Marked Hyperprolactinemia Caused by Carotid Aneurysm

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ABSTRACT: Background: Pituitary dysfunction caused by intracranial aneurysms is rare. We report a patient with the unique feature of hyperprolactinemia to a degree previously seen only with prolactin-secreting tumours. Method: Case report. Result: A 42-year-old woman had a galactorrhea, left-sided headache, reduced vision in the left eye and a left temporal hemianopsia. Serum prolactin was elevated (365 µg/L). Cranial computed tomography (CT) revealed a suprasellar mass, which carotid angiography showed to be a left internal carotid artery aneurysm. At craniotomy, this aneurysm and a smaller one of the ophthalmic artery were repaired, and the patient’s vision returned to normal. The prolactin level fell to normal. Follow-up CT showed no evidence of pituitary adenoma or hypothalamic lesion. Conclusions: Carotid aneurysm can cause reversible pituitary dysfunction. A prolactin level >300 µg/L is not a reliable cut-off for distinguishing prolactin-secreting adenomas from other causes of elevated prolactin. A co-existing prolactinoma was felt to be ruled out by both a normal CT scan and normal prolactin levels following aneurysm repair. Patients with marked hyperprolactinemia should be considered for angiography or MRI to rule out carotid aneurysm, since the consequences of pituitary exploration in this setting are potentially grave.


Carotid aneurysm is a rare cause of pituitary dysfunction.1-3 Because the clinical presentation may differ little from that of a pituitary tumour, it is crucial to diagnose the aneurysm prior to surgical exploration of the sella.

We report the case of a patient with pituitary dysfunction caused by a giant carotid aneurysm, in whom a unique feature was hyperprolactinemia to a degree previously accepted to be pathognomonic of a prolactin-secreting tumour.4

CASE REPORT

A 42-year-old woman was admitted to our institution because of galactorrhea of four months duration. Past medical history included five normal pregnancies and a hysterectomy for uterine fibroids. Two years prior to admission, she noted intermittent left hemicranial headaches which radiated to her left eye. One year later, she developed blurred vision in the left eye. Six months before admission, she noted decreased libido and a 25 pound weight loss followed by spontaneous bilateral galactorrhea. She took no medications, and denied polyuria, polydipsia, cold intolerance or changes in her appearance.

References:
The general physical exam was unremarkable, except for bilateral expressible galactorrhea. Neurological exam was notable for diminished visual acuity (20/200) and a temporal upper quadratic hemianopsia in the left eye. Complete blood count and serum biochemistry were normal. Normal values were obtained for serum thyroxine, free thyroxine index, triiodothyronine, thyroid stimulating hormone, luteinizing hormone, follicle stimulating hormone and growth hormone. The 8 a.m. cortisol was low (160 nmol/L; normal 248-690); the 4 p.m. cortisol was normal (165 nmol/L; normal 83-414). Serum prolactin was markedly elevated (365 µg/L; normal < 25).

Dynamic pituitary function testing was performed via injection of thyrotropin releasing hormone (TRH) 200 µg i.v., C2I insulin 0.1 µ/kg i.v., and luteinizing hormone-releasing hormone 100 µg s.c. There was a blunted TSH response to TRH and LH response to LHRH. Prolactin, which was 363 µg/L at baseline, rose to a peak of 499 at 30 min after TRH. Hypoglycemia was not achieved, making the interpretation of the blunted cortisol and growth hormone response impossible.

Skull X-ray revealed enlargement of the sella turcica and chiasmatic groove (Figure 1). Non-contrast CT scanning of the cranium demonstrated a suprasellar mass eroding the posterior clinoid (Figure 2). The history of severe unilateral headaches beginning eighteen months prior to the onset of galactorrhea and the documentation of asymmetrical visual changes led to suspicion of a possible cause other than prolactinoma. Carotid angiography was performed and showed a 2 X 2 cm partially thrombosed aneurysm arising from the left internal carotid artery, above the anterior clinoid process (Figure 3). An MRI scan was not performed because of the perceived urgency of performing surgery before aneurysmal rupture.

At craniotomy a large aneurysm arising from the left paraclinoid internal carotid artery above the opthalmic artery was found. The aneurysm ballooned upward and posteriorly, compressing the optic nerve, optic chiasm, and hypothalamus. There was also a smaller aneurysm at the origin of the opthalmic artery which had not been appreciated on angiography. Both aneurysms were clipped without incident.

Post-operatively, the patient noted improved vision in the left eye (20/40). The post-operative angiogram showed two surgical clips at the level of the previous aneurysms and no evidence of residual aneurysm (Figure 4). Three months post-operatively the galactorrhea resolved, but the prolactin level remained elevated (77 µg/L). Eighteen months after the surgery, the prolactin level was still elevated (58 µg/L). The patient did not return for follow-up until four years after surgery, at which time the prolactin level was normal (19 µg/L), as were the TSH and prolactin responses to TRH infusion. A high resolution CT scan of the brain with coronal cuts of the sella done at this time showed no evidence of adenoma of the pituitary or lesions of the hypothalamus (Figure 5). An MRI scan was not performed because of the presence of surgical clips.
prolactinemia is likely via hypothalamic or pituitary stalk compression, resulting in interference with the delivery of prolactin-inhibiting factor to the pituitary. Two studies of patients with non-prolactin secreting sellar tumours with secondary hyperprolactinemia have, however, demonstrated a lack of correlation between suprasellar extension with compression of the pituitary stalk and degree of prolactin elevation. 6, 8 In our patient the extremely high level of prolactin suggests an additional stimulating effect by a putative hypothalamic prolactin-stimulating factor. It is not known whether the pulsatility of an aneurysm can stimulate the secretion of such a factor or stimulate the anterior pituitary itself. Studies in experimental animals have shown that irritative lesions of the anterior hypothalamus could potentially cause release of prolactin-stimulating factor. 9 The reason that the prolactin level in our patient normalized only years post-operatively is unclear, but implies reversible stimulation of prolactin-secreting cells.

In summary, our patient illustrates that carotid aneurysm is a cause of reversible pituitary dysfunction, and that a prolactin level greater than 300 μg/L should not be considered to be a safe cut-off in distinguishing pituitary tumour from other causes of elevated prolactin. Thus, even patients with this degree of hyperprolactinemia should be subjected to cerebral angiography or magnetic resonance imaging if there is any clinical suspicion of carotid aneurysm, since CT scanning is not reliable for this purpose and the surgical consequences of pituitary exploration in this setting are potentially grave.

REFERENCES