

## Pituitary Metastasis: Central Diabetes Insipidus unmasked by Corticosteroids – Case Series and Review of Literature

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**Key words:** Pituitary metastasis, Central diabetes insipidus, Pan-hypopituitarism, Corticosteroids

### Background

Metastasis to the pituitary gland is rare encounter and is more common amongst the elderly population with advanced malignancy. An estimated 1% of all pituitary tumour resections are metastatic. Primary sites that frequently metastasize include breast and lung carcinomas. In the recent decade, advancement in the field of oncology with multiple modalities of therapy has led to prolonged survival of patients with advanced stages of malignancy. Herein, we present three cases and review of literature of pituitary metastasis presenting as central diabetes insipidus (CDI) incidentally unmasked following administration of corticosteroids.

### Objective

To establish the common clinical features, establish variations in clinical presentations and natural progression of disease in patients with pituitary metastasis.

### Methods

Three cases of central diabetes insipidus unmasked by corticosteroids in pituitary metastasis were presented. A total of 9 other cases with central diabetes insipidus as first clinical manifestation unmasked by corticosteroid published from 2007-2018 were reviewed. Pertinent references were searched using windows remote search model on PubMed. The key words “pituitary metastasis” and “diabetes insipidus” searched in all fields resulted in 161 articles, of which articles of cranial diabetes insipidus as initial presentation without the use of corticosteroids were excluded. Searching “central diabetes unmasked by steroids” retrieved 8 additional references. Searching “metastatic carcinoma of pituitary” retrieved 35

additional references. About 20 new references were identified from the bibliographies of the articles reviewed. Ultimately, we identified a total of 18 references relevant to this research from the search terms. All references were reviewed to retrieve relevant references for this study. Non-English articles were excluded.

### Results

A compilation of 9 previously reported cases of central DI unmasked by corticosteroids from 2007 to 2017 along with the present 3 cases were performed (Table 1). There was equal gender prevalence with a mean age of 61 (range 56-80 years old). More than 75% of the cases described here had previously been diagnosed with advanced malignancies of varying primary sites. The remaining 25% presented with varying symptoms of hypopituitarism as the harbinger to the discovery of the primary neoplasm. Amongst the literature review and cases presented, primary malignancies with pituitary metastasis included lung adenocarcinoma (33%), breast carcinoma (25%), nasopharyngeal carcinoma (16%), renal cell carcinoma (8%), hepatocellular carcinoma (8%) and gastric adenocarcinoma (8%). It is noteworthy that two of three present cases identified, were the result of directly infiltration of nasopharyngeal carcinoma to the pituitary gland. There is limited data documenting the prevalence of nasopharyngeal carcinoma with pituitary metastasis within the Asian population.

### Conclusion

Central diabetes insipidus unmasked by corticosteroids is a less recognized, potentially lethal but fully reversible complication of pituitary metastasis. Symptoms or signs of central diabetes insipidus should be sought in all

patient with advanced malignancies presenting with polyuria and hypernatremia. Prompt restoration of pituitary hormones is warranted in affected patients to

allow timely restoration of hormonal balance and preventing endocrine emergencies.

## Introduction

Metastasis to the pituitary gland is a rare encounter representing less than 1% of all pituitary lesions. The first reported case of metastasis to the pituitary gland was identified and reported (in German) by Benjamin L. in 1857 discovered during an autopsy of a patient with disseminated melanoma<sup>(1, 5, 17)</sup>. The increased prevalence in detection of pituitary metastasis denotes advancement in oncology treatment and options. These pituitary metastasis in advanced malignancies are most typically identified in the elderly population with diffuse malignancy. The most common primary tumours with metastasis to the pituitary gland are breast, lung and gastrointestinal malignancies. Their scarcity and usually indolent course, as well as the lack of specific clinical and radiological features, impede their differentiation from other more common sellar area lesions, particularly when history of malignancy is absent. Advancement in the field of oncology within the last decade has progressed with enhanced imaging modalities, improved surgical techniques, radical radio- and chemo-therapeutics for the treatment for systemic malignancies. This has led to augmented diagnosis of pituitary metastasis from primary tumours. Despite that, pituitary metastasis remains a challenge for diagnosis and remains poorly recognised and under reported. These pituitary metastasis are often discovered incidentally presenting with central diabetes insipidus having become unmasked after initiation of corticosteroids.

