, she was examined at the Vancouver General Hospital. At that time she was unresponsive to painful stimuli. Her fundi was normal. Her eyes diverged and the pupils reacted sluggishly to the light. Muscle tone was flaccid in all four extremities, deep tendon reflexes were absent and plantar responses were flexor. Blood pressure was 80/60, pulse rate 80 per minute. The right ear drum was slightly red. Peripheral blood leukocyte count was 10,000 per cu. mm. with 75% stab cells. She was treated with dexamethasone and mannitol. Pentobarbital was administered as an initial bolus of 10 mg. per kg. and continued at a rate of 2 mg. per kg. per hour.

Examination two and one-half hours after admission showed that she did not respond to commands. She responded to painful stimuli by opening her eyes. When her mouth and pharynx were suctioned, she blinked. Her corneal reflexes were sluggish, her gag reflex was present, and her facial movements were symmetrical. Intracranial pressure (ICP) monitoring was begun. The first recorded pressures were 18, 20 torr and subsequently remained at less than 10 torr so barbiturates and ICP monitoring were discontinued on the 5th day. CSF cultures grew H. influenza sensitive to ampicillin so chloramphenicol was discontinued.

On the fifth day she was still quadriplegic and required mechanical ventilation. On command she would open her mouth and eyes. She seemed more aware of her surroundings. Her corneal reflexes were brisk, and her gag reflex was present but depressed. On the eighth day her facial movements were weak and swallowing was impaired. On painful stimulation she withdrew her right foot slightly to pain, but could not withdraw her foot when asked to do so.

She was afebrile for the first six hospital days. On the seventh day her temperature started to rise and reached 40.9°C on the 10th day. On the ninth day a chest roentgenogram showed bilateral pneumonia. On lumbar puncture the opening pressure was 13 cm. The CSF was clear and colourless, and contained 52 red blood cells per cu. mm., 9 leukocytes per cu. mm., protein 30 mg/dl and glucose 90 mg/dl. Her peripheral blood contained 24,500 leukocytes per cu. mm. on day 10. On the eleventh day her abdomen became distended, tense, silent and tympanitic. Abdominal roentgenogram showed gas throughout the bowel, with multiple air fluid levels, and air in the bowel wall suggesting a diagnosis of necrotizing enterocolitis. Air was also present in the portal venous system. Her course thereafter was one of continued

deterioration. She died on the twelfth day of her illness, 33 hours after the onset of abdominal distention.

Pathological Findings

Post mortem examination showed slight cerebral edema. Herniation of cerebellar tonsils was not seen on gross examination. Microscopic examination of serial sections of lower brain stem and spinal cord showed discrete areas of infarction in the brainstem. The largest consisted of almost symmetrical areas of infarction throughout the first cervical segment (C_1), involving the pyramidal decussation (Fig. 1). The infarcts appeared to be of the same age. Most of the infarcted tissue had been removed by foamy macrophages, which replaced the parenchyma. A few neuroaxonal swellings were present at the periphery of the infarct. Occasional hypertrophied astrocytes were present in viable tissue surrounding the infarct. Just below the frank infarction, at the C_2 and C_3 levels, there were bilateral neuroaxonal swellings at the subpial surface of the cord, these being damaged corticospinal axons which run on the surface between C_2 and C_4 (Crosbie et al. 1962). Sections of cerebellar tonsils showed infarction with macrophages replacing parenchyma. These were similar to the infarcts in the cord. Sections of the brain and of the cord, including levels T4-7 and L1 showed no infarction, residual meningitis, arteritis, or thrombosis.

General necropsy revealed that the immediate cause of death was necrotizing gastroenteritis. The cause of this was unknown, though treatment with antibiotics, ischemia, and intragastric nutrition with a hyperosmolar solution were all considered theoretical possibilities. Bilateral bronchopneumonia was present.

