Wound healing in trophic ulcers in spina bifida patients

VINOD KUMAR SRIVASTAVA, M.B.B.S, M.Ch.
Neurosurgical Unit, J. N. Medical College, Aligarh Muslim University, Aligarh, India

Eight cases of tethered conus medullaris with trophic ulcers in the foot in spina bifida patients are presented. The ulcers, which had remained unhealed for 1 to 8 years prior to surgery, healed in a few weeks after the tethered conus was released. The possibility of some neurotrophic factor in wound healing is suggested.

KEY WORDS • tethered conus medullaris • trophic ulcer • neurotrophic factor • wound • spina bifida

A WOUND generally heals with regular wound care, administration of appropriate antibiotic agents, and rest; however, even when treated, trophic ulcers can remain unhealed for years. The common causes for this are diabetic neuropathy, spina bifida, tabes dorsalis, leprosy, and peripheral nerve injury.1 Eight cases of trophic ulcers in patients with spina bifida are reported here. These ulcers remained unhealed for 1 to 8 years, but healed within a few weeks after back surgery with release of a tethered conus medullaris. The fact that these wounds healed following release of the conus medullaris suggests the possibility of a neurotrophic factor or influence.

Clinical Material and Methods

Patient Population

This series included eight spina bifida patients with trophic ulcers, whose cases are summarized in Table 1. The first patient underwent surgery at the National Institute of Mental Health and Neurosciences, Bangalore, and the others at the Neurosurgery Unit of J. N. Medical College, Aligarh Muslim University, Aligarh. In Case 1, surgery was performed because of an increasing neurological deficit, which remained unchanged after surgery; however, the trophic ulcer, which had been treated unsuccessfully for 2 years, healed in 10 days. A marked reduction in the serosanguineous discharge from the ulcer was noted within the first 24 hours after surgery. Follow-up examination at 1 year revealed no recurrence of the ulcer. This finding led the author to take a special interest in patients suffering from spina bifida who also had trophic ulcers.

Thirty-five patients with spinal dysraphism presented for treatment between January, 1988 and January, 1993. Nine of these had associated trophic ulcers. Two individuals refused surgery and were followed for 1 1/2 and 2 years without healing of their ulcers. The other seven and the one operated on earlier constitute the corpus of this study.

Clinical Features

All eight patients were boys between 4 and 18 years of age. The mean age of onset of ulceration was 4.75 years and the mean duration of the ulcer was 3.5 years. Five patients had only one limb involvement. Three patients had involvement of both feet, two of whom also had associated perianal ulcers. Five patients exhibited wet ulcers with a large quantity of serosanguineous discharge, requiring two to three dressings each day. Five patients had autoamputation of toes because of repeated ulcerations and the resultant resorption of the bone underneath (Table 1). The ulcer margins had black pigmentation in six individuals. Skin temperature was reduced in one foot in four boys, in both feet in three, and in one leg including the foot in one. The affected limb was swollen distally and the adjoining areas of the ulcers were shiny and stretched in all cases. Dorsum pedis pulsations were normal in all eight patients; however, five patients had associated orthopedic deformities (Table 1).

Three patients had dermal sinuses in the lower back; in one, there was a puckered sinus 7 cm away from the midline at the tip of a bone prominence. One patient had a lipoma on the lower back, one had a lipomeningocele, and one had a myelomeningocele. There was a tuft of hair over the lesion in three cases. One patient had a black pigmented area that had a diameter of 1 in. at the midline of the lower back. Neurologically, seven patients had cauda equina involvement, one of whom had an extensor plantar...
reflex, suggesting mixed conus-cauda equina involvement. One patient had no neurological deficit.

Radiological Findings

Plain x-ray films of the lumbosacral spine showed abnormal fusion of laminae with spina bifida at lower levels in Cases 1, 2, and 8. In Case 2, the laminae were stretched as far as 7 cm away from the midline, where a tuft of hair with a puckered sinus could be seen on the skin. All the other cases showed widening of the canal with a bifid spine in the lower lumbar region. In Case 8, the laminae were fused in the form of a bone spur that protruded through the spinal canal, causing diastematomyelia.