We report 3 clinical cases of pituitary metastasis diagnosed after incidental presentation of central diabetes insipidus after corticosteroid administration. A review of 9 other reported cases from 2007-2017 along with the current three cases were reviewed to establish common clinical features and clinical course of pituitary metastasis.

### Case 1

Madam L, 67 year old lady diagnosed three years ago with stage four lung adenocarcinoma with extensive metastases to liver and bone presented to our centre with history of poor oral intake, generalised lethargy and reduced urine output for the past three days. She was admitted and treated as community acquired pneumonia and lymphangitis carcinomatosa where she received a week's course of intravenous ceftriaxone and 4mg dexamethasone daily. Initial sodium and potassium on admission was 145mmol/L and 4.3mmol/L. However, five days later she developed confusion and polyuria with urine-output up to 4L/day. Initial computed tomography (CT) of the brain showed thickened enhancing pituitary stalk and posterior lobe of the pituitary gland. Magnetic

resonance imaging (MRI) showed a lobulated lesion in the right side of the posterior pituitary measuring 0.7 x 1.0 x 0.4cm and a well-defined lesion measuring 0.7 x 0.8 x 0.6cm at the superior aspect of the pituitary infundibulum. Biochemical markers supported the diagnosis of central diabetes insipidus with serum Na 156mmol/L, serum Osmolarity 309mmol/L and urine Osmolarity 145mmol/L. She was treated with subcutaneous desmopressin and intravenous fluids. Further tests demonstrated panhypopituitarism and oral desmopressin, thyroxine and hydrocortisone was initiated. Patient opted for palliative care succumbing to 3 months after admission.

### Case 2

Madam S, 56 year old lady diagnosed with stage four nasopharyngeal carcinoma presented to us with symptomatic hyponatremia in March 2017 having undergone combined chemo-radiotherapy the previous year. She was dehydrated with sodium of 115 mmol/L and potassium 3.9mmol/L. She responded to hydration as sodium increased to 128 mmol/L. However, a week later sodium levels dropped to 119mmol/L despite hydration. CT brain revealed increasing size of primary tumour with intracranial extension involving cavernous sinus, pituitary fossa and left temporal and pontine infiltration. Other investigations support the diagnosis of syndrome of inappropriate antidiuretic hormone secretion and she responded to fluid restriction. In addition, she was diagnosed with hypocortisolism (9am: 26nmol/L) and commenced on oral hydrocortisone 10mg twice daily replacement. Repeat CT staging on revealed disease progression and a pituitary mass of 1.7 x 1.4 x 1.7cm. She was readmitted for chemotherapy but discharged without hydrocortisone in June 2017. In July 2017, she was admitted for her third cycle of chemotherapy with sodium of 123mmol/L, potassium 3.4mmol/L and was restarted on oral hydrocortisone 25mg tds. Unfortunately, she developed thirst and polyuria the same day with increasing sodium trend. Laboratory investigations revealed low urine osmolarity and high serum osmolarity with increasing serum sodium levels supported the diagnosis of central diabetes insipidus and subcutaneous desmopressin was administered. Complete pituitary hormonal panels support pan-hypopituitarism. She was discharged with hydrocortisone, thyroxine replacement and oral desmopressin but readmitted in September 2017 as her condition deteriorated. Ct brain showed disease progression with enlarging pituitary metastasis. Patient opted for palliative care and succumbed within 1 month.