DISCUSSION

The blood supply to the cervical spinal cord comes from the anterior spinal artery which arises as a midline union of a branch from each vertebral artery. There is general agreement that the upper portion of the cervical cord is supplied from above, the lower portion from below (Fortuna et al, 1971). In this patient, signs and symptoms of increased intracranial pressure were already present, and the lumbar puncture probably precipitated a cerebellar pressure cone, indicated in life by the respiratory arrest. At postmortem examination no gross cerebellar herniation was seen, but the cerebellar tonsils were infarcted. During the 12 days the patient lived after her respiratory arrest, the gross herniation probably had time to recede, leaving the infarcted tonsils as a marker of previous herniation. The herniated cerebellar tonsils produced sufficient compression of structures in the foramen magnum to obstruct caudal flow in the vertebral artery branches which give rise to the anterior spinal artery and thus caused infarction of the upper cervical cord (Fig. 2). Since the infarctions were bilateral, either both arteries were compressed before they united to form a single anterior spinal artery, or perhaps the central arteries, branches of the anterior spinal artery which supply the central portion of the cord (Turnbull, 1971), were compressed rather than the anterior spinal artery. The small discrete infarcts in the dorsal half of the cord may have resulted from compression of the pial arterial plexus.

Hypotension with resultant failure of perfusion of the cord may also have contributed to production of the infarct. The levels of cord usually infarcted due to hypotension and ischemia are T4 (T4-7) and L1 (Lazorthes 1972), which were not involved in

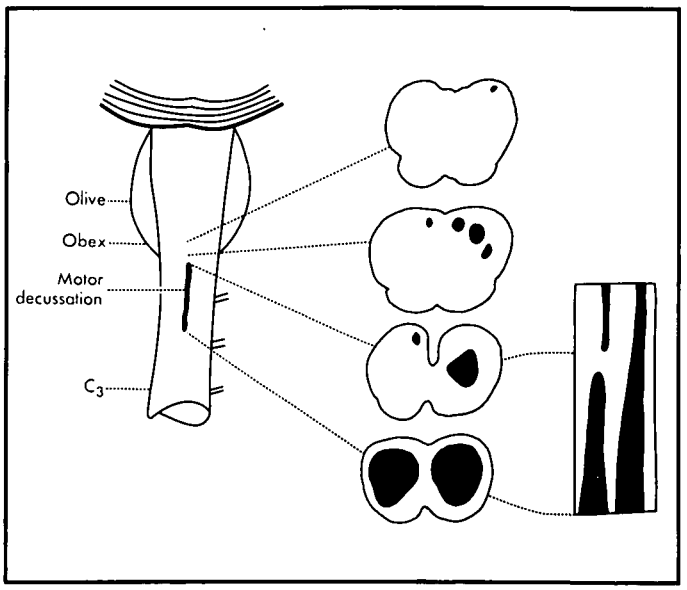

Figure 1 — Distribution of infarcts in brain stem and upper cervical cord. Black areas denote infarction, which involves chiefly the corticospinal tracts as they decussate. Ventral surface of brainstem and cervical cord is depicted on left, cross sectional area of infarct in the centre drawings, and the longitudinal extent of the infarct on the right.

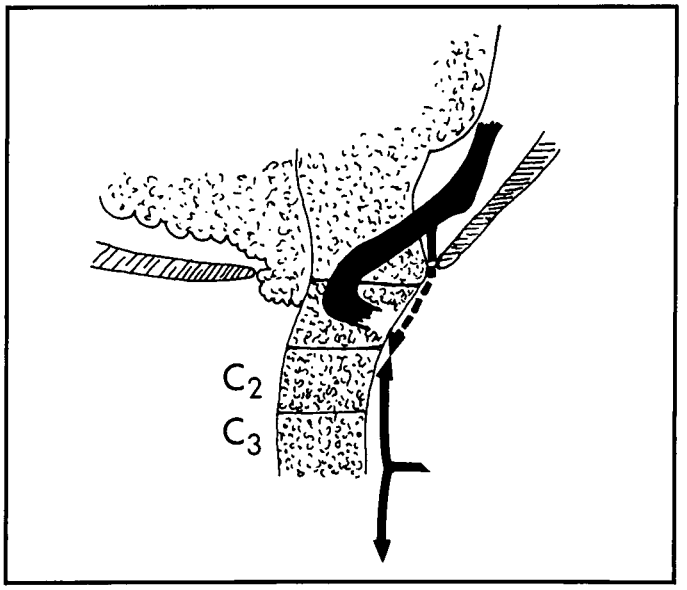

Figure 2 — Proposed mechanism for infarction. Cerebellar tonsils protrude down through foramen magnum and compress cord and descending branches of the vertebral arteries against the bony edges of the foramen magnum. Arrows denote direction of flow. - - - indicates region of abolished arterial blood flow to C1 segment.
(Illustration adapted from Lazorthes 1972)

