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TRAUMATIC RUPTURE OF THE AORTA AND PARAPLEGIA

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Abstract. During the past 10 years, 15 traumatic ruptures of the aorta isthmus have been diagnosed. The most likely mechanism is that of a brutal deceleration. Three patients developed a complete mid-thoracic flaccid paraplegia. One died in a few hours of a complete rupture, the second after a few days from renal insufficiency, the third 12 days after, having been operated upon on the same day of the accident. All had associated lesions. Diagnosis can be very difficult in these patients, especially in the first few hours. Paraplegia is here considered as an aggravation ischaemic symptom with anuria and loss of blood circulation in the lower limbs. In delayed cases the vascular mechanism of the infarction of the spinal cord is discussed, either by compression of the intercostal arteries or by direct compression of the aorta itself. In our cases, the most likely mechanism is that of an obstruction of the lumen of the aorta by a torn inner coat. These patients must be transferred as quickly as possible to a cardiovascular surgical unit and operated upon immediately. Three cases of preoperative, rapidly progressive paraplegia with neurological recovery after the operation, have been reported in the literature.

Key words: Traumatic rupture aorta; Paraplegia; Pseudo-coarction syndrome.

DURING the past 10 years at the Pasteur Hospital of Colmar, 15 traumatic isthmian ruptures of the aorta have been diagnosed. Amongst these 15 cases, most with the history of a *brutal deceleration*, three have been associated with a progressive mid-thoracic flaccid paraplegia.

The diagnosis in these 15 cases was based on (most of them with multiple injuries) the most usual associated radiological and clinical signs: enlargement of the upper mediastinum, tracheal deviation towards the right, left-sided haemothorax, associated or not to an acute 'pseudo-coarctation' ischaemic syndrome, the femoral pulses being diminished or absent with oliguria or anuria (Table 1). The diagnosis was confirmed by angiography or necropsy (Gschaedler *et al.*, 1978).

Paraplegia appears to be an *ischaemic* complication symptom, although being a syndrome in its own right, and has been reported in the literature mostly in cases of dissecting aneurism or as a complication of aortic surgery, especially after prolonged clamping (Corbin, 1961). The results of cardiovascular modern surgery are nowadays promising.

The post-traumatic incidence, mostly after a deceleration compression mechanism or a blunt chest trauma, has been reported by Binet and Langlois (1961), Hughes (1964) and Langlois, Binet and Jegou (1971).

Otherwise, the review of the specialised literature is rather scarce on this particular subject, as paraplegia is just briefly mentioned.

Mortality, in our series of traumatic rupture of the aorta, has been overall very high, approximately 50 per cent and extremely high in our series of cases associated with paraplegia (100 per cent).

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TABLE I

Signs caused by traumatic rupture of the aorta (Review of 15 cases)

Case No.	Year	Age	Mediastinum enlarged	Right tracheal deviation	Left haemothorax	Ischaemic syndrome		
						Femoral pulses	Paraplegia	Anuria
1	1966	20	Yes	−	+	Present +	−	−
2	1967	22*	No	+	...	Absent −	Yes	Yes
3	1968	25*	Yes	+	...	Diminished	...	Yes
4	1969	19*	Yes	+	+	Absent	Yes	Yes
5	1970	42*	Yes	+	...	...	...	...
6	1973	39	Yes	+	...	Diminished	...	...
7	1974	19	Yes	±	...	Diminished then absent	...	...
8	1975	25*	Yes	+	+	...	...	...
9	1975	21	Yes	−	+	...	...	...
10	1975	27	Yes	±	...	...	...	...
11	1975	46*	Yes	±	...	...	...	...
12	1976	50	Yes	±	...	...	...	...
13	1976	28*	Yes	+	...	Absent	Yes	Yes
14	1976	44	Yes	+	...	...	...	...
15	1977	23	Yes	−	...	...	...	...

* Indicates died.

Case Reports

Case No. 1. 1967: 22-year-old man. Car accident with the knowledge of a violent deceleration. Associated lesions: haematoma of the right lung apex and pleural effusion. Neurologically: very rapid installation of a complete mid-thoracic level flaccid paraplegia. This patient died suddenly after a few hours by complete rupture of the aortic isthmus shown at necropsy.

Case No. 2. 1969: 19-year-old man. Car accident with also a knowledge of a brutal deceleration. Associated lesions: multiple rib fractures, bilateral haemothorax, lung contusion, abdomen, kidney and liver rupture, retroperitoneal haematoma, fracture of the pelvis. Neurologically: progressive mid-thorax level flaccid paraplegia becoming complete in a matter of hours after the accident. Operated upon the day after the accident, died on the fourth day of an acute renal insufficiency. The angiography (Fig. 1) shows the isthmian localisation of the incomplete rupture.