### Case 3

Mr C, a 55 year gentleman was diagnosed with advanced nasopharyngeal carcinoma, undifferentiated type, NOS, TNM: T4 N1 M0, Stage: IVA. He had completed combined chemo-radiotherapy in December 2017. Reassessment CT done in June 2018 revealed residual tumour at left inferior orbital fissure, left optic canal and left pterygopalatine fossa with local infiltration into the left cavernous sinus, pituitary sella and right sphenoid sinus. He was subsequently planned for chemotherapy with Paclitaxel/Carboplatin by the oncologist. He was admitted with symptoms of feeling unwell, vomiting, hypotension and hypoglycaemia. Morning serum cortisol and thyroid function revealed hypopituitarism. He was initiated on oral hydrocortisone and thyroxine replacement and was discharge home. He presented 4 weeks later via emergency with symptoms of polyuria, with inability to compensate and with documented urine output of more than 200mls per hour. Investigations and water deprivation test confirmed the diagnosis of central diabetes insipidus with serum osmolality of 291, serum sodium of 149 and urine osmolality 164mmol/L. Further history revealed that the onset of polyuria was soon after initiation of steroids however patient was able to compensate with large volumes of fluid intake, up to 4L/day. He was discharged home with oral desmopressin 0.1mg daily in combination with other pituitary hormone replacement. He is planned for palliative chemotherapy in view of inoperable advance malignancy.

## Discussion

The pituitary gland is an uncommon location for metastatic disease, although neoplasms from almost every tissue have been reported to metastasise there. In approximately two third cases of advanced malignancies, the patients were known to have metastatic disease prior to the discovery of pituitary metastasis. On the contrary, a third of patients, pituitary symptoms were the harbinger to the discovery of the primary neoplasm. The most frequent sources of metastases are: breast carcinoma (53% of pituitary metastatic lesions) and lung carcinoma (19%)<sup>(1, 5, 17)</sup>. Metastatic spread is more common to the pituitary posterior lobe. A review of 201 cases of pituitary metastases demonstrated that the posterior lobe was involved in 84.6% (n = 170), with isolated posterior and anterior lobe lesions seen in 50.8 and 15.4% of cases respectively<sup>[2, 3, 5]</sup>. Many explanations for this predilection have been proposed. The posterior lobe is perfused directly by the inferior hypophyseal arteries, while the anterior lobe is supplied by a portal system around the infundibulum from the superior hypophyseal arteries, thus direct haematogenous spread may be more likely to seed to the capillaries of the stalk and posterior lobe. A further contributing factor is the fact the posterior lobe has a larger contact area with adjacent dura, facilitating meningeal spread though the suprasellar cistern<sup>[5]</sup>.

Central diabetes insipidus (DI) is relatively common in pituitary metastasis, present in 42.3% (95% CI 36.2–48.5) of patients at presentation in one pooled study (n = 248)<sup>[5]</sup>. Moreover, in a patient with known metastatic disease,

the development of DI and radiographic evidence of a pituitary mass is strongly suggestive of a pituitary metastasis. DI in the setting of metastasis may be associated with a thickened pituitary stalk in combination with absence of the normal high T1 signal intensity in the posterior lobe<sup>(27)</sup>. The high incidence of DI in metastatic lesions is consistent with the similarly high incidence of posterior lobe involvement.

Our study described three cases of occult ADH deficiency masked by concurrent ACTH deficiency; only once glucocorticoid replacement therapy had been administered did the symptoms diabetes insipidus appear. This ‘masking’ phenomenon could be due to a multitude of factors, both from ADH-dependent and ADH-independent mechanisms, resulting in impaired renal-free water clearance. Recognition of this phenomenon in patients with adrenal deficiency and risk factors for developing CDI is important in early diagnosis and management of this phenomenon<sup>[6]</sup>.

The reasons for this are complex. Firstly, cortisol induces resistance of the V2 receptor (or at a post-receptor level) to ADH, thus in states of glucocorticoid deficiency, the effects of ADH are amplified<sup>[7]</sup>. Secondly, Corticotrophin Releasing Hormone (CRH) stimulates ACTH and ADH release, thus glucocorticoid deficiency upregulates CRH and thus ADH release<sup>[7,8]</sup>. Lastly, hypocortisolaemia results in renal sodium loss and volume depletion, potent stimulators for increased (but “appropriate”) ADH release. As such, when glucocorticoid deficiency is ameliorated, these compensatory mechanisms fail, and DI ensues. The high rate of DI in our study relative to the literature may be partly explained by our assessment of ADH function both before and after glucocorticoid replacement. Half of our cases had DI on initial assessment, similar to the prevalence in other studies, however the prevalence in our cohort increased to 75% after correction of hypocortisolaemia. Thus assessment of ADH function both before and after glucocorticoid replacement appears to increase the sensitivity for diagnosis of DI in the setting of pituitary metastasis. Overall life expectancy in patients with sellar metastases is 6–22 months.

