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CASE REPORT

TRAUMATIC CEREBRAL FLACCID PARAPLEGIA

By P. J. TEDDY, D.Phil. F.R.C.S.[1,2] J. R. SILVER, M.B., B.S., F.R.C.P.,[1] J. H. E. BAKER, B.Sc., M.B., B.S., M.R.C.P.[1] and A. OHRY, M.D.[3]

[1] *National Spinal Injuries Centre, Stoke Mandeville Hospital, Aylesbury, Bucks, HP21 8AL, U.K.* [2] *Department of Neurological Surgery, the Radcliffe Infirmary, Oxford OX2 6HE, U.K. and* [3] *Chaim Sheba Medical Centre, Tel Hashomer, 52621, Israel.*

Summary. Two cases of paraplegia caused by head injuries sustained in recent military conflicts are presented. The underlying pathological disturbances could be related to the CT scan appearances and are discussed in the light of previously reported series. Both cases were remarkable for an early and prolonged paraplegia which was flaccid, rather than spastic, and for the sensory impairment produced.

Key words: Cerebral trauma; Flaccid paraplegia; Mechanism; Outcome.

Introduction

THERE have been several previous accounts of paraplegia as a result of a variety of intracranial lesions—usually affecting the frontal or parietal parasagittal regions. Parasagittal meningiomas, superior sagittal sinus thrombosis and ischaemia following rupture of anterior communicating artery aneurysms have all been implicated (Harris and Strong, 1971). Holmes and Sargent (1915) described the effects of missile wounds to the parasagittal region from the First World War, commenting on the early appearance of rigidity and attributing this to damage to the superior sagittal sinus. Russell and Young (1969) reporting on selected cases from the Oxford series of World War II missile wounds favoured a purely cortical disturbance but also commented on the striking early appearance of rigidity or spasticity of the lower limbs in their cases. We report two cases from more recent conflicts in which injuries to the parasagittal region resulted in paraplegia which was flaccid, and compare the clinical and pathological features with those noted in the previous series.

Case reports

Patient 1

E.W. a left-handed 35-year-old Marine was hit by shrapnel from a 500 lb bomb on 27.5.82 during the landing on East Falkland Island. He sustained a concussive head injury and lacerations of the right forearm and knee. At the field hospital a stellate laceration over vertex of the head was explored but the outer table of the skull was found to be intact. The following day he was transferred to a hospital ship and was noted to be confused and moving all four limbs, but within 24 hours had developed a total flaccid paraplegia. X-rays of the thoracic and lumbar spine were entirely normal

