A Congenital Defect in the Spinal Cord of the Manx Cat

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Abstract. The lumbar part of the spinal cords of two Manx kittens showed syringomyelia associated with spina bifida occulta. The syringomyelia may have been caused by a secondary mechanism, such as occlusion of blood vessels, that occurred after formation of the spinal cord. There was no apparent hyperplasia of neural tissue. Incomplete fusion of vertebral arches may also have been caused in part by such a vascular defect.

The term ‘spina bifida’ refers to a group of developmental defects characterized by failure of fusion of vertebral arches with or without protrusion and dysplasia of the spinal cord or its membranes. Past investigators have examined this type of defect in congenital cases; however, as these were usually isolated lesions that were fully formed when recognized, little was added to our knowledge of their etiology. Patten [11, 12], after studying serial sections of human embryos with spina bifida, concluded that the defect was due to an overgrowth or hyperplasia of the cells of the neural tube which hindered closing of the neural tube and adjacent vertebral arches. An older, somewhat less popular theory holds that rupture of the already closed neural tube causes the spina bifida. This latter concept, although overshadowed by the overgrowth theory, has been revived by the work of Gardner [3] and more recently by the review of Padget [10].

In an attempt to examine the development of spina bifida, several workers have induced the defect utilizing a variety of methods: the application of teratogenic drugs such as trypan blue [4, 15], variation in maternal diet [16], and surgical intervention, such as splitting the roof plate of the neural tube [2]. Unfortunately the results obtained from their experiments have also failed to explain the mechanism of the developmental defect.
In a study of congenital anomalies of the lower spine and spinal cord in Manx cats, James et al. [6] described osseous defects such as dysgenesis of sacral and coccygeal vertebrae. In two of nine cats studied, James et al. [6] reported that normal lumbar and sacral vertebrae were present but two caudal vertebrae had a thin membrane in place of dorsal bony arches. Cephalad to this type of lesion the spinal cords displayed a bilateral cavitation in the dorsal white matter extending from the level of lowest sacral to lumbar 4. At its most caudal extension the cavity spread across the entire dorsal half of the cord.

The present report, part of a wider investigation of the Manx cat, describes two kittens with a similar type of cavitation in the lumbosacral spinal cord. Unlike the findings of James et al. [6], however, the bony defect in our kittens was in the lumbar region rather than in the caudal vertebrae.

Materials and Methods

Two female, stub-tailed, Manx kittens were born with cutaneous defects over the lumbar spinal cord. A small amount of fluid exuded through the abnormal skin. With continuing growth it became apparent that both animals had a flaccid paralysis of the hind limbs. It was assumed that the lesions were a form of spina bifida, and the following investigation was undertaken to explore this possibility.

The kittens, aged 6 weeks, were anesthetized by an intraperitoneal injection of 3.5% chloral hydrate and perfused via the aorta with Carnoy’s B fluid. The brains and spinal cords were removed, embedded in paraplast, serially sectioned at 15 μm, mounted on glass slides, and stained with thionin.

Results

Both kittens had basically similar defects and are described jointly. The spinous processes of the two posterior lumbar vertebrae were bifid; however, there was no protrusion of meninges or spinal cord. The remainder of the vertebral column, both above and below this level, was normal. The brains and spinal cords appeared normal as far caudad as the lumbar area. Each spinal cord ended in the sacral area as a flattened, elongated mass, giving the impression of a double cord. The mass was tightly attached to the surrounding dura and bony sacrum.

Microscopically the spinal cords were normal in the cervical and thoracic regions (fig. 1). In the lumbar region, both cords had the beginning of a cavity in the dorsal funiculi close to the midline (fig. 2). Rostrally the cavity in the
Fig. 1. Cross section of normal thoracic spinal cord. Thionin stain.