A compilation of 9 previously reported cases of central DI unmasked by corticosteroids from 2007 to 2017 along with the present 3 cases were performed (Table 1). There was equal gender prevalence with a mean age of 61 (range 56-80 years old). More than 75% of the cases described here had previously been diagnosed with advanced malignancies of varying primary sites. The remaining 25% presented with varying symptoms of hypopituitarism as the harbinger to the discovery of the primary neoplasm. Amongst the literature review and cases presented, primary malignancies with pituitary metastasis included lung adenocarcinoma (33%), breast carcinoma (25%), nasopharyngeal carcinoma (16%), renal cell carcinoma (8%), hepatocellular carcinoma (8%) and gastric adenocarcinoma (8%).

Previous studies have reported a high prevalence breast carcinoma and lung carcinoma, however the Asian population shows a significant predilection for pituitary metastasis of nasopharyngeal carcinoma. The prevalence of NPC combined with the progression of disease with direct infiltration of the pituitary gland accounts for the presentation. However, there are no comparable studies looking into the frequency of nasopharyngeal carcinoma with pituitary metastasis.

The common presenting features in the present case series included significant polyuria, polydipsia with some patients presenting with an acute confusional state with the inability to compensate after the initiation of corticosteroids for pan-hypopituitarism. Symptoms of central diabetes insipidus was masked by the relative ADH deficiency. Most cases of pituitary metastasis presenting with central diabetes insipidus as the primary clinical presentation have led to an early diagnosis of pituitary metastasis. The perplexity arises when symptoms are masked and when patients present with symptoms of hyponatremia. Often, these preliminary signs to the presence of pituitary metastasis are often overlooked as these pathognomic symptoms are rare.

Advancement in imaging modalities with interest in neuroimaging has led to precision diagnosis pituitary metastasis. Amongst the literature review and cases presented, initial imaging by CT scan required more comprehensive images requiring MRI. The clinical outcome limited by late presentation in combination with advanced systemic disease should not be a limitation for precision imaging. Oncology offers targeted stereotactic radiosurgery as an effective palliative approach for most patients with pituitary metastasis.<sup>(18)</sup>

## Conclusion

Central diabetes insipidus unmasked by corticosteroids is a less recognized, potentially lethal but fully reversible complication of pituitary metastasis. Symptoms or signs of central diabetes insipidus should be sought in all patient with advanced malignancies presenting with polyuria and hypernatremia. Prompt restoration of pituitary hormones is warranted in affected patients to allow timely restoration of hormonal balance and preventing endocrine emergencies.

**Table 1. Brief summary of cases discussed from clinical patients and literature review identifying clinical presentations, investigations, diagnosis and clinical outcome.**

No	age	Sex	Diagnosis	Serum osmo mmol/L	Urine osmo mmol/L	Na mmol/L	Clinical outcome	Imaging	Notes
CR1	67	F	Diagnosis : lung adenocarcinoma with extensive metastases to liver and bone  Clinical presentation : developed confusion and polyuria 5 days after initiation of dexamethasone	309	115	156	Opted for palliative care succumbing 3 months after admission	MRI showed a lobulated lesion in the right side of the posterior pituitary measuring 0.7 x 1.0 x 0.4cm and a well-defined lesion measuring 0.7 x 0.8 x 0.6cm at the superior aspect of the pituitary infundibulum.	
CR 2	56	F	Diagnosis : nasopharyngeal carcinoma stage Iv with pituitary metastasis  Clinical presentation : Started on Hydrocortisone of pan-hypopituitarism and developed polyuria				Patient opted for palliative care and succumbed within 1 month.	CT brain revealed increasing size of primary tumour with intracranial extension involving cavernous sinus, pituitary fossa and left temporal and pontine infiltration.	
CR 3	55	M	Diagnosis : nasopharyngeal carcinoma stage Iv with pituitary metastasis  Clinical presentation : Started on Hydrocortisone and thyroxine for symptoms of pan-hypopituitarism. Developed DI, however was compensated with increase fluid intake. Presented 3 weeks after initiation of corticosteroids with inability to compensate orally	294	161	148	For palliative chemotherapy with paclitaxel/Carboplatin	CECT Residual tumour at left inferior orbital fissure, left optic canal with local infiltration into the cavernous sinus, pituitary sella and right sphenoid sinus	