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this case . Experimental work in dogs indicates that perfusion of the spinal cord is maintained with increased cerebrospinal fluid pressure at arterial perfusion pressures of greater than 50 mm. Hg (Griffiths et al, 1978). Perfusion is maintained when the cord is compressed directly until perfusion pressure drops below 65-70 mm. Hg (Griffiths et al, 1979). In this case compression and failure of perfusion may have combined to produce the infarct, although the blood pressure during the respiratory arrest is not known.

Four patients have been described who survived following an episode of respiratory arrest after lumbar puncture performed early during the course of bacterial meningitis (De Sousa et al, 1978; Tal et al, 1980; Swart and Pye, 1980; see Table I). All patients had headache, vomiting and some degree of lethargy or decrease in their level of consciousness. All four had a lumbar puncture, which confirmed the diagnosis of bacterial meningitis. Within hours of admission, presumably after the LP was performed, each had a respiratory arrest, developed quadriplegia and required mechanical ventilation. In case 4 it is stated that there was "no hypotension" during the period of respiratory arrest; in cases 2 and 3 the arrest is described as "cardiorespiratory". These four cases are probably similar to the present case - that is, meningitis, some early increase in intracranial pressure, coning, and compression of the small arteries at the foramen magnum by herniated cerebellar tissue.

The four cases summarized in Table 2 are also similar in some respects, but did not have meningitis. All had cerebral pathology, all but one (Case 6) had lumbar puncture, one (Case 8) had a respiratory arrest prior to lumbar puncture. All were maintained on a ventilator, and all had small areas of necrosis in the cervical spinal cord which the authors attributed to compression of the vascular supply by large amounts of herniated cerebellar tissue which were found around the spinal cord at necropsy. In these cases it is probably vessels of the pial arterial plexus which were compressed. The blood pressures in these patients at the time of the respiratory arrest were not stated.

Table 3 describes three cases, all of whom had meningitis, a lumbar puncture, no respiratory arrest, and damage to the spinal cord at levels below T1. The mechanism damaging the cord is probably either a vasculitis or arachnoiditis. Case 10 and 11 are probably similar to the two "acute poliomyelitic" cases described by Turner (1948), in which there was sudden onset of a

TABLE I

Reported cases of myelopathy following lumbar puncture on children with meningitis

Case	Reference	Age - Sex (yrs.)	Organism	Time of respiratory arrest	Time on respirator	Clinical Features and Outcome	Postulated mechanism of damage to cord
1	Present report	6 F	H. influenza	20 min after LP	12 days	Level at decussation of pyramids. Flaccid quadriplegia. Death.	Infarct of cervical cord. Compression of anterior spinal artery by herniated cerebellum + hypotension
2	Tal et al (1980) Case 1	3+1/3 M	H. influenza	8 hrs. after admission	6 mon.	Flaccid paralysis of arms, spastic legs. Recovery, residual deficit.	Ischemia due to cardiac arrest
3	Tal et al (1980) Case 2	3+1/2 F	N. meningitidis	3 hr. after admission	NS	Cranial nerve signs. Flaccid quadriplegia. Sensory level below C1. Recovery, residual deficit.	Ischemia due to cardiac arrest
4	Swart & Pye (1980)	15 M	N. meningitidis	7 and 12 hr. after admission	2 wk.	Cranial nerve signs. Quadriplegia below cervico-medullary junction. Recovery, residual deficit.	"Vasculitis"
5	De Sousa et al (1978)	3 F	H. influenza	"shortly after" transfer to referral centre	9 wk.	Cortically blind. Flaccid quadriplegia. Recovery, residual deficit.	"Vascular injury"

NS — *Not stated*

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TABLE 2

Reported necropsy cases of cervical cord infarction attributed to compression of vascular supply to cord by herniated cerebellar tissue