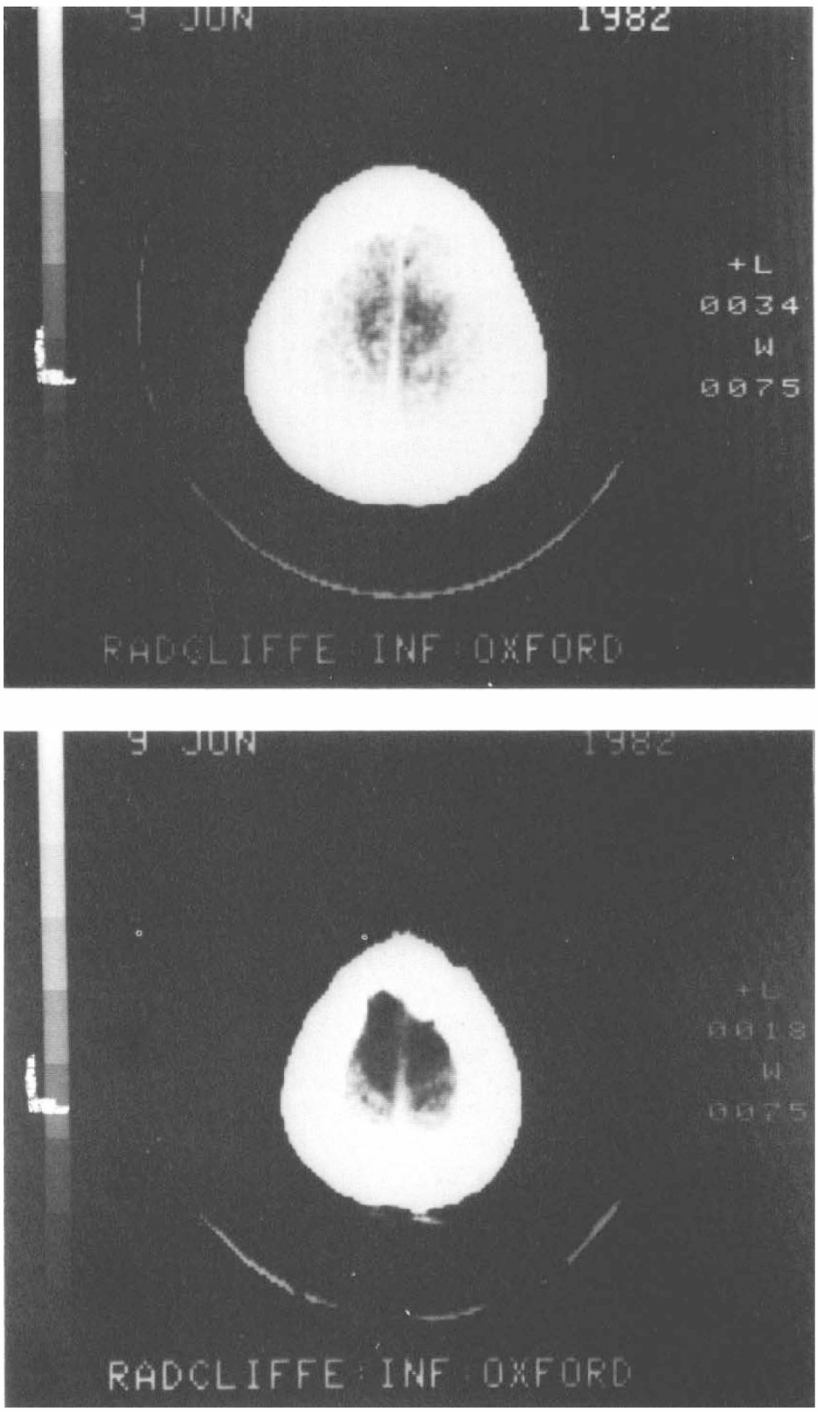

FIG. 1
A and B; Case 1 bilateral parasagittal low density areas seen on CT scan.

but skull X-ray revealed metallic fragments in the left temporal region. He was treated with antibiotics and high doses of steroids. A CT scan in Uruguay showed low density in the parasagittal region directly below the scalp laceration. On 8th June 1982 he was transferred to Stoke Mandeville Hospital and then to the Radcliffe Infirmary. On examination he was alert and orientated with a mild nominal dysphasia, minimal dysarthria and bilateral anosmia. He had a total flaccid paraplegia with impaired sensation to all modalities to the level of L.2 with sacral sparing, and there was also minimal weakness and clumsiness of the left hand. The plantar responses, which were just detectable, were extensor. There was complete loss of scalp for 3 sq cm over the vertex and a small penetrating wound low in the left posterior temporo-parietal region. X-rays demonstrated shrapnel in the right forearm, the right knee and the left temporal region but not in the spine. A repeat CT scan confirmed bilateral parasagittal low density in the posterior frontoparietal region (Fig. 1), fragments of bone and metal in the left temporal region and a tiny metal fragment in the right cerebello-pontine angle. A urinary catheter had been inserted on the hospital ship to facilitate transport to the U.K. but the patient had been able to void spontaneously prior to this. On 10th June 1982 the posterior temporo-parietal wound was explored, the fragments of bone and shrapnel embedded in the underlying cortex removed, the dura repaired and skin grafts applied to the scalp and forearm.

His post-operative recovery was complicated by a pulmonary embolus but otherwise he made a steady and almost complete recovery. Within three weeks his dysphasia had resolved, he could weight bear but could not walk unaided and had normal sphincter function. His sensory abnormality had almost resolved but there was residual impairment of joint-position sense in the feet. At this stage the legs were still hypotonic but there was ankle clonus. He was transferred back to Stoke Mandeville Hospital and subsequently to Headley Court. Three months later he could walk unaided and had only mild spasticity of the right leg. Almost one year after his injury he had just mild impairment of power and joint-position sense in the right foot, and he was able to rejoin his unit.

