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Lesson of the Week

Pituitary apoplexy

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Pituitary apoplexy may occur without evidence of prior endocrine dysfunction and may simulate aneurysmal subarachnoid haemorrhage

Pituitary apoplexy, a complication of pituitary adenomas, is characterised by sudden headache, visual impairment, and ophthalmoplegia and is usually associated with haemorrhagic infarction of the tumour. This condition may be mistaken for aneurysmal subarachnoid haemorrhage, and the true diagnosis may be delayed or even remain unsuspected because the underlying pituitary adenoma is frequently not clinically apparent.¹ We describe a case of pituitary apoplexy in which urgent computed tomography (CT) and cerebral angiography were essential to establish the diagnosis.

Case history

A 61 year old right handed man with no medical history of note suffered sudden onset of giddiness, vomiting, and headache, which worsened over the next two days. On admission soon afterwards he was stuporous and dysphasic with a complete third nerve palsy on the left. Visual fields could not be tested accurately because of poor comprehension, but other cranial nerves and fundi were normal. He had photophobia and mild neck stiffness but no pulsatile exophthalmos and no cranial or vascular bruits. There was spontaneous movement in all limbs, tone was normal, reflexes were brisk and symmetrical, and plantar responses were flexor. He was in sinus rhythm at 90 beats/min, heart sounds were normal, and blood pressure was 160/90 mm Hg. He had a fever of 38.4°C; respiratory system, abdomen, thyroid, and body hair distribution were all normal. Chest x ray appearances and an electrocardiogram were normal.

He was initially suspected of having an aneurysmal subarachnoid haemorrhage with a left temporoparietal haematoma. Skull radiographs, however, showed a slightly expanded pituitary fossa with possible early erosion of the anterior clinoid process and the petrous apex on the left. CT of the head showed high density material in the pituitary fossa and suprasellar region extending into the left sylvian fissure. With contrast enhancement a small oval structure was visualised in the left suprasellar region. This appearance was consistent with either subarachnoid haemorrhage from a parasellar aneurysm or pituitary apoplexy. Next day he continued to deteriorate with deepening stupor, loss of all eye movements on the left, progressive right hemiparesis, and a fall in blood pressure to 90/60 mm Hg. He was given intravenous dexamethasone and volume repletion and subjected to urgent bilateral carotid angiography via a right femoral approach. Displacement of vessels on the left indicated a lesion above the pituitary fossa which extended superiorly and laterally towards the temporal fossa and which was consistent with pituitary apoplexy. Immediate left

frontoparietal craniotomy was performed. A large haemorrhagic tumour was seen arising from the pituitary fossa, encircling the optic chiasm and optic nerves, and extending leftwards, lateral to the internal carotid artery and the cavernous sinus, back to the membrane of Liliequist. It was evacuated by Cavitron ultrasonic suction apparatus with preservation of the optic chiasm and nerves. Histologically the pituitary tissue showed extensive haemorrhage and small numbers of uniform adenoma cells with negative immunocytochemical staining for prolactin and growth hormone.

Postoperatively the patient was given replacement therapy with hydrocortisone, thyroxine, and testosterone. Two weeks and also two years later triple bolus testing showed no appreciable function of the anterior pituitary, so replacement therapy was continued in the long term. Diabetes insipidus was not a problem, with a normal rise in urinary osmolality on water deprivation. His left third and fourth nerve lesions persisted after more than two years, but there was recovery of the sixth nerve paralysis. Visual field testing showed an incomplete right homonymous hemianopia with macular sparing, and the remaining left temporal visual field was reduced. Corrected visual acuities were right 6/7.5, left 6/12. Both optic discs, particularly the left, were paler than normal. He was troubled by mild nominal dysphasia and memory disturbance but the right hemiparesis had resolved. He gave no retrospective history of endocrine dysfunction antedating his episode of pituitary apoplexy. Serial CT scans of the head showed evidence of old infarction in the left temporal lobe but no recurrence of the pituitary tumour.

Discussion

Pituitary apoplexy is becoming increasingly recognised as a complication of pituitary adenomas,² occurring in over 10% of cases.¹ Manifestations typically include headache, meningeal irritation, and visual loss or ophthalmoplegia, or both, with additional features depending on the extent of spread of hypophyseal contents and haemorrhage. In our patient lateral spread caused ophthalmoplegia, whereas spread to the temporal lobe resulted in prolonged dysphasia and memory disturbance. Clinically pituitary apoplexy may be difficult to differentiate from aneurysmal subarachnoid haemorrhage and it may be unsuspected because signs and symptoms of prior endocrine dysfunction are absent in most cases.¹ Occasionally pituitary apoplexy develops slowly over several weeks, but usually it follows a similar time course to that of aneurysmal subarachnoid haemorrhage and evolves fully within two days.³

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Third, fourth, and sixth nerve lesions may all occur in pituitary apoplexy, but third nerve lesions are well known in association with posterior communicating artery aneurysms; and a combination of third, fourth, and sixth nerve lesions may occur with aneurysms of the intracavernous portion of the internal carotid artery. Even so, the presence of cranial nerve palsies, particularly if multiple, should suggest the possibility of pituitary apoplexy with the consequent urgent problem of hypopituitarism.

