Traumatic Infarction of the Spinal Cord

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SUMMARY. *Infarction of the spinal cord is an occasional complication of thoracic and aortic surgery and dissecting aneurysm. Only once has it been reported following injury. This paper recounts the histories of four children each of whom survived an automobile accident; each with permanent paraplegia due to infarction.*

RÉSUMÉ: *L'infarctus du cordon médulaire est une complication rare de la chirurgie thoracique et aortique ainsi que de l'anévrisme disséquant. On n'en a dénombré qu'un seul cas survenu à la suite d'une blessure. Cet article rapporte le cas de quatre enfants, qui, ayant survécu à un accident de la route, sont tous atteints de paraplégie permanente causée par cet infarctus.*

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Infarction of the spinal cord caused by trauma has been reported only once, by Hughes (1964). A 62-year-old woman walked away from a motor accident in which her 2nd, 3rd and 4th ribs had been fractured. The pleura over the aorta had been torn just above the diaphragm. She had extensive mediastinal bruising, and a hemothorax, which was operated on the day after injury. Six days after injury she developed complete paralysis of the legs and absence of all sensation up to T3. Roentgenograms disclosed no fracture of the spine and the myelogram showed no block. She died 7 days after injury. At autopsy a transverse intimal tear almost encircling the aorta was found 4 cm below the origin of the left subclavian artery. The tear was plugged with thrombus continuous with a large mural hematoma and the damaged tunica media and adventitia. The hematoma surrounded and compressed the origins of the 2nd, 3rd, 4th and 5th intercostal arteries. The whole diameter of the cord was infarcted from T3 to T7, the lowest level examined.

Infarction of the spinal cord following aortic and thoracic operations is well known (Mosberg et al., 1954; Brewer et al., 1972). This paper presents the histories of four children, each of whom survived an automobile accident. Three were left with permanent paraplegia and one with a permanent lesion of the conus medullaris. In the first three cases there was a delay of 2 to 11 hours from the time of injury until the onset of paraplegia. In the fourth case the onset of infarction was less clearly defined. In none of them was there any fracture or dislocation of the spine by roentgenographic examination. Myelography in cases 3 and 4 was negative.

CASE REPORTS

Case 1

In December, 1941, a 10-year-old boy was pushed along a snow-packed icy road by the locked wheels of a bus. He was bruised about the chest and left upper abdomen. On admission to hospital it was noted that he moved his legs voluntarily quite well. Within a few hours swelling appeared around the left zygoma and cheek and within another few hours this swelling was symmetrical on both sides of his neck and on his cheeks. No surgical emphysema was observed at any time. The first x-ray showed a small left pneumothorax, and perhaps three fractured ribs on the left side. When the attending surgeon examined the abdomen for evidence of ruptured spleen 9 hours after injury, the boy drew both legs up. Two hours later, however, he asked his nurse how long his legs were going to be "numb like that." When the nurse asked him to move his legs she found he was paralysed.

A sensory level was found just above the umbilicus. This level remained constant. It seemed certain that the boy did not empty his bladder after he was injured. His urine was normal. Lumbar puncture yielded clear fluid.

When examined, 40 hours after injury, he was rational and co-operative. His face was flushed but the swelling of neck and cheeks had diffused somewhat. Neurological examination of arms and cranial nerves was negative. Respirations were restricted on the left side of the chest. The skin on the left side of his chest seemed to be marked by the texture of his clothing, the outline being due to small hemorrhages. There was little edema of skin or muscles over the chest and back, however. When either leg was moved the child was sometimes able to guess which leg it was, but he explained that he could tell by the movements of his back muscles. He had complete loss of power and sensation in front below the umbilicus. On his back, sensation was present down to the 2nd or 3rd lumbar level on both sides but not below that. Beevor's sign was positive.

Numerous roentgenograms of chest, ribs, and vertebrae were taken. There

was no evidence of any hematoma in the mediastinum, but it was thought the pharynx and trachea might be displaced anteriorly from the front of the vertebral column. Stimultaneous cisternal and lumbar punctures were done with No. 19 gauge needles and on jugular compression the fluid rose equally rapidly in each tube. On release the cisternal fluid dropped more quickly than the lumbar fluid.

Exploratory laminectomy was carried out 44 hours after injury. The incision centered on D8 spine, exposing the laminae of D6 to D10, and the spines and laminae of D7 to D10 inclusive were removed. No evidence of local injury, swelling or hemorrhage was seen. When the dura was opened the spinal cord appeared normal.