Fig. 2. Anterior lumbar area of spinal cord. Arrows outline first indication of lesion in dorsal midline. Thionin stain.
dorsal funiculi and the lumen of the central canal were separated by a fibrous
area (fig. 3, 4). Caudally, however, the enlarging cavity and the lumen of the
central canal soon became confluent (fig. 5, 6), and the cord began to flatten
dorsoventrally. This flattening initially gave the impression that the cord was
duplicated, a finding disproved microscopically. With the flattening of the
cord, the cavity once again is separated from the lumen of the central canal
(fig. 6). The dorsal surface of the cavity was continuous throughout its length.

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**Fig. 3.** Cross section of anterior lumbar area with cavity in dorsal funiculi. Thionin stain.

**Fig. 4.** Cross section of middle lumbar cord with enlarged cavity in dorsal funiculi. Thionin stain.

**Fig. 5.** Cross section of posterior lumbar cord. The cavity is now confluent with the central canal. Thionin stain.

**Fig. 6.** Cross section of sacral cord. The cord is flattened dorsoventrally. The lesion is separated from the central canal and is covered dorsally by ectoderm. Thionin stain.
In all areas of the cord the remaining cells appeared normal (fig. 6). Although the dorsal funiculi and most of the dorsal horns were missing, the remaining gray matter had a large number of normal ventral motor horn cells.

Discussion

Two cases of spina bifida occulta associated with syringomyelia or tubular cavitation of the white matter of the spinal cord were studied. The cavities, which rostrally were confined to the dorsal funiculi, became confluent with the lumen of the central canal in the lumbar area. The source of the slight fluid loss observed clinically around the lumbar cutaneous lesions was not established. The source could have been in part cerebospinal; however, as a break could not be found in the ectoderm covering the dorsal surface of the cavities, seepage through the attenuated dorsal ectoderm would have to be assumed.

Unfortunately, the present observations provide no clear-cut explanation of the causative mechanism of the syringomyelia or spina bifida occulta. Several facets of the disorder can be considered, however. Firstly, and most obviously, there is a wide discrepancy in the extent of the two lesions. Since the vertebral column forms slightly later in development than the spinal cord and since it has been shown experimentally that a normally developing neuraxis is necessary for normal development of the overlying axial skeleton [5, 13], a developmental disorder in the nervous tissue during closure of the vertebral arches could cause a concomitant lesion in those arches. This was not the case in either of these two animals.

Secondly, syringomyelia similar to that reported here has been found in segments of isolated feline spinal cord [1]. In these experiments the spinal cord was transected at L4, crushed at S5, and deafferented throughout the length of the isolated segment. In 14 cases there was no gross morphological change in the intact cord, but in one cat a cavity in the dorsal funiculi extended rostrally from the area of the crush. In the affected segments of spinal cord the dorsal spinal arteries and veins had been interrupted during the operation. The ventral spinal vessels, however, were intact with little if any cellular change in the area of the cord supplied by these vessels. A similar finding has been reported by KLINKERFUSS and HAUGH [7] who also studied isolated feline spinal cord. When the cords used in the present study were examined, the dorsal vessels overlying the area of the lesion appeared to be obliterated whereas the ventral vessels were patent. It is possible, therefore,
that the cavity in the spinal cord of the Manx kittens may have been caused by some secondary mechanism such as occlusion or interruption of the dorsal arteries or veins. This interpretation agrees with the earlier findings of Woodward and Freeman [17], who studied ischemia of the spinal cord in dogs, and with the theories of McGrath [8], Scheinker [14], and Netsky [9], who studied syringomyelia in dogs and humans, respectively.

Several questions, however, remain. What is the time of onset of the lesions described, and what is their exact cause? How much defect is there in the spinal cords of apparently normal Manx cats? Is there any chromosomal abnormality associated with the defects? James et al. [6] have shown that the Manx cat has lesions resembling those found in some humans with spina bifida, and it is possible that our current investigation of defects in the Manx cat may shed some light upon the cause of the morphologically similar human lesions.

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