

# Intrasellar plasmacytoma presenting as a non-functional invasive pituitary macro-adenoma: Case Report & Literature Review

B. P. Sinnott · B. Hatipoglu · D. H. Sarne

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**Abstract** We report an uncommon case of an intrasellar plasmacytoma presenting as a non-functional invasive pituitary macro-adenoma as the first manifestation of multiple myeloma.

A 57 year old woman was referred to our department with a presumed diagnosis of a non-functioning pituitary macro-adenoma. She reported a 3 month history of headaches and a 2 week history of sudden onset of right facial numbness. Preoperative endocrine evaluation was remarkable only for a modestly elevated serum prolactin. A magnetic resonance imaging (MRI) scan revealed  $3.6 \times 5 \times 4$  cm enhancing homogeneous intrasellar mass with extension into the sphenoid and cavernous sinuses bilaterally; the optic chiasm was not displaced. She underwent transphenoidal surgery of the sellar lesion. The surgical specimen was heavily infiltrated with abnormal plasma cells, which stained almost exclusively for Kappa light chain immunoglobulins. An extensive investigation was undertaken to look for occult myelomatous disease. A diagnosis of multiple myeloma was made 1 month later based on a combination of clinical, pathological and radiologic features. She underwent radiation therapy directed towards the residual sellar tumor, followed by chemotherapy and autologous stem cell transplantation.

Review of the world literature revealed only 22 previous reports of patients in whom a solitary plasmacytoma or multiple myeloma first presented as a sellar mass; in all cases mimicking clinically and radiologically a non-functioning invasive pituitary adenoma however with additional cranial nerve involvement.

Intrasellar plasma cell tumors are rare tumors which may mimic non-functioning invasive pituitary tumors. The diagnosis should be suspected in patients with well preserved anterior pituitary function and cranial nerve neuropathies in the presence of significant sellar destruction.

**Keywords** Pituitary adenoma · Plasmacytoma · Multiple myeloma

## Introduction

Pituitary adenomas are the most common etiology of sellar masses [1]. Intra-sellar plasmacytomas are rare causes of sellar tumors of non-pituitary origin and may mimic non-functional pituitary adenomas clinically and radiologically.

We describe a case of a 57 year old female in whom an intra-sellar plasmacytoma mimicked clinically and radiologically a non-functioning invasive pituitary adenoma. Review of the world literature revealed only 22 previous cases of myelomatous disease presenting for the first time as non-functioning invasive pituitary adenomas.

## Case presentation

A 57 year old postmenopausal Caucasian female was referred to our department with a diagnosis of a non-functional pituitary macro-adenoma. She had been in her usual state of health until 3 months prior to admission, when she had noted the onset of diffuse intermittent headaches. Three weeks prior to presentation she reported the progressive onset of right sided facial numbness. Her medical history was positive for well controlled diabetes treated with glimepiride. She was married with 8 grown children. Her family history was negative for endocrinopathies or malignancy. She denied symptoms of polydipsia, polyuria, fatigue, bone pain,

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B. P. Sinnott (✉) · B. Hatipoglu · D. H. Sarne  
University of Illinois at Chicago,  
1819 West Polk Street,  
M/C 640, Chicago, IL, 60612, USA  
e-mail: bridgetsinnott@yahoo.com

**Table 1** Laboratory findings on presentation and post-operatively

LABS	On presentation	Post-operative	Reference range
TSH	1.16	0.68	0.4–4.0 mIU/ml
TT4	7.2	7.8	4.5–12.0 ug/dL
TT3	1.11	–	0.7–1.4 ng/ml
Cortisol	12.7 (5PM)	14.1 (8AM)	5–25 ug/dL
FSH	53.6	36.3	9.7–111 mIU/ml
LH	12.6	9.3	>14 mIU/ml
Prolactin	26.7	–	3–20 ng/ml
IGF-1	–	222	71–290 ng/ml
Total Protein	5.5	8.1	6–8 g/dL
Albumin	2.6	4.2	3.4–5 g/dL
Calcium	9.8	10	8.6–10.6 mg/dL
Electrolytes	Normal	Normal	
Creatinine	0.7	0.7	0.4–1.2 mg/dL
Hb/Plts	13.9/265	13.9/310	12–16 g/dL/150–400 thous/uL

back pain, nasal discharge, galactorrhea, visual changes or symptoms of infectious diseases.

Her physical examination was remarkable for a sensory deficit in the right V<sub>1</sub> cranial nerve distribution; other cranial nerves were intact. Visual fields were intact to confrontation. Peripheral nervous system examination was normal. Visual field testing by Goldman dynamic perimetry yielded normal results. There was no evidence of lymphadenopathy, organomegaly or purpura. No clinical evidence of malignancy or endocrinopathies was apparent.