A myelogram was performed in six patients, showing dilatation of the terminal dural sac in all cases. A low-lying conus medullaris could be seen in two patients. Case 7 exhibited an intradural extramedullary block at the L-1 vertebral level. Computerized tomography (CT) scans were obtained following myelography in five patients. All five scans revealed a low-lying conus medullaris and, in Case 8, diastematomyelia with a lipomeningocele at the L-2 level was seen. Plain CT, performed in two cases, showed a low-lying conus in both.

Operative Findings

The conus medullaris was tethered either to a cutaneous lesion, meningomyelocele, epidermal cyst, or lipomatous meningocele. The common feature appeared to be traction on the conus medullaris. At surgery, the cause of tethering was removed in each case without section of the filum terminale (Fig. 1).

In Case 1, the laminae of L-3 and L-4 were fused. Between the two laminae, two foramina were observed; the foramen on the left was blind and a probe could be easily passed through it to a depth of 1 in. An L1–4 laminectomy was performed. A dural sheath was found entering through the foramen. This dura was separated from the foramen and opened. The conus medullaris and the cauda equina nerve roots were in their normal location, except that a number of nerve roots had deviated and become attached to the dura at the point where the dural sheath entered the foramen on the right side. The nerve roots were separated from the dura, which was then closed. The rest of the wound was closed in layers.

In Case 2, the dermal sinus, which was 7 cm distant from the midline, led to a tract that could be traced through a bone spur to the dural sac. This spur had been created due to abnormal growth of both laminae. A few nerve roots were seen tethering the conus to the dural sleeve that led to the dermal sinus. These roots were gently released from the dura.

In Case 3, the spinal cord was low lying, attached to the dural sac at S-1, and not pulsatile. The cauda equina roots ran in a cranial direction. After separating the cord from the dural sac at S-1, the cord became pulsatile, indicating release of pull on the conus medullaris.

In Case 4, a transverse incision scar caused by a meningomyelocele was not disturbed. A vertical midline incision was made above the lipomatous swelling. A laminectomy was performed at L-2; the dura was exposed at L2–3 to the point where it merged with the meningomyelocele sac. The dura was opened and a large cavity was found with the cord lying anteriorly. Numerous nerve roots were seen extending from the cord toward the meningomyelocele sac. These nerve roots were released by sharp dissection and the cord started pulsating. In Cases 5 and 7, at laminectomy, a dural sheath could be traced up to the dermal sinus. When the dura was opened,
a number of deviant nerve roots were found attached to the dura. An intradural epidermal cyst, to which the conus was tethered, was disclosed in Case 6.

Case 8 had a neural arch anomaly in which both aminae were fused with a foramen in between. The fused bone indented the spinal canal and gave rise to diastematomyelia. It was through the foramen that the dural sleeve could be traced, ending in a lipomatous meningocele. A laminectomy was performed with excision of the bone spur and the meningocele sac. The wound was closed in layers.

Results

The swelling surrounding the ulcer subsided in all eight cases and the skin regained its normal color, temperature, and texture in the 1st postoperative week. The granulation tissue became healthier, and the ulcers either healed or showed definite signs of healing by the 10th day. The maximum time of healing was 2 months in Case 2. The median follow-up period in seven patients was 12 months (range 6 to 24 months). Case 8 had no follow-up examination. None of the ulcers has recurred.

The most striking feature in the five patients with serosanguineous discharge was a drastic reduction in the quantity of discharge in four cases within 24 hours after surgery; these four required only a single dressing per day compared to two to three dressings per day preoperatively. Two patients who had large perianal ulcers apart from the trophic ulcers in the limbs showed a similar reduction in serosanguineous discharge. These ulcers each took 2 months to heal.

Discussion

Healing of ulcers within weeks after releasing the tethered conus medullaris is a striking phenomenon. One expects that wound healing and recovery of hypesthesia would be concomitant. In the cases reported here, the ulcers healed although there was no improvement in the neurological deficits. Lassman and James have made similar observations on patients with trophic ulcers in spinal dysraphism.

