Unsuspected chronic subdural haematoma following a motor vehicle accident

A case report

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Summary

A case of unsuspected chronic subdural haematoma following a motor vehicle accident is presented. Attention is drawn to some of the cardinal features of the lesion, viz. its insidious, often unrecognized evolution, its predilection for the elderly and the debilitated, and the difficulty attendant upon precise histological dating of longstanding lesions beyond approximately 6 months' duration. The pathogenesis of the lesion is reviewed.

Chronic subdural haematoma may develop insidiously after insignificant, often forgotten trauma which may have ruptured small vessels crossing the subdural space. Infants, the debilitated and the elderly appear to be particularly susceptible. In the elderly the initial symptoms and signs are often attributed to coexistent cerebral arteriosclerosis. The unsuspected lesion may therefore progress and ultimately lead to death due to increased intracranial pressure. Alternatively the lesion may resorb completely, leaving only a thin yellow membrane. The following case report describes an unsuspected chronic subdural haematoma which eventually led to death.

Case report

In April 1983 the body of an 80-year-old Black man was received at the Government Mortuary in Diepkloof, Johannesburg. Apparently the deceased had been involved in a motor vehicle accident nearly a year before. He had been admitted to hospital after the accident, at which time he was conscious and fully orientated. Lacerations of the left upper lip and face were present, as well as over the left knee and forearm. Neurological examination disclosed bilateral cataracts but no neurological deficit; the skull was not fractured. The patient was admitted for neurological observation and discharged 24 hours later. No further neurological follow-up was undertaken, although the patient was readmitted to hospital 6 months later for orthopaedic manipulation of a fixed flexion deformity of the right knee.

Since the motor vehicle accident he had apparently been bedridden, refusing to eat and often complaining of vague aches and pains, none of them suggesting the existence of a central nervous system lesion. This debilitated state continued until the patient's sudden death at home in bed, there being no apparent precipitating incident to account for it.

In view of the fact that the deceased had been involved in a motor vehicle accident a year previously and had been bedridden ever since, the body was submitted to the Government Mortuary. At autopsy the following features were noted: The body was that of an old, extremely emaciated Black man, weighing 29 kg and with a length of 1.60 m. Postmortem lividity was present over the back of the body, which also showed a fixed flexion deformity of the right knee and bedsores over the right buttock. Internal examination disclosed little of note other than marked organ atrophy. However, intracranial examination revealed the following: A dural neomembrane was present over the entire left cerebral hemisphere, this membrane varying in thickness from 0.2 to 0.4 cm. The area of maximal thickness overlay the left temporoparietal region and was adherent to the underlying cortex over an area measuring 3 x 4 cm. The cerebral hemispheres appeared swollen with flattening of the gyri and narrowing of the sulci; some ventricular compression appeared to be present with a mild degree of tentorial herniation (as judged by shift of the uncus more than 2 mm medially). Histological examination of the dural neomembrane (Fig. 1) revealed relatively hypocellular connective tissue in which both haemorrhage and haemosiderin were noted. Further sections, which included the underlying brain (Fig. 2) disclosed a number of blood vessels extending into the cerebral sulci; the underlying brain showed some oedema of the white matter. In summary, the histological features were those of a subdural haematoma which had been present for a period of about 1 year (consistent with the clinical information), as well as accompanying cerebral oedema.

Fig. 1. Photomicrograph of the subdural membrane showing laminated structure and area of fresh haemorrhage (H and E x 32).
In essence, this case represented some of the classic features of the chronic subdural haematoma with its insidious, often unsuspected evolution, its occurrence in an elderly and debilitated person, and the difficulty attendant upon attempts at precise histological dating of the lesion beyond a certain time interval.

Discussion

Subdural haematomas were described as long ago as 1561 by Ambroise Paré, and the entity of chronic subdural haematoma was described by Wepfer in 1681. Subdural haemorrhage occurs into the space between the dura and the arachnoid, and may occasionally be non-traumatic, as a result of hypernatraemia and other hyperosmolar conditions. Although the source of traumatic haemorrhage is variable, in most instances it originates from tears in one or several bridging veins, i.e. those veins which pass through the subdural space before entering their respective dural sinuses. Less frequent origins of subdural haemorrhage include intracerebral haemorrhage with extension of blood into the subdural space, the tearing of arterial or venous channels from contused areas at the brain surface and the tearing of dural sinuses associated with penetrating wounds.

Experimentally, subdural haemorrhage from parietal bridging veins in animals subjected to subconcussive blows has been observed; should the animal survive, the tear is usually sealed by a fresh thrombus which on rare occasions may spread over larger areas of the vein or even extend into the sinus. In addition, ectopic bridging vessels, i.e. those vessels which bridge the subdural space remote from a sinus, are especially vulnerable to mechanical stress; the fact that they are located over the lower parietal lobe may possibly account for the frequent observation of haemorrhage in the temporoparietal region. The further significance of these bridging veins is that they may be accompanied by a small arterial branch, and hence their tearing may result not only in venous haemorrhage but also in arterial bleeding; this mechanism was ascribed by one author as responsible for the arterial origin of 11% of the cases of subdural haematoma treated by him. In the absence of ectopic bridging vessels, arterial haemorrhage may occur from a small cerebral artery through a tear in the arachnoid; this is usually an arterial twig from a branch of the middle cerebral artery lying at a right angle just beneath the arachnoid. In one series almost all such tears occurred in the temporoparietal region, a site of predilection for subdural haematomas.