Hormonal evaluation prior to surgical intervention revealed a minimally elevated prolactin level, suggestive for stalk compression. GH status was not assessed pre-operatively. Other pre-operative laboratory work-up was nor-

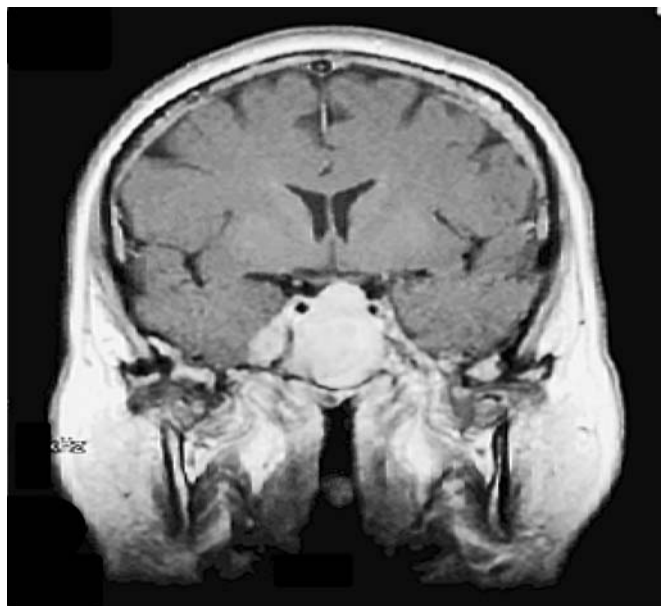
mal apart from an inappropriately normal LH level for a postmenopausal woman (Table 1). She did not have clinical or biochemical evidence of diabetes insipidus (DI).

A computerized tomography scan of the head revealed a multilobulated enhancing, infiltrating intrasellar mass extending into the sphenoid sinus – 3.3 × 4 × 3.5 cm. The tumor eroded the medial aspect of the pterygoid process, clivus and right petrous apex. There was also evidence of extension into the nasopharynx and posterior nasal fossa. A magnetic resonance imaging (MRI) scan revealed 3.6 × 5 × 4 cm enhancing homogeneous intrasellar mass with extension into the sphenoid and cavernous sinuses bilaterally, more so on the right to encase the internal carotid arteries; the optic chiasm was not displaced (Fig. 1). The radiologic findings supported the pre-operative diagnosis of a non-functioning invasive pituitary macroadenoma with stalk compression, however the possibility of other etiologies could not be excluded.

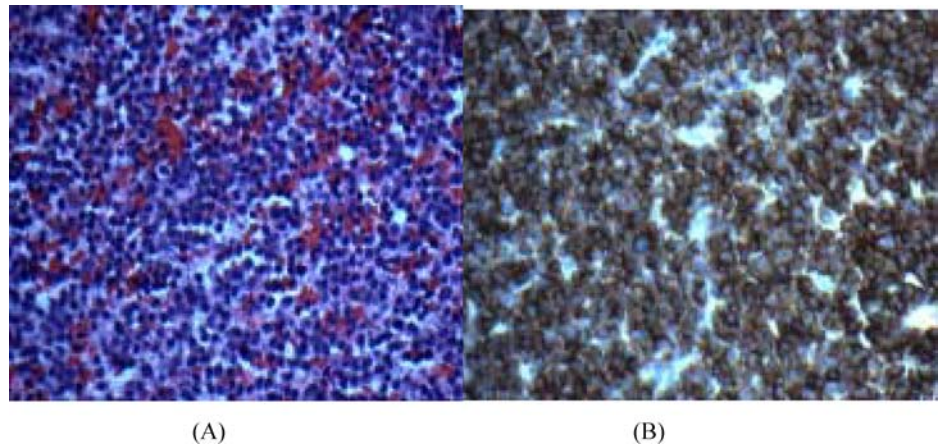
Transphenoidal surgery of the tumor was performed. The excised specimen consisted of multiple irregular red-brown soft tissue fragments, measuring in aggregate 0.5 × 0.5 × 0.5 cm. Post operative course was remarkable for the development of diabetes insipidus. She was discharged from the hospital on the seventh post operative day, on DDAVP, dexamethasone 4 mg q 8 h followed by a taper and glyburide.

The histological appearance of the specimen was that of a highly cellular neoplasm composed of mature plasma cells with round eccentrically placed nuclei. Immunohistochemical staining was performed to ascertain that the neoplastic cells consisted of a single clone of B cells. Tumor cells showed widespread immunoreactivity for CD 138 and CD 43. The cells stained negatively for chromogranin, synaptophysin, PLAP, EMA, CEA, CAM 52, prolactin, GH, ACTH,

**Fig. 1** Coronal MRI scan: reveals the presence of 3.6 × 5 × 4 cm multilobulated, homogeneously enhancing intrasellar mass with extension into the sphenoid sinus and cavernous sinuses, more so on the right to encase the right carotid artery. Superiorly the mass contacts the undersurface of the optic chiasm left of the midline



**Fig. 2** (A) Mature plasma cells with round eccentrically placed nuclei. (B) Tumor cells showed widespread immunoreactivity for CD 138 (2B); no staining for chromogranin, synaptophysin, PLAP, EMA, CAM 5.2, CEA, prolactin, GH, ACTH, FSH, LH and TSH. The majority of plasma cells stained positive for  $\kappa$  light chains, indicating that this tumor was composed of a monoclonal population of plasma cells



FSH, LH and TSH. Histologic staining was positive for immunoglobulin, kappa ( $\kappa$ ) light chains, confirming that this tumor was a plasmacytoma.

