

# SQUAMOUS CELL CARCINOMA OF THE SPHENOID SINUS PRESENTING AS A SELLAR MASS



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## INTRODUCTION

Although the most common cause of a sellar mass is a pituitary adenoma (either secreting or non-secreting)<sup>1</sup>, there are many other important causes. This case report discusses a patient who presented with ophthalmoplegia from a large sellar-suprasellar lesion, and was subsequently diagnosed with a squamous cell carcinoma of the sphenoid sinus with invasion into the pituitary gland causing panhypopituitarism.

## CASE REPORT

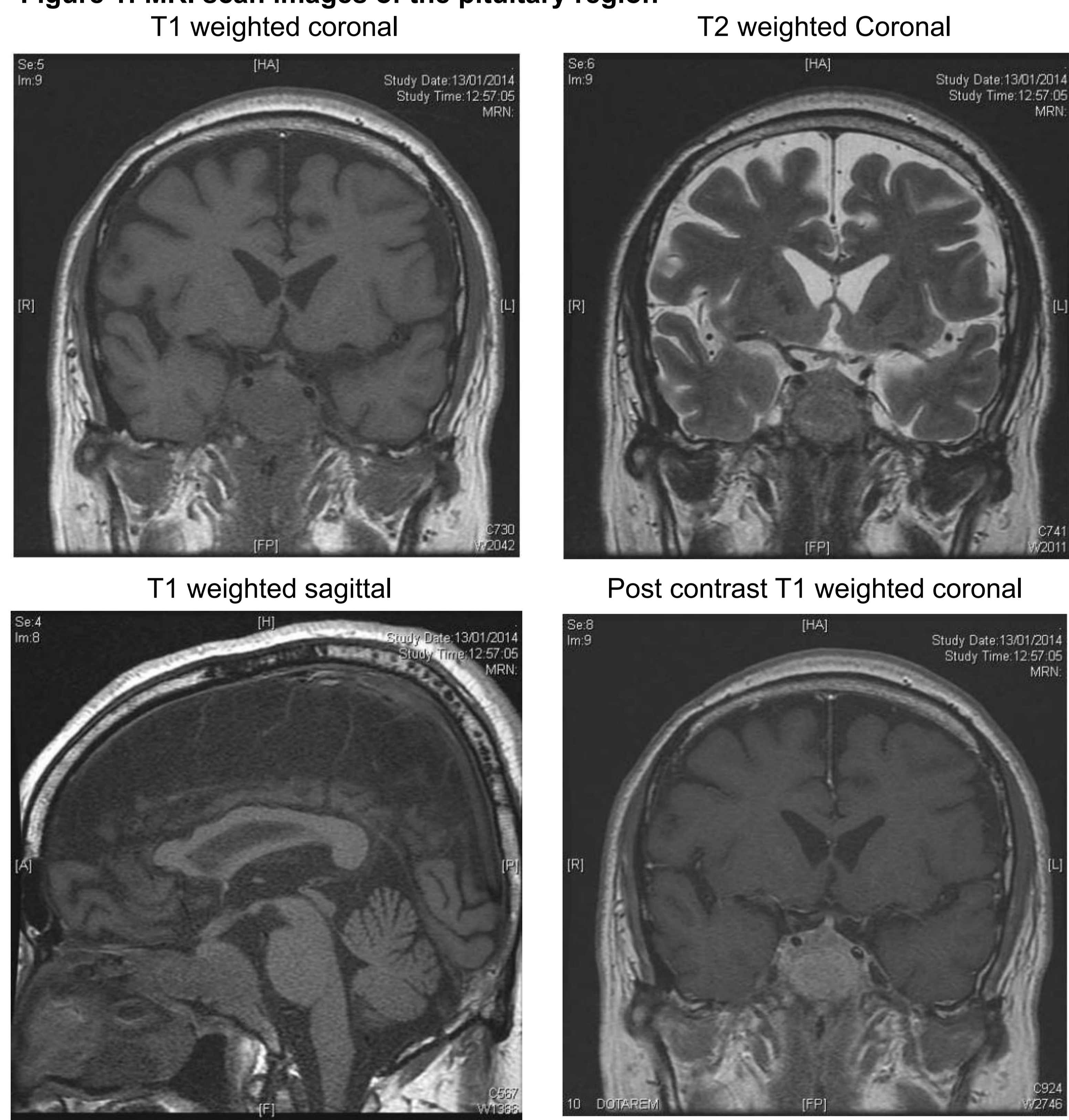
A 60 year-old Chinese male with schizophrenia and a 20 pack year smoking history, was admitted to Neurology for headache and diplopia without visual field loss. Brain imaging with Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) revealed a 2.9 cm sellar mass based in the sphenoid sinus, bulging into the suprasellar cistern with post contrast enhancement and pituitary stalk thickening. The optic chiasm was indented, with involvement of the cavernous sinuses and superior portion of the clivus.

