Acute Psychosis related Pituitary Haemorrhage

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Introduction
Pituitary apoplexy (PA) is a rare and potentially life threatening clinical syndrome as a result of haemorrhage or infarction in the pituitary1. It has been described mostly in association with a pituitary adenoma, more likely in macroadenomas, but only in isolated cases with a structurally normal pituitary gland. The pathophysiological mechanism underlying traumatic pituitary apoplexy has not been established and is rarely described2. Neuropsychiatric symptoms as a presentation of hypopituitarism is also very rare3.

Case History
44 year old retired male athlete. Fit and Well. Not on any medication.

Day 1
Sectioned under Mental Health Act. Transferred to Mental Health hospital.

Day 0

Day 0a
Lumbar puncture: Normal.
MRR: revealed high T1 signal from the right half of the moderately enlarged pituitary gland suggestive of a small bleed within the gland.

Diagnosis: Pituitary Apoplexy.

Follow up
1 Month post Discharge: SST baseline 228nmol/L and 30 mins 372nmol/L. Pituitary profile remained normal.

Discussion
PA most commonly is associated with a bleed into an adenoma, however pituitary apoplexy may also occur in non-adenomatous or even within a normal pituitary gland especially during pregnancy4. Pituitary adenomas are prone to bleed and undergo infarction and necrosis. This is because adenomas are predominantly supplied by a direct blood supply, rather than the portal system in the normal pituitary. In addition, prolactinomas contain arteries not found in a normal anterior pituitary. These are likely the reasons pituitary apoplexy is rarely reported in a normal pituitary. Diagnosis of PA is frequently missed, not only because of its low incidence, but also the presentation can be slowly progressive as in our case. This subtype presentation can be dependent upon the extent of bleeding as well as the degree of oedema and necrosis5.

In a recent literature review, only 13 post-traumatic pituitary apoplexy cases were identified from 1983 to 20166. The mechanism underlying post traumatic pituitary apoplexy has not been established. Possible hypothesis including traumatic shearing force causing the destruction of the pituitary stalk and lead to the consequent blockage of pituitary portal vasodilation or alteration of the venous vasculatization pattern following haemorrhage encouraging ischemic-haemorrhagic occurrences6.

In traumatic cases, PA symptoms usually become evident after few hours, with a mean delay of presentation of 14 days, whereas in some cases can even be up to 2 months. High temperatures in PA could be thought to be a sign of infection and may delay the diagnosis, but is in fact a typical finding in patients with Mll7.

Psychotic presentations following panhypopituitarism has been described, from various aetologies including an ectopic posterior pituitary, Russell’s Virus Bite, Sheehan’s syndrome, Traumatic Brain Injury (TBI), and after glucocorticoid therapy8-12. The pathogenesis of hypopituitarism is postulated to be a result of interactions between pituitary hormones and the dominant neuromodulators2. Krishnamurthy et al. (2013) in a post mortem study of schizophrenic subjects, molecular changes were identified in the pituitary gland with differentially expressed molecules including hypothalamic-pituitary-adrenal axis-associated constituents such as cortisol, pro-adrenocorticotropin hormone, arginine vasopressin precursor, agouti related protein, growth hormone, prolactin and secretagogen, as well as molecules associated with lipid transport and metabolism. They concluded that these hormones may present some diagnostic utility with further research in schizophrenia8.

Conclusion
MRI of the pituitary should be obtained at 3-6 month intervals until the anatomy is stable and then yearly for 5 years. A month post discharge from the hospital and recovery from the acute event, repeat endocrine testing to determine if the endocrine defects persist to confirm whether the patient needs to remain on life-long hormone replacement.

It is important to think of PA even after minimal head trauma especially in the setting of abnormal behaviour. The presentation may be subacute and MR imaging should be done even in the presence of normal CT.

References