Patient 2

O.B.C. a 21-year-old soldier sustained a direct bullet wound to the head in the Lebanon on 7th June 1982. Immediate hospital admission was arranged when he was noted to be conscious and alert but with a total flaccid paraplegia and a sensory deficit to all modalities below T.5 with complete sphincter paralysis. He had a small entry wound in the right parietal area and skull X-ray revealed an extensive depressed fracture below this extending to the midline at the vertex. Cervical and dorsal spine films were normal and a CT scan demonstrated no intracerebral pathology. A few hours later the patient developed weakness of the left hand and became progressively less responsive. The compound depressed skull fracture and entry wound were explored and the wound found to be tangential to the skull. A small dural laceration was repaired but there was thrombosis of the superior sagittal sinus. The thrombus was removed and the sinus repaired. Post-operatively the patient had several fits but his progress was otherwise uneventful and on 16th June 1982 he was transferred to a spinal

injuries centre. At that time he was fully alert but still had a total flaccid paraplegia and weakness of the left arm—the latter resolving over the next few days. Repeat CT scan showed two small areas of low attenuation in the posterior parietal parasagittal region. Four months later he had no sensory or sphincter deficit but had a moderate paraparesis which was still flaccid. He was able to walk slowly using a Zimmer frame.

Discussion

Patients with paraplegia secondary to head injuries are only infrequently encountered in peace time, thus two patients seen recently who sustained lower limb paralysis due to missile wounds to the head are of interest, both for their clinical presentation and for the intracranial pathology noted on CT scan and at operation.

Russell and Young (1969) reported 24 cases from the Oxford Series of missile wounds to the head. These patients, who were casualties from the second World War, had paraplegia associated with blunt injury to the parasagittal area. The injuries were all caused by small fragments of shrapnel which did not penetrate the dura and had not involved the sagittal sinus. This was in contrast to those cases reported by Holmes and Sargent (1915) in which paraplegia was usually related to glancing bullet wounds to the head. Holmes and Sargent described early and profound stiffness of the affected limbs as well as a severe cortical sensory impairment and suggested that the underlying pathology was damage to the superior sagittal sinus.

Russell and Young also described weakness of the legs coming on soon after injury to the head, occasional weakness of one or both arms, various degrees of sensory impairment, sphincter disturbances and almost invariably an early and profound increase in tone of the lower limbs with increased reflexes. However, they attributed the clinical picture to direct damage to the paracentral parasagittal cortex rather than to secondary cortical impairment from sagittal sinus thrombosis. The particular area of cortex involved explained fairly readily the observed sensory, motor and sphincter impairments. However, the immediate spasticity was less easily explained and they suggested that the bilateral nature of the cortical injury and possible damage to the supplementary motor areas might be important.

Our two cases were very similar to one another in terms of neurological deficit, but differed in terms of pathology, representing both types of mechanism of injury put forward by Holmes and Sargent and Russell and Young. In the first patient there was no evidence of sagittal sinus thrombosis, either clinically or on the CT scan, whereas in the second case sagittal sinus thrombosis was seen at craniotomy. Yet in each patient there was an identical clinical picture which differed from almost all those cases described by the previous workers in that early flaccidity, rather than spasticity, was an outstanding feature. Also, in each case the cortical damage was localized to the posterior fronto-parietal parasagittal region, as was seen on the CT scan. The temporal penetrating wound in Case 1 seems unlikely to have contributed to the signs noted in terms of leg movement, or of the sensory impairment in the legs.

Conclusion

Two cases of traumatic paraplegia produced by localized blunt injury to the posterior fronto-parietal parasagittal region, as demonstrated on the CT scan, both presented with a flaccid paraplegia, a sensory level and a severe sensory loss—a picture more typical of an acute spinal injury. The profound decrease in tone of the lower limbs differed from virtually all those spastic cases reported in larger previous series. One case was associated with sagittal sinus thrombosis but in the other there was direct damage to the cortex without obvious thrombosis of the sinus. Neither had sustained a spinal injury.

Résumé

Deux cas de paraplégie, secondaires à un traumatism crânien reçu durant récent conflits militaires, sont présentées. Les dérangements pathologiques et les données de la tomodensitométrie (TDM), ayant rapport, sont discutés. Les deux cas sont remarquables parceque la paraplégie, d'apparition précoce, est prolonguée et flaccid, et à cause de l'altération sensoriel.

Zusammenfassung

Zwei Fälle von Beinlähmung die durch Kopfverletzungen in militärischen Handlungen werden präsentiert. Die fundamentalen pathologischen Störungen konnten in Bezierung gebracht werden zu den CT Erscheinungen und werden diskutiert von früheren Berichten. Beide Fälle waren beachtenswert durch eine frühe und in Länge gezogene Beinlähmung die schlaff mehr als krampf war und für die Gefühlssinne Herabsetzung.

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