Bilateral carotid aneurysms unmasked by severe hypopituitarism

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Summary: We describe a patient who initially presented with severe hyponatraemia and grand mal seizures, without any focal neurological symptoms. The final diagnosis was that of giant bilateral carotid aneurysms extending into the sella turcica with anterior hypopituitarism. To the best of our knowledge, this is the first case report of symmetrical carotid aneurysms manifested exclusively by an acute endocrine emergency with none of the concomitant usual focal signs such as headache, failing vision, oculomotor palsy or subarachnoid haemorrhage.

Introduction

Carotid aneurysms mimicking pituitary tumours are responsible for parasellar syndromes with neurological and mass effect symptoms dominating the clinical presentation.1-5 The patient reported is exceptional in two ways. Firstly giant bilateral intracavernous carotid aneurysms were present and secondly the grand mal seizures leading to diagnosis resulted from severe hyponatraemia related to an anterior hypopituitarism, with none of the usual symptoms of parasellar syndromes.

Case report

A 73 year old woman was transferred to our hospital because of grand mal seizures and persistent hyponatraemia. Nine months earlier, she had been admitted to another hospital for transient diplopia resulting from sudden palsy of the right external oculomotor muscle. The only neurological investigation performed was a normal electroencephalogram. The diplopia disappeared within a few days, with a diagnosis of transient cerebral ischaemia. Five months prior to admission, the patient noted progressive weight loss, increasing fatigue and severe orthostatic dizziness with falls. During this period, she was hospitalized three times in another hospital, where hyponatraemia (115–125 mmol/l) was repeatedly documented but remained without valid explanation. When increasing confusion and grand mal seizures occurred, the patient was finally transferred to our hospital.

On arrival, the patient was alert. Physical examination disclosed no abnormality, except weak but equal tendon reflexes. There was no diplopia. Serum sodium was 125 mmol/l with inappropriate renal sodium losses (113 mmol/day in the 24 h urine collection), potassium was 3.2 mmol/l (28 mmol/day in urine), plasma osmolality was low at 262 mOsm/kg with diluted urines (386 mOsm/kg in the 24 h collection). The urea nitrogen was 3.2 mol/l, creatinine 106 μmol/l and uric acid 0.17 mmol/l. There was a slight normochromic normocytic anaemia with a haemoglobin at 104 g/l. An X-ray film of the chest was normal, while the standard skull X-ray showed an enlarged sella turcica due to bony destruction of the floor of the sella and of the anterior and posterior clinoids.

The provisional symptomatic treatment included restriction of fluid intake, administration of intravenous hypertonic saline and frusemide, and of oral urea. Thyroid function tests showed: thyroxine 55.0 nmol/l (normal 80.6–154), triiodothyronine 0.51 nmol/l (normal 1.3–3), TSH 0.14 mU/l (normal 0.2–5.5), an 08.00 h plasma cortisol was 143.5 nmol/l (normal 248–690) and a free 24 h urinary cortisol 65 nmol (normal 55–276); LH was <0.4 mg/l (normal post menopausal >5.5) and FSH 0.5 mg/l (normal post menopausal >11); GH was <0.75 mg/l (normal <5.5) and somatomedin C 0.2 U/ml (normal 0.4–1.5), prolactin was 1400 mU/l (normal <240). Because of the critical clinical situation of the patient no further dynamic tests were performed, except a diagnostic and therapeutic intramuscular injection of corticotrophin (Synacthen depot® 2 mg). Plasma cortisol peaked to high values within 2 days, while serum sodium normalized and 24 h natriuria fell to low values.
The neuroradiological images are shown in Figure 1. Computed tomography scan demonstrated an intra- and parasellar non-homogeneous space occupying lesion. The tumour enhanced homogeneously and vividly after intravenous contrast injection. A digital angiography demonstrated that the mass resulted from bilateral intracavernous giant carotid aneurysms extending into the sellar cavity. In a magnetic resonance imaging (MRI) study, a series of sagittal T1 weighted images as well as two series of coronal T2 weighted images centred on the sellar region were obtained: bilateral aneurysms of the carotid siphons could specifically be diagnosed due to the characteristic black appearance of the lesions on all images (flow void phenomenon). Fundoscopic examination, visual acuity and visual field testing were normal.

Surgical treatment was considered unreasonable and discarded since it implied ligation of both internal carotid arteries. Medical treatment with cortisone acetate 25 mg and l-thyroxine 125 μg daily rapidly resolved the hyponatraemia and abolished all symptoms presented by the patient

**Discussion**

The present case report illustrates the exceptional situation where bilateral intracavernous carotid aneurysms presented as a severe hyponatraemia resulting from anterior pituitary insufficiency.

Symptomatic hyponatraemia, as initial symptom of hypopituitarism is, by itself, unusual, although classically described. In our own experience, however, hyponatraemia was the symptom leading to hospitalization and diagnosis in 12 out of 40 patients with complete pituitary insufficiency (personal data). The hyponatraemia in such situations is generally attributed to an increased secretion of ADH and greater sensitivity to its action of the collecting tubules resulting from glucocorticoids and thyroid hormone deprivation, although hyporeninism has also been held responsible for abnormal sodium losses.

Hypopituitarism results in the majority of cases from hypophyseal tumours and/or Sheehan's syndrome in older series, amounting to 59% of all cases in a report collecting 1060 pituitary insufficiencies. The next most frequent aetiologies are intra- or parasellar tumours such as craniopharyngiomas, meningiomas and germinomas, trauma, irradiation, and previous surgery in the sellar region.

Carotid artery aneurysms mimicking pituitary tumours are uncommon but well described. They can account for up to 10% of the lesions causing a parasellar syndrome, while 1.4% to 5% of all intracranial aneurysms referred to neurosurgeons projected into the sella turcica in other series.

However, pituitary dysfunction is thought to be rare in patients with intracavernous aneurysms of the internal carotid artery, the absence of endocrine symptoms even being considered as an important diagnostic feature to differentiate aneurysms from pituitary adenomas. Aneurysms were not described as cause of hypopituitarism in a review by Sheehan and Summers. More recent literature did, however, report the coincidental occurrence of...
intracavernous aneurysms, when the aneurysms are, however, exceptional. Angiography is systematically performed in order to rule out this uncommon aetiology, particularly when a transsphenoidal approach of the lesion is planned. MRI appears to be a very valuable diagnostic tool as it shows vascular elements without any contrast injection: flow sensitivity is indeed one of the major advantages of MRI. Rapid flowing blood appears usually as areas without any signal intensity while slow flowing blood appears hyperintense (paradoxical enhancement). As a consequence, MRI allows easy differentiation between carotid arteries and cavernous sinus, and an aneurysm is demonstrated by a signal void black area while if thrombosed, a typical high signal intensity region identifies the lesion.
References

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