Radiological abnormality of the pituitary fossa in our patient suggested a diagnosis of pituitary apoplexy. This, however, could not be accepted without investigation to exclude other conditions. For example, coexisting intracranial aneurysms with the potential to rupture may be found in 7% of cases with pituitary adenomas,¹ and deformity of the fossa may also result from an intrasellar or parasellar aneurysm in the absence of a pituitary tumour. CT is often enough to confirm a diagnosis of pituitary apoplexy, but the scan may initially be normal in patients with small pituitary tumours in whom radiographs of the pituitary fossa are also normal.⁴ In our patient the appearance above the pituitary fossa on contrast enhancement of the scan raised the possibility of a parasellar aneurysm, though this was later excluded by urgent cerebral angiography.

The clinical course of pituitary apoplexy is unpredictable but varies from mild cases which may remain undiagnosed^{1,2} or which improve with conservative management⁴ to severe cases like ours, which are frequently fatal if left untreated.³ Ophthalmoplegia often resolves spontaneously, but

recovery of vision is optimised by early decompression. The partial recovery seen in our patient, even after prompt neurosurgical intervention, reflected the severity of the initial lesion.

Pituitary apoplexy has no predilection for any particular type of adenoma.¹ The failure of pituitary tissue to stain for prolactin and growth hormone together with the absence of any clinical features of Cushing's disease or thyroid dysfunction suggest that our patient probably had a non-functional adenoma. Further immunocytochemical staining for adrenocorticotrophic, luteinising, follicle stimulating, and thyroid stimulating hormones, however, would have been necessary to confirm this. Multiple pituitary hormone deficiencies occur in most patients after pituitary apoplexy and if unrecognised may contribute to morbidity and mortality. The transient fall in blood pressure noted in our patient before the diagnosis was confirmed may have resulted from deficiency of adrenocorticotrophic hormone and cortisol. Our patient was typical in that prolonged hormone replacement therapy was necessary.³

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WORDS

● **TISSUE** I could never remember which was the warp and which the weft (or woof) until I discovered that "warp" derived from the same root as the German *werfen*, to throw. Therefore, "warp" signified the lengthwise stretched fibres on a loom, whence by elimination the crosswise fibres carried by the shuttle were the weft, which term is derived from the same root as "weave." A **WEB** is that which is woven. In clinical terms it is a membrane bridging a space, as the oesophageal web, or the abnormal fold of skin between digits (webbed fingers/toes), such as occurs normally in aquatic birds. Web, weave, and weft are Teutonic in origin.

The Latin *texere*, to weave, has given us "textile," "texture," and one of the most commonly used words in medicine, **TISSUE**, defined as a collection of similar cells and the intercellular substance surrounding them.¹ **TISSUE PAPER** is thin soft gauze-like unsized paper for wrapping delicate articles, and modified for clinical use to take advantage of its capacity for absorbing liquids. A single sheet of this paper is a **TISSUE**. I prefer the pronunciation tiss-yoo to the equally popular tish-oo, for fear of being branded a punster, having regard to its emunctory use after sternutation. A popular brand is sold as "Kleenex for Men"; the mind boggles. Connected with *L. texere* is *L. tela*, a web, as in the anatomical term "tela choroidea

inferior," the delicate web-like pial membrane that forms the roof of the fourth ventricle of the brain. And where would we be without **TEXTBOOKS**? A text is the "tissue" of a literary work (*Oxford English Dictionary* definition)—that is, the words in their original form. A textbook is a standard work for the study of a particular subject. For medical publications, however, "standard" in this definition is weakened by the multiplicity of rival textbooks, and by their ephemeral life expectation—such is the pace of expansion of information to be recorded. The "book" of textbook derives from the Teutonic for beech, as very early writings were made on the wood of this tree. In German the terms are almost identical: *Buch*, book and *Buche*, beech. Thus a beech forest is *Buchenwald* (now of ill repute). The German for tissue is *Gewebe*, which encloses the "web" moiety.

Finally, we come to Greek. Ancient Greek, in the area of weaving, has given medicine **HISTOLOGY**, the study of the minute structure of tissues; also its derivatives **HISTOPATHOLOGY**, **HISTOCHEMISTRY**, **HISTOCOMPATIBILITY**, and so on; also the fungus *histoplasma capsulatum*, and **HISTOPLASMOSIS**, the disease caused by it. Another branch from the stem yielded **HISTAMINE**, coined in 1913 by Barger and Dale, who separated this substance from the tissue of intestinal mucosa. It is a powerfully vasoactive

substance released from many tissues and in particular from mast cells. Their choice of name was based on the related aminoacid **HISTIDINE**, so named by Kossel in 1869, on extracting it from sturgeon's sperm (*Oxford English Dictionary Supplement*). All these words stem from the Greek *histos*, which piece of etymological wayfaring I deferred till now. *Histos*, not surprisingly, means "web," but it also means the mast of a ship. It was adopted for the beam of a loom to which the warp was fixed, which in ancient times stood upright like a mast. Thus, web, weave, tissue, and histology are branches of the same semantic tree. It is *histos*, the mast, that gives us **HISTOGRAM**, a graphic device in which numerical values are represented by vertical rectangular bars.

This article should end here. To avoid possible confusion, however, between the above mentioned **MAST** cell, and the ship's mast, *histos*, I must add that the mast cell gets its epithet from the German *Mast* (pronounced "must"), meaning food, because it is packed with basophil granules of vasoactive substances, of which histamine is clinically important in acute allergic reactions. A mast cell with its granules looks, under the electron microscope, like a polythene bag full of cherries. —B J FREEDMAN

1 *Stedman's medical dictionary*. 24th ed. Baltimore: Williams and Wilkins, 1982.