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Chronic subdural haematoma

One of the physician's, and especially the geriatrician's, nightmares is to find himself in a coroner's court with the pathologist describing finding a chronic subdural haematoma in a patient in whom a confident clinical diagnosis had been made of senile dementia or cerebrovascular disease. Most neurosurgeons have learnt the lesson that once the diagnosis of chronic subdural haematoma has been even whispered then no matter how unlikely it may seem they have no choice but to pursue it—if necessary to the stage of exploratory burr holes. These anxieties have been fed by tales of dramatic cure in moribund patients by the simplest of surgical procedures, and, though some of these accounts might be apocryphal, Cameron's recent valuable review of 114 cases of chronic subdural haematoma[1] substantiates the rewarding results that may be achieved. The newly appointed neurosurgeon trying to establish his reputation can, therefore, still dream of attending a public figure with a chronic subdural haematoma.

The problems in clinical diagnosis stem from several factors. These include aetiology and pathophysiology, the evolution of the haematoma, and the wide variation in reaction of the often aging brain to the presence of the haematoma and the consequent lateral and vertical distortion and displacement of the cerebral hemispheres and midbrain. Furthermore, these factors vary considerably, not only from one patient to another, but at different stages and times in the same patient. Perhaps the starting point of the diagnostic conundrum should be the importance of trauma in aetiology—variously estimated by different authors[2 3] as relevant in 50%-71% of cases. The trauma is so often mild, and so easily forgotten or overlooked by the aging patient, that its true place is difficult to estimate. Indeed, the distinction between the traumatic and the "spontaneous" haematoma (such as one following a severe bout of coughing) is largely artificial: the more important factors are the low intracranial pressure, a mobile brain, and stretched fragile cortical veins traversing the subdural space.

The most frequent presenting symptoms in reported series have been intellectual deterioration and change in personality. The relative infrequency of symptoms and signs of raised intracranial pressure (headache and papilloedema) contrasts with other intracranial space-occupying lesions and increases the problems of diagnosis, particularly when the patients have previous evidence of cerebrovascular disease. Why this lack of evidence of raised intracranial pressure should be peculiar to chronic subdural haematoma has been much debated; the explanations have included the gradual development of shift of the cerebral hemisphere and variation in regional cerebral blood flow.[4] The classic fluctuation in symptoms, and particularly in level of consciousness and intellectual state, occurs in up to one-third of patients, but the traditional explanation—osmotic effects of the haematoma—has been neither confirmed nor refuted.[5 6] Nevertheless, the recent finding of a high output of tissue plasminogen activators from the outer membrane of chronic subdural haematomas supports the view that fibrinolysis plays an important part in their persistence and progressive enlargement.[7] The duration of symptoms has at times been exaggerated, so that Cameron's finding of an average of 63 days in patients with bilateral haematomas is helpful. Indeed, the history rarely exceeds three months, a useful point in differentiation from senile or "cerebrovascular" dementia. Hemiparesis occurs in only about a quarter of patients, and clearly its absence cannot be relied on to differentiate between chronic subdural haematoma and the senile dementias.

What are the implications for the general physician of these problems of clinical diagnosis, and have recent advances in methods of investigation eased his burden? Much will depend on his attitude towards diagnosis and whether it is based on probability rather than on a duty totally to exclude a particular diagnosis. With chronic subdural haematoma he can be forgiven for the latter approach in view of the excellent results of what is almost minor neurosurgery not requiring a general anaesthetic. There is a school of neurosurgical thought which has favoured non-surgical management of these patients, with or without osmotic diuretics and steroids,[8–10] but such is not the view of most British neurologists and neurosurgeons, and the physician should not allow that minority view to influence his clinical judgment.

The plain skull radiograph of good quality is the first essential of investigation, because the likelihood of visible pineal calcification increases with age. Once the pineal has been identified on the lateral radiograph, then its central position on the anteroposterior radiograph goes a long way towards excluding the presence of a clinically important haematoma: gross lateral shift is characteristic of the significant lesion and "balanced" bilateral lesions are rare. Radioisotope scanning may show a chronic subdural haematoma, but it can never be relied on to exclude one.

For the physician who has ready access to computerised axial tomography the anxieties of clinical diagnosis will be

greatly relieved. The CAT scan has a high degree of accuracy in the diagnosis of chronic subdural haematoma since it clearly shows displacement of the hemisphere even when the actual haematoma may be "isodense" and therefore not visible.[11] But CAT scanning requires, firstly, a co-operative, placid patient (and failing that one who is anaesthetised if not already in coma), and, secondly, immediate access to CAT scanning time, which is still at a premium in many parts of Britain. Otherwise there is still a place for management by plain skull radiographs and exploratory burr holes, especially when the physician knows that for reasons of the patient's age and infirmity his neurosurgical colleagues will not approach other intracranial lesions. The indiscriminate referral of every aging and infirm patient with progressive or fluctuating dementia for CAT scanning to exclude a chronic subdural haematoma reflects both clinical and economic irresponsibility.