Acceptance of permanent paralysis was so difficult for the parents that the boy was brought to The Hospital for Sick Children. It was agreed that the spinal cord should be re-explored. This was done 44 days after injury. To quote from the note dictated by the house surgeon, "The spinal cord was now examined carefully throughout the length exposed. It was noted that about the level of what could have been the 6th thoracic spine the spinal cord began to taper off gently. Its diameter became appreciably narrower as one descended two segments. At about the level of the 8th thoracic vertebra the cord was roughly ¾ of its width above and appeared to take a rather gentle bend in an anterior direction. The cord was gently retracted from side to side allowing the antero-lateral quadrants to be examined but nothing unusual was found. There appeared to be no hematomyelia."

Case 2

A press clipping from a local newspaper describes the injury to a 5-year-old girl in November, 1950. ". . . was loading a truck with some 3 tons of buckwheat . . . When the truck left the barn Dianne was riding on the side of it. As the truck began to move she became scared and jumped, the dual wheels at the back going over her stomach and legs. How the child escaped instant death is a miracle and the only theory that can be advanced is the fact that the ground was of a sandy nature and must have 'given' allowing the child's body to sink a little with the weight of the truck."

The next information is from the local surgeon, in a comprehensive report which accompanied the child to hospital. "She was attended by her family doctor who found that she was a little shocked with pulse of 120, and slightly pale, but showed no gross injuries. She was moving both legs and both arms and was conscious though in pain. A little over an hour later she vomited severely and her condition deteriorated. On arrival at hospital she was pale and almost pulseless. She was given oxygen, and plasma intravenously by syringe. This brought her blood pressure up to 110 m.m. systolic, pulse 110 to 120 and her color improved. Examination of lungs and chest was negative. The abdomen was distended and there was dullness in the flanks. Urine obtained by catheter was normal. The child complained of pain in the left side. There was complete anaesthesia below D12 and partial anaesthesia from about D8 to D12. There were no voluntary leg movements and there were no reflexes in the legs.

Laparotomy was done for suspected intra-abdominal hemorrhage from a ruptured spleen. No blood was found but acute dilation of the stomach was discovered. The child's condition began to improve after the stomach was emptied. She was given blood before and during the operation. Over the next few days she was given 300 cc of blood, more intravenous fluid and her stomach was aspirated frequently. There was a temporary suppression of urine. She seemed to have sensation when a catheter was passed and it is said that she moved her right foot.

On admission to The Hospital for Sick Children, 11 days after injury she was alert and co-operated well, but had complete paraplegia and sensory defect up to the second dorsal segment. Respirations were affected. She had difficulty clearing her bronchial secretions. Three weeks after injury, bronchoscopic suction was done because of collapse of the upper lobe of the right lung. Two weeks later the lung was again expanded. On inquiry the parents described swelling of both sides of the face up to the cheek bones, while the child was lying at home waiting transfer to hospital.

Roentgenograms made in both hospitals showed the cervical , dorsal and lumbar spines to be normal. Films of the chest showed normal bony thorax, heart and diaphragm. The lung fields were clear. A transverse fracture of the right ilium and fractures of both pubic bones were found but the fragments were not displaced.

Case 3

In June, 1967 a 15-month-old baby girl was run over by a motor car. The history was given by the family doctor when she was transferred to The Hospital for Sick Children, about 24 hours after injury. One of the rear wheels made tire marks over the posterior part of her chest from the left axilla over the scapula to the midline. There was a statement that "her body was kicked out by the action of the wheels into the street." She sustained lacerations of her scalp on the left side reaching onto the right side of the forehead. Roentgenograms of the skull were negative. Neurological examination of the eyes was negative and the extremities moved purposefully and equally. Abrasions and contusions marked the anterior chest wall opposite the left scapular tire mark. The chest moved reasonably well. Breath sounds were heard on both sides, but less distinctly on the left. The heart rate was 100 to 110. Examination of the abdomen was negative. The scalp was repaired and the wounds were cleaned and sutured.

At about 20 hours after injury it was noted that the child had a full bladder but no discomfort. Urine was expressed by pressure. At that time her legs did not move although movement had previously been seen by two doctors. No tendon reflexes or abdominal reflexes were present.

