CLINICAL AND ANATOMICAL OBSERVATION OF A PATIENT WITH A COMPLETE LESION AT C₁ WITH MAINTENANCE OF A NORMAL BLOOD PRESSURE DURING 40 MINUTES AFTER THE ACCIDENT

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Abstract. An unusual case where there was complete section of the cord at the level of C₁-C₂ is presented. He was admitted in deep coma, approximately 10 to 15 minutes after the accident. Without the use of drugs, the blood pressure was maintained at 130 Torr for 25 minutes after admission, but progressive hypotension ensued.

Key words: Spinal cord injury; Tetraplegia; Pressure response; Brain death.

Case Report. Mr R., 18 years old, of Portuguese origin, was crossing the street one evening when he was hit by a car. When the emergency mobile squad arrived, approximately 6 minutes later, he was found lying face downwards in a state of complete cardio-respiratory arrest. He was immediately intubated, manually ventilated and at the same time external cardiac massage caused the heart to start again. The transfer was rapid, the hospital being less than 2 km distant. He was therefore admitted to the emergency department approximately 15 to 20 minutes after the accident. He was in a deep comatose state with bilateral mydriasis unresponsive to light. All other reflexes were absent. No spontaneous respiration could be detected but on the haemodynamic side the blood pressure was constant for another 20 minutes at 130 Torr (the maximum only was recorded) and cardiac frequency was 90 per minute. This was particularly interesting as the patient had not received any intravenous solution or drugs before admission, nor during the period when these blood pressure values were recorded. The X-ray examination showed no cranial fracture, no apparent lesion of the cervical spine and only a transverse fracture at the middle third of the left femur which accounted for a concomitant loss of blood of at least 1½ litres. At the end of the radiological examination (20 minutes after his admission and approximately 35 to 40 minutes after the trauma), the blood pressure fell dramatically to 50-40 Torr accompanied by a sinus bradycardia at a rate of 40 to 35 per minute. Faced with the association of deep coma, total areflexia, bilateral mydriasis and hypotension with bradycardia, the doctor on duty diagnosed the high probability of an irreversible coma. This diagnosis was 'confirmed' by an I.P.P.V. disconnecting and pure oxygen test. These tests were performed for 15 minutes. The diagnosis of brain death seemed most evident and the patient was transferred, orally intubated and artificially ventilated, to the resuscitation department. It was expected that death would follow shortly afterwards.

It was only a few hours later, next morning, before an EEG was to be performed to confirm brain death, that reappearance of signs of brain function occurred, *i.e.* opening of the eyelids, myosis and deglutition reflexes caused by the oral intubation tube. The possibility of a spinal cord injury was evoked evidence of priapism (very unusual in coma). The blood pressure remained at 50 Torr and the cardiac frequency still at 40 per minute. Very little renal secretion was recorded.

The intravenous perfusion was then increased under central venous pressure monitoring and oral intubation was changed to a nasal one. A perfusion with Dopamine re-established the blood pressure at 90-100 Torr and the cardiac frequency at 80 pulsations per minute and this had to be maintained to keep the blood pressure at an acceptable

level. As soon as the fluids were administered in greater quantity the rectal temperature dropped from 36°C to 32°C and gradually returned to normal after a few hours.

On the same day the EEG showed a spontaneous reactive ground rhythm with diffuse slow waves. Caloric vestibular stimulation showed total inexcitability. The X-rays of the cervical spine were re-examined and appeared normal. Other additional X-rays were taken in flexion and extension to try to detect any anomaly at the level of C1-C2. As the level of consciousness appeared to improve during the following 2 days, a sensory neurological examination was possible with some cooperation from the patient, the responses being obtainable with eyelid and eye movements. It appeared that no sensation, neither touch nor pinprick, could be recorded until one reached the level of the trigeminal nerve. At this level it was difficult to ascertain if there was any difference of appreciation of sensation between the three different branches of this nerve. No reflexes were ever found below the expected level, except the bulbo-cavernosus, anal and balano-anal reflexes. Air-myelography was performed which confirmed the diagnosis of a complete spinal cord section at C1 (Fig. 1 without, and Fig. 2 with Metrizamide (Amipaque®)). Bilateral carotid and vertebral angiography were normal. Because of an ischaemic syndrome the left leg was amputated on the second day. A somatosensory evoked potential study was planned but could not be carried out as on the 15th day the patient died during the period of autonomic hyperthermic dysreflexia at 41°C, associated with pulmonary oedema.

The pathological examination not only confirmed complete cord section at the level of C1-C2 (Fig. 3) but the presence of a small clot the size of a pea on the anterior aspect of the spinal cord. It also showed a dislocation of C1-C2 which had been the cause of the spinal cord injury.

FIG. 2

Air-myelogram plus metrizamide (R) shows an obvious transverse lesion at the level of C1.

FIG. 1

Air-myelography shows a lesion at the level of C1.