Polyuric > 150cc urine output per hour																																				
1	71	F	<p>Diagnosis: Primary lung malignancy with cerebral and pituitary metastasis diagnosed partial CDI initially obscured by concomitant central hypocortisolism and possible bronchogenic carcinoma-associated SIADH, only becoming overt after steroid replacement.</p> <p>Clinical Presentation: lethargy, poor oral intake, functional decline and progressive confusion on a background of visual blurring for a number of months.</p> <p>Low t4 8 pmol/L 8–21  TSH 1.21mIU/L 0.34–5/60  Am Cortisol 147 nmol/L 240–618  LH &lt; 1IU/L 11–59  FSH 2 IU/L 17–11</p> <p>Urine output &gt; 2.7L /day</p>	355	392	163	<p>No biopsy due to advanced malignancy – palliative care with hypopit replacement</p> <p>transferred to an inpatient hospice on nasal DDAVP and oral dexamethasone</p>	<p>MRI-well defined suprasellar mass measuring 26 mm x 26 mm x 19 mm, and multiple intracerebral hypodensities with perilesional oedema consistent with metastases</p>	<p>Indication for steroid- 1. For allergies cover for CT scan  2. dexa for perilesional odema</p> <p>March 2015</p>																											
2	80	F	<p>hepatocellular carcinoma with pituitary metastasis</p> <table border="1"> <tr> <td>GH</td> <td>0.360 ng/mL</td> <td>0.010–3.607 ng/mL</td> </tr> <tr> <td>PRL</td> <td>6.83 ng/mL</td> <td>&lt;12.3 ng/mL</td> </tr> <tr> <td>TSH</td> <td>1.288 μIU/mL</td> <td>0.350–4.940 μIU/mL</td> </tr> <tr> <td>FT3</td> <td>1.78 pg/mL</td> <td>1.71–3.71 pg/mL</td> </tr> <tr> <td>FT4</td> <td>&lt;0.40 ng/dL</td> <td>0.70–1.48 ng/dL</td> </tr> <tr> <td>ACTH</td> <td>1.4 pg/mL</td> <td>7.2–63.3 pg/mL</td> </tr> <tr> <td>Cortisol</td> <td>3.1 μg/dL</td> <td>4.0–19.3 μg/dL</td> </tr> <tr> <td>Aldosterone</td> <td>10 pg/mL</td> <td>36–240 pg/mL</td> </tr> <tr> <td>Plasma renin activity</td> <td>0.5 ng/mL</td> <td>0.2–3.9 ng/mL</td> </tr> </table>	GH	0.360 ng/mL	0.010–3.607 ng/mL	PRL	6.83 ng/mL	<12.3 ng/mL	TSH	1.288 μIU/mL	0.350–4.940 μIU/mL	FT3	1.78 pg/mL	1.71–3.71 pg/mL	FT4	<0.40 ng/dL	0.70–1.48 ng/dL	ACTH	1.4 pg/mL	7.2–63.3 pg/mL	Cortisol	3.1 μg/dL	4.0–19.3 μg/dL	Aldosterone	10 pg/mL	36–240 pg/mL	Plasma renin activity	0.5 ng/mL	0.2–3.9 ng/mL	-UA-	-UA-	-UA-	<p>January 2012, the patient died of hypovolemic shock.</p>	<p>MRI of the brain revealed a tumor measuring 13 mm × 13 mm in the sella turcica, which had spread across the suprasellar region</p>	<p>Postmortem examination of the pituitary tumor revealed tumoral hepatocytes in a thick trabecular pattern, the typical appearance of well differentiated HCC</p> <p>2015</p>
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			ADH	0.27 pg/mL	0.3–4.2 pg/mL				
			LH	<0.10 mIU/mL	7.5–56.2 mIU/mL				
			FSH	0.27 mIU/mL	9.2–124.7 mIU/m				
			Clinical presentation :sudden-onset anorexia accompanied by hypotension and bradycardia. Ix revealed panhypo pit. Diabetes insipidus (DI) developed five days into the replacement therapy						
3 *	69	M	Clinical presentation: with vomiting, low blood pressure and hypoglycaemia. Ix revealed panhypo pit. Diabetes insipidus (DI) developed five days into the replacement therapy	-UA-	-UA-	-UA-	Bronchoscopy and biopsy demonstrated a pulmonary adenocarcinoma. Hence we concluded to a lung cancer with multiple pituitary and adrenal gland metastases.	MRI demonstrated an inhomogeneous pituitary hypertrophy, with convexity of the sellar diaphragm, a nodular thickening of the pituitary stalk, and a loss of high intensity signal from the posterior pituitary	2016
			Serum cortisol 12.6 (100-250 ng/mL) Free T4 6.8 pg/mL (8-18 pg/mL) TSH 0.005mUI/mL ( 0.5-4 IU/mL), Testosterone 0.025 (2.5-10 ng/mL), FSH 0.5 IU/mL ( 1-8.4 IU/mL), LH 0.1IU/L (10.5 IU/L) Low urine osmolarity.						
4 *	77	M	Clinical presentation: 1-day history of confusion, insomnia and reduced appetite. No focal neurological deficit, visual field defect or ophthalmoplegia. laboratory testing showed hypopituitarism-started replacement	308	-UA-	148	palliative care initiated-patient succumbed shortly after diagnosis	(CT) brain scan was performed, revealing an enhancing (1.5 × 1.7 cm) suprasellar mass with also oedema of the overlying optic tract	Complicated by cranial DI and SIADH –
			Free thyroxine	4.8 pmol/l					
			TSH	0.99 miu/1					
			LH	<0.5					
			FSH	<0.5					
			Prolactin	95 miu/1					
			Testosterone	<0.3					
			Cortisol	44 nmol/1					