Subsequent hormonal investigations revealed panhypopituitarism (central hypocortisolism, hypothyroidism and hypogonadism) with hyperprolactinaemia likely from pituitary stalk compression and anti-psychotic medication. There were however no symptoms of central diabetes insipidus (DI). Hormonal replacement with oral hydrocortisone and thyroxine was commenced.

An initial biopsy showed focal moderate to severe keratinising dysplasia but no definite invasive malignancy, and a repeat biopsy was planned. Repeat imaging done for worsening symptoms 2 weeks later showed an increase in the size of the sellar mass to 4 cm. Trans-sphenoidal debulking surgery was carried out, and intra-operative biopsy and frozen section revealed the diagnosis of squamous cell carcinoma. Staging CT scans did not reveal any lymph node or distant metastases. Post operatively, patient developed central DI with a biphasic pattern.

In view of the advanced intracranial involvement of the tumour, palliative chemotherapy and radiotherapy was planned but patient unfortunately developed progressive worsening neurological deficits likely from tumour progression and passed away.

Figure 1: MRI scan images of the pituitary region



### Short Synacthen Test (with IV synacthen 250 mcg)

Time	Cortisol (nmol/L)	ACTH (pmol/L)	Notes
0 min cortisol (2 pm)	44	2.1	
30 min cortisol	383		
60 min cortisol	471		Peak >500 nmol (normal)
ACTH (at 0 min)		2.1	

## CONCLUSION

Squamous cell carcinoma of the sphenoid sinus can rarely present as a sellar mass. Although pituitary adenomas are the most common cause of sellar masses, atypical features such as pituitary stalk thickening necessitate a high index of suspicion to look for other causes.

## DISCUSSION

Although pituitary adenomas account for 80 to 90% of sellar masses<sup>2</sup>, a number of atypical features made us suspicious that this was not a pituitary macroadenoma, including: cranial neuropathy without visual field loss, pituitary stalk thickening, and the epicentre of the mass being the sphenoid sinus rather than the sella. Differential diagnoses before biopsy included invasive infections, local (like in this case) or metastatic malignancies, parasellar masses (e.g. craniopharyngioma, meningioma), or infiltrative causes.

Before definitive surgery, our patient already had evidence of hormonal hypofunction in the adrenal, thyroid and gonadal axes. Hormonal hypofunction is relatively common in non-pituitary sellar masses. In a previously published case series, 75% of adults with infiltrating or malignant sellar lesions presented with at least one hormonal abnormality (hormonal deficiency or hyperprolactinaemia)<sup>3</sup>, with the most common pituitary dysfunction being central hypothyroidism. Interestingly, in a previous report of a case on sphenoid sinus squamous cell carcinoma presenting as a sellar mass<sup>4</sup>, no evidence of hormonal dysfunction was reported despite the sella being replaced entirely by the mass.

Laboratory Test	Result	Unit	Reference Interval
8 am cortisol	225	nmol/L	240 – 618
Thyroxine, Free	8	pmol/L	8 – 21
TSH	0.05	mIU/L	0.34 – 5.60
Testosterone	<1	nmol/L	5 – 30
LH	<1	IU/L	1-9
FSH	2	IU/L	1 – 19
Prolactin	569	mIU/L	77 – 274 (gender specific)
IGF-1	112	ug/L	81 – 225 (age specific)
Growth Hormone	0.33	ug/L	

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