Many explanations have been put forward to account for the latent interval between the head injury and the development of clinical symptoms. Obviously, the slower the accumulation of blood in the subdural space the greater the amount which can be tolerated before symptoms develop. However, the chronic subdural haematoma is believed to cause symptoms as a result of progressive enlargement due to an increase in osmotic pressure within the haematoma. Although this may be a result of haemolysis of erythrocytes and disintegration of plasma proteins within the lesion, providing the osmotic stimulus to fluid imbibition and having the secondary effect of lesion enlargement and cerebral compression, many authors feel that the essential cause of the delayed increase in volume of the haematoma is either repeated bleeding from the original source or haemorrhage from thin-walled new vessels within the organizing granulation tissue.

An important diagnostic feature of subdural haematomas is their progressive metamorphosis with time. However, because the subdural space has limited absorptive capacity, the blood clot encapsulates and becomes organized. Furthermore, in the subdural space this burden of organization is thrown upon poorly vascularized connective tissue in contact with one surface of the clot — this is in contrast to accumulations of blood in serous body cavities, where organization takes place from all surrounding surfaces.

As a subdural haematoma evolves through several stages its age can be assessed histologically, enabling one to determine the time of occurrence of the injury. However, since formation of granulation tissue does not begin everywhere along the dural surface at the same time and at the same rate, it is advisable to examine several specimens of dura from different areas. However, difficulties in timing the injury occur when newly formed blood vessels attempting to organize the haematoma rupture, giving rise to fresh haemorrhage (as in this case), which also has the secondary effect of enlarging the haematoma by the mechanism alluded to above. Further difficulties arise with longstanding lesions, and beyond approximately 6 months' duration it is impossible to estimate the age of the lesion; calcification or ossification of surrounding membrane merely indicates that it is several months or even years old. Similarly, dead blood showing cholesterol clefts may be present after years and is indicative of long duration of the lesion.

The complication of cerebral oedema and temporal lobe herniation

Cerebral oedema denotes an abnormal increase in the water content of the central nervous system. If water accumulates within cells it is referred to as cytotoxic oedema, in contrast to extracellular water accumulation or vasogenic oedema. A third category of oedema is interstitial oedema, characterized by an increase of sodium and water in the peri-ventricular white matter; this is commonest in association with obstructive hydrocephalus.

The commonest form of oedema is vasogenic oedema due to leakage of plasma and water directly into the central nervous system through or between damaged capillary endothelial cells; fluid then collects in the extracellular space. The significance of this oedema is that it may displace the brain, causing various forms of herniation, such as subfalcial, central (diencephalic), temporal lobe (uncal, parahippocampal gyrus) and foramen magnum herniation. The degree and exact pattern of herniation will vary according to the nature and location of the pathological process; symmetrical herniation will usually occur with generalised brain swelling or with masses situated close to the mid-sagittal plane, whereas more pronounced herniation of one temporal lobe will tend to accompany lateralized lesions and may be especially striking in the case of masses situated far laterally.
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REFERENCES

Rekenaartomografiese diagnose van intratorakale lipoom

'n Gevalsbespreking

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Summary

A case of intrathoracic lipoma in a child is presented. The diagnosis was made before operation by means of computer-assisted tomography, a non-interventional procedure.


'n Vier-en-'n-halfjarige dogter wat vir 10 maande by 'n buitekliniek behandeling ontvang het vir pulmonale tuberkulose het met opvolg- röntgenologiese ondersoek 'n homogeneity opsteek in die onderlongsplek getoon. Die 8ste en 9de tussenribbies was verwyd. Die mediastinum was na regs en die linkerhemidiafragma na onder verplaas. Geen hilus of mediastinale kliere kon waargeneem word nie. Die beeld was beskou as verdag van 'n intratorakale massa (Afb. 1).

Rekenaartomografie is vervolgens uitgevoer en hieruit is vasgestel dat daar 'n groot massa in die linkerhemitoraks aanwezig was met 'n digtheidmeting van -100 Hounsfield eenhede (HE). Dit dui op vetweefsel, en die diagnose van 'n lipoom in die grootte van 'n intratorakale massa (Afb. 2).

Tydens operasie is daar 'n groot ekstrapulmonale lipoom in die linkerhemitoraks gevind. Om die massa te verwys, moes gedeeltes van drie ribbes saam verwys word. Histologiese ondersoek het die diagnose van 'n lipoom bevestig.

Bespreking

Intratorakale lipomata is seldsaam.1,2 Die ouderdomsinsidensie van gerapporteerde gevalle strek vanaf 11 maande tot 66 jaar. Presenterende tekens en simptome hang af van die grootte van die tumor.3 Die meeste pasiente is asymptomies en die afwykings word meestal tydens roetine-borskasopnarnes waarneem. Sekere radiologielse tekens mag van beperkte waarde wees by die diagnose van intratorakale lipome, nl. verandering in die vorm van die letsel tydens asetnhaling,4 lae digtheid as gevolg van die vetinhoud, periferie van massa meer radiodeursigtig as sentrale dele,5 en rib erosie as gevolg van druk.6 Intratorakale lipomata kan ekstrapleuraal of intrapulmoner geleë wees. Ekstrapleuraal lipomata is seldsaam en intrapulmonale lipomata is uitslegs seldsaam. Volgens Keesey7 kan intratorakale ekstrapleuraële lipome as volg ingedeel word: (i) algehele intratorakale lipoom — waar die tumor geheel en al in die torakale holte geleë is; en (ii) sandloper-intratorakale lipoom — waar daar beide 'n intra- en ekstrapulmonale komponent van die tumor is, nl. (a) mediastinoservikaal — waar die massa vanaf die mediastinum in die nek strek; en (b) transmuraal — waar die tumor deur die torakswand streek.

Intratorakale lipomata, soos lipomata elders, is by uitstek benigne letsels hoewel dit al beweer is dat 'n liposarkoom uit 'n lipoom mag ontstaan.8 Ander auteurs beweer egter dat liposarkoom as 'n primêre tumor ontstaan.9