			SHBG	32 nmol/l					
			Five days after discharge, the patient re-presented to hospital with cranial DI Urine output > 4L /day primary lung cancer with metastasis to the pituitary was made, complicated by cranial DI						Jan 2016
5	82	M	Clinical presentation : sudden headache. History of distal radical gastrectomy for stage IIa adenocarcinoma- presenting with panhypopit.	-UA-	260	155	Transsphenoidal tumor excision was performed and intraoperatively, the lesion had a fibrous pseudo-capsule with a soft core of avascular necrotic tissue. Refused investigations for possible gastric carcinoma recurrence and adjuvant oncologic therapy. He succumbed three weeks after surgery.	MRI showed a single 1.2 cm isointense pituitary lesion with loss of the usual posterior lobe hyperintensity on T1-weighted sequence. Contrast imaging revealed a heterogeneously enhancing pituitary tumor suggestive of intralesional hemorrhage with a thickened stalk of 3 mm	
			Diagnosis : Advanced metastatic adenocarcinoma  serum cortisol levels (38 nmol/l). Developed DI after hydrocortisone – with Urine output > 1.2L /4 hour						
6	56	M	Clinical Presentation: fatigue, 50-pound weight loss, anorexia, constipation and nonspecific abdominal pain for 4 months. Pituitary function evaluation revealed panhypopituitarism.  Diagnosis: RCC of a horseshoe kidney with symptomatic isolated pituitary gland metastasis  Hormone 1-year prior Presentation Normal range TSH (mIU/L) 1.72 0.07 L 0.4-4.5 Free T4 (ng/dl) 0.6 L 0.8-1.8 FSH (mIU/ml) 1.5 L 1.6-8 LH (mIU/ml) 0.2 L 1.5-9.3 AM cortisol (µg/dl) 3.3 L 4-22 Prolactin (ng/ml) 78.8 H 2.0-18 Testosterone (ng/dl) 290 (1 pm) Not done 250-1,100  Treatment with steroids unmasked central di	-UA-	-UA-	-UA-	Right heminephrectomy confirmed RCC. Soon after he complained of progressively worsening headache and visual disturbance. Histopathology from urgent trans-sphenoidal hypophysectomy revealed RCC. The patient began post-surgical radiotherapy, but eventually he declined further treatments. In the end, he was placed on hospice where he passed away.	CT head showed 2.6 cm × 1.8 cm × 2.5 cm sellar mass likely with bilateral cavernous sinus extension.  (MRI) was contraindicated due to retained bullet fragments in his left upper chest.  Repeat CT head found that the sellar mass was 3.6cm × 2.2cm × 2.5cm and included a 1.0 cm suprasellar extension that abutted the optic chiasm	May 2015