Case No. 3. 1976: 28-year-old man. Car accident with a probable deceleration mechanism. Shocked. Fracture of the second, third, fourth and fifth costae on the left side accompanied by a *left haemothorax*, which was drained in the local hospital. Transferred first to the neurosurgical unit* with the diagnosis of traumatic paraplegia caused by a suspected dorsal vertebral fracture (T7). The onset of paraplegia here, was *progressive*, appearing within *6 hours* after the accident. On admission, the paraplegia was complete, flaccid, no conus reflexes. As sensation is concerned: incomplete below T7, complete below T10 on the right side; complete below T9 on the left. The

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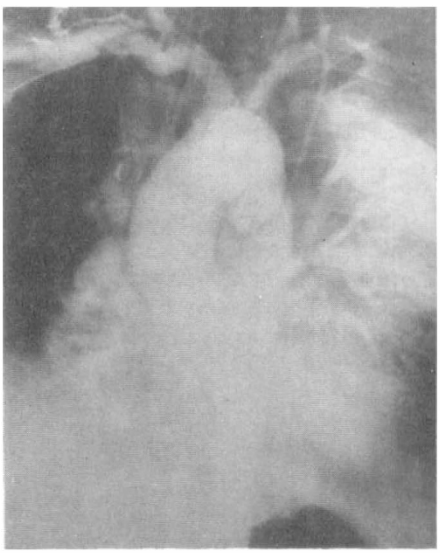

FIG. 1

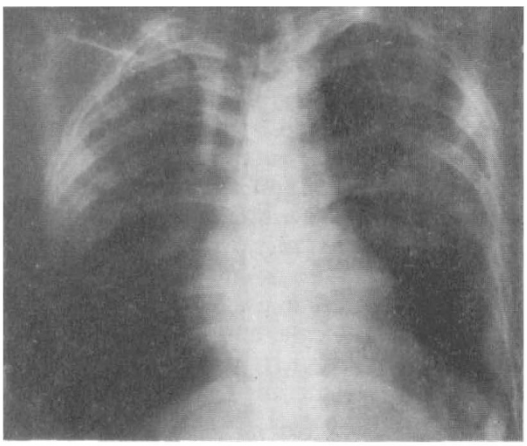

FIG. 2

first X-rays after the accident were, in fact, of bad quality and doubtful as to the presence or not of a vertebral fracture. An air myelography was performed immediately, which was negative and showed no evidence of fracture. He was, then, immediately transferred to the traumatology resuscitation unit, as usually done in the absence of a neurosurgical indication. At the time of his transfer arm blood pressure was 160 mm Hg, pulse 60 p.m. and one noticed *the absence of the femoral pulses*. A new chest X-ray showed the classical image of *the right-sided deviation of the trachea*, a suspect enlargement of the upper mediastinum, a ruptured diaphragm on the left side (Fig. 2). Another sign worth while noting: *the total absence of kidney secretion* in the past 18 hours after the accident, an indwelling catheter having been placed in the first hospital. Transferred to a specialised cardiovascular surgical unit, he was operated upon the same evening but, unfortunately, died of a haemorrhage 12 days after.

Discussion

We think that the last case is particularly instructive for the resuscitation team, the neurosurgeon and the paraplegist. Traumatic rupture of the isthmus of the aorta is the most frequent form of aortic ruptures reported in the literature and has been associated with the knowledge, in most cases of compression and/or deceleration mechanism.

All our cases reported are traffic accidents: paraplegia first incomplete, then rapidly complete, is considered as an *aggravation symptom* and can appear within a few hours to a few days.

Paraplegia as *a syndrome of trauma to the spinal cord* by vertebral injury can be associated with a ruptured aorta (Walsh, 1964; Meinecke, 1968), which can be there the main cause of death.

In the reported cases of delayed paraplegia by aortic trauma, compression by a mural haematoma of the second to the fifth intercostal arteries resulting in an extensive infarction of the spinal cord was shown at necropsy (Hughes, 1964; in Langlois's *et al.* case, a large aneurysm was compressing the descending aorta

and was responsible 15 days after the accident for the 'pseudo-coarctation' syndrome followed by paraplegia 24 hours after. The patient was operated upon and survived but remained paraplegic. Only very few cases of neurological recovery of preoperative paraplegia by aortic trauma are mentioned in the literature (Demuth *et al.*, 1965; Herendeen *et al.*, 1968; Thevenet *et al.*, 1975). The vascularisation of the spinal cord being just sufficient to prevent irreversible damage to the neurons, the condition remaining that these patients are operated upon immediately within a few hours after the accident.

As our three cases are concerned, the possible mechanism would appear to be *a more or less immediate obstruction of the aorta's lumen* at the isthmus, by one of the torn internal coats, thus being responsible for the observed ischaemic syndrome: paraplegia, anuria and absence of blood circulation in the lower limbs.

With less extensive damage to the aorta, the neurological picture can appear to be varied, sometimes very slight indeed, until further reasons (dysphagia, dyspnoea, intrathoracic pain, interscapular systolic murmur, sometimes a Claude Bernard Horner syndrome) can orientate the diagnosis towards an incomplete rupture, sometimes after a long interval.

Conclusion

Although paraplegia as a symptom of aortic trauma can be considered as a rare occurrence, three cases within 10 years in a general hospital seemed to us well worth while mentioning. The diagnosis in the first few hours after the accident can be tricky, even for the most experienced examiner, and demands that the patient must be then transferred as fast as possible to a cardiovascular surgery unit and nowhere else.

Résumé

Trois cas de paraplégie à rupture sous-isthmique de l'aorte au cours d'un traumatisme sont rapportés. Ces trois blessés sont morts et présentaient tous les trois un syndrome de 'pseudo-coarctation de l'aorte' avec anurie et absence de pouls fémoraux. Ces blessés doivent être transférés immédiatement dans un service de chirurgie cardio-vasculaire.

Zusammenfassung

Es werden 3 Fälle von Querschnittlähmung beschrieben die durch eine traumatische Aortenruptur (Pars isthmica) verursacht wurden. Verschiedene Mechanismen, ischämischer Art, Differentialdiagnose, werden erwogen. Alle 3 Patienten starben.

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