She had complete motor and sensory defect below D.8 in front and D.11 behind. Beevor's sign was positive; the anal sphincter was relaxed. No fractures or dislocations were seen in the dorsal and lumber spine. A complete myelogram was normal.

Case 4

A 2½-year-old boy was struck by an automobile in 1968. He was briefly unconscious and had a 3 inch occipital scalp laceration. Bruising occurred over the thoracolumbar region posteriorly and over the right ribs. He was in shock. His blood pressure was 60/40 and his pulse 200; his abdomen was distended. Cranial nerves were normal. He had normal power and sensation in his limbs, but knee and ankle reflexes were absent.

On the evening of admission to The Hospital for Sick Children a ruptured spleen was removed. Following the operation he was unable to void and a catheter was inserted. Inability to void persisted. On the 17th day after injury he was unable to stand or walk because of extremely weak extensors of both hips and virtually no power at the knees and ankles. He had total anaesthesia of the sacral dermatomes 1 to 5 on both sides and his anal sphincter was lax. Knee jerks were present, the right being more active than the left. The ankle jerks were absent; the plantar responses flexor.

Roentgenograms of the skull and spine a few days after injury and a myelogram 4 weeks after injury were all normal.

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DISCUSSION

In eight cases of spinal cord infarction caused by dissecting aortic aneurysm, Thompson (1956) has listed involvement of some intercostal or lumbar arteries or both. These had been shorn off, thrombosed, stretched or severed. In his Case 8, reported in detail, "a small intimal tear was found at the origin of the left common carotid artery. About 1.5 cm distal to the left subclavian artery a transverse intimal tear 3 cm in length was present in the posterior wall of the aorta. Further examination of the aorta revealed the paired ostia of eight intercostal and three lumbar arteries. The right 5th, left 6th and 7th intercostal arteries were found to be stretched. The courses of the remaining intercostal and lumbar arteries were interrupted near their origins. Thrombus was present in the first lumbar arteries."

In Case 1, presented here, it seems reasonable to conclude that the boy's chest, mediastinum, thoracic spine and trunk were subjected to a number of severe compressive, twisting and shearing stresses. In the light of his subsequent history and the pathological studies cited it appears that his thoracic aorta was stretched, twisted or otherwise injured in such a way that stretching or rupture of one or more intercostal arteries occurred.

The arteria magna of Adamkiewicz (Lancet, 1967) arises from an intercostal artery at about the level of the diaphragm, nearly always on the left. If this vessel had been severed or thrombosed it would explain the infarcted cord found at the second exploration. The same result, however, might have been due to damage of a number of intercostal arteries.

In Case 3 the sensory and motor defect was at virtually the same level as in Case 1. The explanation of the etiology of the lesion is probably similar.

In Case 2 the sensory and motor level was at T2. Interference with the blood supply to the cord may have resulted from damage to a large branch from the costo-cervical trunk in the lower cervical region, or to destruction of one or more intercostal arteries. These arteries (Lancet, 1967) to the spinal cord supply a length of cord both below and above the level of entry.

In Case 4 the question of infarction is more doubtful. If correct, it would be due to damage to a major contributing vessel in the upper lumbar region. In Cases 1 and 2 there is evidence of swelling extending up through the neck into the face from contusion in the mediastinum.

As for other causes of paraplegia; luxation or subluxation at the time of injury is ruled out by the delayed paralysis, the absence of any abnormal roentgenographic findings and the atrophy of the spinal cord seen in Case 1.

Infarction of the spinal cord due to accident or trauma, followed by survival, has not been described before. The injury producing infarction appears to be damage to the aorta and thrombosis, stretching and rupture of intercostal and lumbar arteries. The pathological process is probably similar to the changes which have been described following dissecting aneurysm of the aorta and in the case reported by Hughes (1964). Another factor is the tearing of the intima of the aorta presumably producing a condition similar to the onset of dissecting aneurysm. If the observations in this paper are valid, research into the pathology of injury to the aorta and its smaller branches should be undertaken, particularly in institutions where autopsies are done on relatively large numbers of automobile accident victims. It is also likely that a large number of patients whose paraplegia has been ascribed to fracture of the spine, or fracture dislocation of the spine, or contusion of the spinal cord, have suffered infarction of the cord. The error can occur because some essential features of the history are missing, but also because one is apt to forget the aorta, an organ seemingly remote from the spinal cord, as a factor in cord damage.

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