									Nov 2017
7	66	F	<p>Clinical Presentation : Hypotensive with sepsis secondary to influenza A/H1N1-pneumonia requiring high ionotropic support. Initiated on corticosteroids in ICU. Developed central DI 24 hours after steroid initiation</p> <p>Diagnosis: Recurrent breast cancer with diffuse bone and mediastinal metastases.</p> <p>Further exploration of pituitary axis testing confirmed panhypopituitarism with a low FSH level in a postmenopausal patient and low serum levels of TSH, free T4, growth hormone, and IGF-1. Also, posterior lobe pituitary insufficiency was confirmed with levels of vasopressin and copeptin</p>	320	148	153	No documentation of clinical outcome	Pituitary MRI showed signal increase suggesting hemorrhage in sagittal T1-weighted and coronal T2-weighted MRI, but coronal T1-weighted MRI with contrast showed inhomogeneous enhancement of pituitary with metastasis.	July 2017
8	56	F	<p>Clinical Presentation : one month of weight loss and decreased appetite. She had no headaches, increased thirst or visual symptoms.</p> <p>Diagnosis : Occult breast malignancy with extensive metastases.</p> <p>Her biochemistries showed panhypopituitarism: ACTH 5.3 (010.2 pmol/L), cortisol 79 nmol/L, Free Thyroxine 6 (8-21 pmol/L), Thyroid Stimulating Hormone 0.81 (0.34-5.60 mIU/L), Insulin-like GF 1 42 (81-225 ug/L), Prolactin 274 (73-478 mIU/L), FSH 3 (17-114 IU/L), Luteinizing Hormone &lt; 1 (11-59 IU/L)</p>	312	133	144	<p>Urine Na :37</p> <p>She was commenced on chemotherapy and brain radiation therapy. Unfortunately, four weeks later, she passed away from cecal perforation and peritonitis.</p>	<p>CT of the body and brain showed a mass in the cecum, suprasellar, and extensive intramuscular, bone, lung, lymph nodes and cerebellar deposits.</p> <p>MRI of the pituitary fossa showed a 1.3 x 0.9 cm suprasellar mass inseparable from the pituitary stalk. The pituitary gland was normal</p>	
9	64	M	<p>Clinical presentation: loss of consciousness and gait disturbance. His serum sodium level was 117mEq/L. MRI revealed pituitary metastasis with anterior hormone deficiencies. Patient was initiated on steroids and polyuria ensued.</p>	-UA-	-UA-	-UA-	<p>Patient underwent subtotal resection of the tumor via a transsphenoidal approach. HPE revealed metastasis</p>	<p>Magnetic resonance imaging revealed a suprasellar tumor that showed inhomogeneous enhancement and was</p>	Jan 2016

Diagnosis : Pituitary metastasis in a patient with male breast cancer that resulted in pituitary dysfunction

from estrogen receptor-positive breast cancer.

attached to the optic chiasm.

The patient underwent conventional post-operative radiotherapy combined with hormone replacement therapy and has remained free of symptoms for 16 months.

July 2014

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