Case	Reference	Age - Sex (yrs.)	Underlying Disease	Interval between LP and respiratory arrest	Time on respirator	Lesion found at necropsy
6	Herrick & Agamanolis (1975) Case 3	12 F	Encephalopathy with fatty liver (Reye's syndrome)	less than 24 hours	96 hr.	Pencil shaped necrosis posterior columns cervical cord
7	Herrick & Agamanolis (1975) Case 5	4 F	Encephalopathy with fatty liver (Reye's syndrome)	? less than 12 hours	61 hr.	Necrosis posterior cervical & thoracic cord
8	Herrick & Agamanolis (1975) Case 6	17 F	End stage renal disease seizures	No LP	53½ hr.	Necrosis cervical cord dorsal and dorsolateral white
9	Herrick & Agamanolis (1975) Case 8	18 M	R. subdural empyema	Prior to LP	56 hr.	Necrosis cervical cord

TABLE 3

Reported cases of damage to the spinal cord due to thrombosis, vasculitis or arachnoiditis in bacterial meningitis

Case	Reference	Age - Sex (yrs.)	Organism	Clinical Features	Lesion. Postulated mechanism of damage of the cord
10	Tal et al (1980) Case 3	3 days M	E. coli	Asymptomatic. Death.	Softening of grey matter thoracic cord and all of lumbo-sacral cord. Thrombosis of anterior spinal artery.
11	Gottshall (1972)	20 M	N. meningitis	Original level midlumbar. Recovery, residual deficit at S3-5.	"Vasculitis". No pathological verification.
12	Haupt et al (1981)	5½ M	D. pneumoniae	Flaccid paraplegia. Sensory level T2. Recovery, residual deficit.	Dense fibrous leptomeningeal adhesions at laminectomy.

flaccid monoplegia during the course of meningitis. These cases likely resulted from either thrombosis, as found in Case 10, or vasculitis. Turner's last two cases and Case 12 are examples of damage to the cord resulting from a chronic arachnoiditis following meningitis.

Gilles and Nag (1971) described anoxic neuronal change or neuronal loss and gliosis in the spinal cords of six children following a cardiac arrest. In all cases the damage was most marked in, or limited to the lumbar cord. In four of these cases no LP was mentioned. One child had a cardiac arrest the day following LP and another (case 4) had a cardiorespiratory arrest during lumbar puncture and at necropsy was found to have widespread neuronal loss and gliosis most marked in the sacral region, decreasing rostrally. These six cases are different from the one here reported. Frank infarction of the cord was absent. Damage was caudal, not high in the cervical cord. These cases represent a mechanism of damage to the cord caused by failure of perfusion alone, without any element of compres-

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sion of vessels to the cord as postulated in this case.

It is interesting that there are so few reports of cases of the type represented in Table 1. There is considerable variability in the blood supply to the spinal cord and possibly some rare and unique arrangement of vessels to the C_1 segment in these patients accounts for the spinal cord infarction at this level. Another factor may be that death usually occurs so quickly after coning that clinical and morphological signs of cord infarction do not develop unless patients are kept alive by mechanical ventilation for a period of time. Of the eight cases reported by Herrick et al (1975), only those four maintained for the longest time on the respirator (a minimum of $53^1/_2$ hours) showed necrosis of the spinal cord at necropsy (Table II).

Infarction of the spinal cord occurring as a complication of L.P. has not been described previously. The extent to which the cervical cord damage in the present case is directly due to the LP is not clear. Cerebral edema occurs in meningitis and perhaps any time there is a cerebellar pressure cone for whatever reason, the situation is such that vascular compression and cord damage can occur. The LP may contribute by producing a more rapid change in pressure which precipitates coning. Horowitz et al (1980) found that the incidence of cerebral herniation of all types was 6% in a clinical study of childhood bacterial meningitis. The herniations occurred within 8 hours of admission, and there were no instances of herniation before lumbar puncture. Apart from the occurrence of headache in 3 - 30% of patients after lumbar puncture, it is difficult to determine the exact incidence of complications of LP. The probable reason for this is that serious complications are rare and depend on factors related to the primary disease for which the LP is performed, particularly on whether or not there is increased ICP. Also, it is not always possible to determine whether the complication is directly due to the LP or to the primary disease.

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