Discussion

Four comments are brought out from this case report:

1. Patients surviving a complete high spinal cord lesion at the level of C1 and C2 are rare. These lesions are usually fatal and found at post-mortem (Davis *et al.*, 1971). Their survival is only possible because of the institution of immediate resuscitation measures. In this particular case, the prompt arrival of the medical

FIG. 3

Post-mortem specimen showing a complete transverse lesion of the spinal cord at C1 due to a dislocation of C1/C2 vertebrae.

rescue squad from the hospital, the rapid intervention and artificial ventilation overcame the hypoxic cardiac arrest due to the respiratory paralysis.

2. It illustrates some similarity between two clinical situations; between that of pure traumatic coma and that of a very high spinal cord lesion in a deep comatose stage. Low blood pressure, bradycardia and hypothermia are well-known signs of an irreversible coma. Moderate hypothermia (but not below 35°C), can accompany irreversible coma. Diabetes insipidus is not constant. Cutaneous nociceptive reflexes exist in irreversible coma (Jørgensen, 1973). The only initial sign suggesting a spinal cord injury was in fact the priapism. One must now be aware that even in the presence of a clinical picture evoking cerebral death the I.P.P.V. disconnecting and pure oxygen test cannot indicate certain cerebral death.

3. A vertebral lesion with a subsequent spinal cord section can pass totally undiagnosed even with good radiological pictures.

4. Haemodynamic modifications after a complete spinal cord section at a high dorsal or at a cervical level have been the subject of several animal experiments in recent years (Rawe *et al.*, 1977; Tibbs *et al.*, 1978; Rawe & Perot, 1979; Alderman *et al.*, 1980; Young *et al.*, 1980). The hypertensive phase is usually of short duration, not more than a few minutes, although the plasma norepinephrine values seem to remain elevated up to two hours after transection when performed at the cervical level (Tibbs *et al.*, 1979). As to the spontaneous re-establishment in man of a normal or even higher blood pressure without any drug or fluid administration, one could evoke the hypothesis of a massive, short lasting, catecholamine liberation either by the adrenal glands, or a secretion outburst of Norepinephrine at the sympathetic nerve endings immediately following the transection of the spinal cord at a high level. Hypoxia, by itself could have been the hypertensive

agent in our case. The study of plasma-circulating catecholamines, as soon as possible after the accident, has now been started on a systematic basis.

SUMMARY

The authors report on their clinical observations concerning a patient with a complete spinal cord injury at the level of C₁, followed by a cardiac hypoxic arrest, due to immediate respiratory paralysis after the accident. Normal cardiac activity was obtained as a result of rapid resuscitation measures, using only intubation and external cardiac massage without any drug administration. The blood pressure was maintained without any drugs at a level of 130 Torr during 40 minutes before it fell to a permanent level of 50–40 Torr on ventilation alone. The diagnosis during the first hours was believed to be that of an irreversible coma with no evidence of vertebral injury. The patient started to recover consciousness after a few days but died on the 15th day. The case is discussed in the light of the literature and of the recent physiological experiments concerning the rapid changes of blood pressure after spinal cord section in animals.

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RÉSUMÉ

Les auteurs rapportent une observation clinique d'une section médullaire traumatique complète en C₁, sans signe radiologique, compliquée d'un arrêt cardio-respiratoire avec les signes cliniques de mort cérébrale. L'arrivée rapide des secours permet d'obtenir, après intubation et massage cardiaque externe, sans administration d'aucune drogue, un redémarrage de l'activité cardiaque. La tension artérielle se maintient sans thérapeutique à 130 Torr pendant environ 40 minutes avant de s'effondrer définitivement. La régression partielle du coma survient quelques heures après. La paralysie était cliniquement totale en C₁ malgré l'absence de tout signe radiologique sauf myélographique. Le décès survient le 15^e jour et l'autopsie confirme la lésion anatomique complète en C₁–C₂. Cette observation est discutée, particulièrement en ce qui concerne la valeur de l'épreuve du 'débranché' plus oxygène pur ainsi que le rétablissement spontané de la tension artérielle pendant un temps relativement court dans une lésion cervicale haute et complète.

ZUSAMMENFASSUNG

Es wird hier von einer akuten, kompletten, durch Strassenunfall verursachte C₁ Quadriplegie berichtet. Der Zwangsläufige Atem und Herzstillstand konnte Dank des raschen Einsatzes des Notartzwagens erfolgreich behandelt werden. Dieser Fall ist aus drei Gesichtspunkten betrachtenwert: (1) Trotz mehreren Halswirbelröntgenaufnahmen, die vollkommen normal waren, bewies die Obduktion einen kompletten Querschnitt im C₁ Bereich. (2) Nach Intubation und Herzmassage entstand eine Wiederherstellung des Blutdruckes auf Normalwerte von 140 Torr und ohne Verabreichung von Katecholaminen oder Volumenzufuhr. Dieser Normalwert bestand 'spontan' etwa 40 Minuten lang. (3) Koma, Mydriasis, totaler Atemstillstand, allmähliches Absinken des Blutdruckes, Eintreten von Bradycardie und Hypothermie, u.s.w. liessen auf Hirntod schliessen. Nur der Priapismus und später das E.E.G. konnten am Anfang die Fehldiagnose von Hirntod wiederlegen.

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