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Plasmapheresis and severe glomerulonephritis

Interest in the technique of plasmapheresis (or plasma exchange), which we considered less than a year ago,[1] remains high, especially as a possible treatment for severe glomerulonephritis.[2–4] Those rare patients with periglomerular proliferation leading to "crescents" of periglomerular cells may have a rapid onset of renal failure and never recover renal function, yet we have no clear outline of how they should be managed.

The most nihilistic view is that drugs and other treatment may only damage a patient, who if she or he needs it might receive a successful allografted kidney or maintain life well on home haemodialysis. At the other extreme is energetic treatment of all patients with crescentic glomerulonephritis even if they are anuric and receiving dialysis. One treatment that has been advocated is plasma exchange, usually with some form of immunosuppression, to remove antibody, immune complexes, and immune reactants such as complement and fibrinogen from the circulation. General guidelines on the use of plasmapheresis in severe nephritis are now emerging, though many questions remain unanswered.

Clinically severe glomerulonephritis—even requiring dialysis in the acute stage—may be completely reversible without specific treatment, especially if the inflammatory changes are confined within the glomerular capillaries. Nevertheless, the more diffuse and extensive the degree of extracapillary periglomerular proliferation, the poorer the prognosis. The watershed where recovery of function becomes less likely than progression into renal failure is not sharp but lies somewhere about the point where 60-70% of glomeruli show enveloping crescents. Renal biopsy, therefore, is an essential part of the management of patients with severe nephritis to establish both the nature and the extent of the glomerular damage.

Even within the group of patients with severe nephritis and extensive crescent formation the accompanying glomerular changes may differ.[2–4] Many glomerular immune deposits may be found; or the glomeruli may appear, in contrast, almost normal apart from the crescent. Particularly in this latter group there may be an associated arteritis. Occasionally the setting for severe crescentic nephritis may be systemic lupus erythematosus (SLE) or Henoch-Schönlein purpura. All these patients with crescentic glomerulonephritis are believed to suffer from vascular and glomerular deposition of circulating antibody-antigen complexes. There is one more group, in whom the lesions arise from glomerular binding of antiglomerular basement membrane (anti-GBM) antibodies.[5 6] The condition may present as isolated nephritis or associated with pulmonary haemorrhage (Goodpasture's syndrome). It is often forgotten that the conjunction of lung haemorrhage and nephritis may be found occasionally in association with severe soluble complex disease, such as poststreptococcal glomerulonephritis,[7] SLE,[8] and Henoch-Schönlein purpura,[9] as well as in polyarteritis. The clinical diagnosis of Goodpasture's syndrome is not synonymous, therefore, with anti-GBM disease, which is defined by the presence of circulating anti-GBM antibody and a continuous linear deposit of immunoglobulin (usually but not invariably IgG) along the glomerular capillary basement membranes.

What can plasma exchange offer these patients? In anti-GBM nephritis the synthesis of antibody seems to stop after a few weeks or months in most patients,[5] and intensive plasma exchange can deplete antibody concentrations for days or weeks,[6 10 11] as well as removing agents of inflammation such as complement from the circulation.[6 10 11] Renal disease improves dramatically in most patients who still have some function at the beginning of treatment, and the relief of pulmonary haemorrhage has been even more consistent and dramatic.[12] Indeed, plasmapheresis is justified whatever the severity of the renal disease in patients with life-threatening haemorrhage. A good renal response is not, however, invariable: even in the hands of the groups working at the Hammersmith Hospital[6 11 13] and in Melbourne[14 15] (who between them have treated almost 50 patients with anti-GBM nephritis by plasma exchange) patients already anuric failed to recover renal function.

Are these results better than those from simple immunosuppression[5 16] or supportive treatment[5 16] only? Occasional cases of spontaneous recovery in renal function, even from anuria, have been described in anti-GBM nephritis,[17 18] and the disease may be indolent, with many years of minor proteinuria or haemoptysis.[5 19] The regular recovery of renal function reported from London[6 11 13] and Melbourne[14 15] is far better than the usual behaviour of the disease in its severe forms as previously documented.[5 16] Other encouraging reports have been published[20–22] but not every group has met with equal success,[10 22 23] though no one else has such a large experience of managing these patients, and Kincaid-Smith and D'Apice[14] emphasise its importance in achieving success. But pulmonary oedema and intercurrent infection[24] may cause exacerbations of the renal or pulmonary disease unrelated to anti-GBM titres, and infection is particularly likely in immunosuppressed individuals.

So far concomitant immunosuppression has been given as a routine. (Research on animals given primary immunisation has shown a rebound to higher titres of antibody after plasma