An obvious question is how the ulcer healed so quickly. An ulcer generally heals with good wound care, appropriate antibiotic therapy, and immobilization of the affected area. No unusual care of the wound was taken following the surgery. Neither the patient nor the ulcerated area was immobilized. The reduction in serosanguineous discharge in the four cases within 24 hours of surgery was too dramatic to be explained on the basis of wound care. If intensive wound care and antibiotics had caused the healing of these ulcers, they would have recurred during the follow-up period.
Wound healing in trophic ulcers

The role of the nervous system in the healing of trophic ulcers is difficult to define. Certain possibilities can be suggested: 1) the release of tension on the tethered conus medullaris led to some changes in the axoplasmic flow of the affected nerves, which in turn caused the healing of these ulcers; 2) the release produced some neurotrophic factor, which led to the healing of these ulcers; and 3) tethering at the conus medullaris caused reduction in activity of the caudal parasympathetic nerves with a resultant overactivity of the sympathetic nervous system, and the release of tension on the conus medullaris helped restore this balance. This last point is an interesting hypothesis; however, if these ulcers resulted from sympathetic nerve overactivity, they should remain dry, whereas five ulcers were wet and required two to three dressings a day. Apart from this, it is doubtful if parasympathetic innervation in the leg played a role in these events.

Trophic phenomena are difficult to explain. In swelling of the affected limb or trophedema, no arterial or venous obstruction has been found and the swelling has been attributed to disturbances in vasomotor control. Black pigmentation or hypermelanosis is also poorly understood. There is a strong possibility that the release of tension on the tethered conus medullaris led to some changes that precipitated the healing of these ulcers. It would be premature to comment on the nature of the neurotrophic influence, but ultimately it will probably be found to be chemical in nature.

References


Manuscript received May 10, 1993.
Accepted in final form May 13, 1994.
Address reprint requests to: V. K. Srivastava, M.Ch., 12 HIG, ADA Colony, Ramghat Road, Aligarh-202002, U.P., India.
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Duration of Ulcer (yrs)</th>
<th>Serosanguineous Discharge</th>
<th>Site of Ulcer</th>
<th>Associated Trophic Phenomena</th>
<th>Nature of Back Lesion</th>
<th>Neurological Involvement</th>
<th>Orthopedic Deformity</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6, M</td>
<td>5</td>
<td>+</td>
<td>Lt foot</td>
<td>+</td>
<td>tuft of hair</td>
<td>conus-cauda involvement</td>
<td>none</td>
</tr>
<tr>
<td>2</td>
<td>18, M</td>
<td>8</td>
<td>+</td>
<td>both feet</td>
<td>+</td>
<td>pukered sinus with tuft of hair 7 cm from midline</td>
<td>cauda equina involvement</td>
<td>talipes equinovarus</td>
</tr>
<tr>
<td>3</td>
<td>8, M</td>
<td>2</td>
<td>-</td>
<td>rt foot</td>
<td>+</td>
<td>dermal sinus</td>
<td>none</td>
<td>equinovarus</td>
</tr>
<tr>
<td>4</td>
<td>5, M</td>
<td>3</td>
<td>+</td>
<td>Lt foot</td>
<td>+</td>
<td>meningomyelocele</td>
<td>cauda equina involvement</td>
<td>none</td>
</tr>
<tr>
<td>5</td>
<td>6, M</td>
<td>5</td>
<td>-</td>
<td>rt foot</td>
<td>-</td>
<td>dermal sinus</td>
<td>cauda equina involvement</td>
<td>none</td>
</tr>
<tr>
<td>6</td>
<td>5, M</td>
<td>1</td>
<td>+</td>
<td>both feet &amp; perianal</td>
<td>+</td>
<td>dermal sinus &amp; tuft of hair</td>
<td>cauda equina involvement</td>
<td>talipes equinovarus</td>
</tr>
<tr>
<td>7</td>
<td>13, M</td>
<td>2</td>
<td>+</td>
<td>both feet &amp; perianal</td>
<td>+</td>
<td>lipoma</td>
<td>equinovarius</td>
<td>equinovarus</td>
</tr>
<tr>
<td>8</td>
<td>4, M</td>
<td>1</td>
<td>-</td>
<td>Lt foot &amp; Lt leg</td>
<td>-</td>
<td>lipomeningocele</td>
<td>cauda equina involvement</td>
<td>equinovarus</td>
</tr>
</tbody>
</table>

* Symbols: + = existence of condition; - = nonexistence of condition.

**TABLE 1**

Clinical features in eight spina bifida patients with trophic ulcer*