Further endocrine and oncologic evaluation was performed as an outpatient (Table 1). Outpatient endocrine work-up revealed persistent diabetes insipidus requiring DDAVP replacement. This complication was attributable to surgery rather than progression of the intrasellar mass.

Post operatively an extensive investigation for myelomatous disease was undertaken including serum and urine protein electrophoresis, a bone survey and a bone marrow biopsy. Evidence of occult myelomatous disease was discovered, one month after surgery, with an elevation in IgG  $\kappa$  levels 1980 mg/dL (768–1632 mg/dL) in the serum and evidence of small lytic lesions <1 cm in the parietal and frontal bones of the skull. In addition, her bone marrow biopsy and aspirate revealed a normocellular bone marrow for her age (50–60%) with increased plasma cells (28%), consistent with involvement by a plasma cell dyscrasia. Urine protein electrophoresis was negative for Bence Jones proteins but IFEX indicated the presence of IgG  $\kappa$ .

Follow-up MRI of the head showed residual tumor in the sella with extension into the sphenoid and cavernous sinuses bilaterally. In addition, lytic lesions were identified in the parietal and frontal bones consistent with multiple myeloma. She underwent tumoricidal radiation directed to the residual tumor located in the sellar region; a cumulative dose of 5040cGy over a 5 week period. Her condition remained stable after radiation therapy. She was started on an intravenous bisphosphonate once monthly. Due to the presence of systemic myelomatous disease, she was treated with 3 cycles of chemotherapy with DVd (Doxorubicin, Vincristine, dexamethasone). Six months after the diagnosis of multiple myeloma, she underwent bone marrow stem-cell mobilization, followed by autologous stem cell transplantation.

A follow-up MRI at 10 months showed stabilization of the abnormality, in conjunction with a normal appearing pi-

tuitary. Endocrine work-up has not demonstrated evidence of evolving panhypopituitarism that may result from possible progression in size of the residual tumor or radiotherapy to the sella. Clinically the patient is doing well with minimal evidence of residual pituitary disease. Post-operative IgG is within normal limits and Bence Jones proteins are negative.

## Discussion

A literature review revealed 22 cases of patients in whom solitary plasmacytoma or multiple myeloma first appeared as a mass involving the sellar region. Sellar plasma cell tumors may mimic non-functioning invasive pituitary adenomas in both clinical presentation and imaging studies. Plasma cell tumors involving the sella turcica, without systemic manifestations of classic multiple myeloma, are invariably overlooked preoperatively because they are exceedingly rare.

Multiple Myeloma (MM) has become a well characterized entity since it was first reported in 1845 [2]. Plasma cell neoplasms may take the form of either MM or solitary plasmacytomas. MM is a multifocal tumor characterized by skeletal destruction with osteolytic lesions, pathological fractures, hypercalcemia, anemia, bone pain and serum monoclonal protein levels [3]. In contrast, a plasmacytoma is a localized collection of plasma tumor cells.

Multiple myeloma involving the nervous system most commonly manifests as encroachment on nerve roots or compression of the spinal cord [4]. Cranial and intracranial involvement by myelomatous disease is less frequently encountered, comprising 3% of 277 cases described by Silverstein et al. [4]. The majority of these cases had typical systemic manifestations of multiple myeloma prior to the development of cranial and intracranial involvement, in contrast to the case series that is presented in this review.

Harvey Cushing in 1932 described 4 cases of intracranial myeloma in his series of 2,000 intracranial tumors [5].

The presentation of a plasma cell neoplasm as a cranial or intracranial tumor is an infrequent phenomenon [4,6]. Clarke divided the manifestations of intracranial involvement into the following 3 groups – 1. Syndromes of cranial nerve palsies; 2. Intracranial tumor syndromes; 3. Intraorbital tumor syndromes. A plasmacytoma mimicking a pituitary adenoma as the initial manifestation of multiple myeloma represents a distinct subgroup of the syndrome of cranial nerve palsies. A review of the world literature by Spaar et al. [6], up to 1980, uncovered only 32 cases of intracranial plasmacytomas. It is even more rare for myelomatous disease to present for the first time clinically as a sellar mass and mimic a pituitary adenoma.

A plasmacytoma is one of a wide variety of intrasellar masses which can mimic a pituitary adenoma. It is believed that the origin of the tumor cells is the surrounding bone or the mucosa within the petrous or the sphenoid bone [7]. Attempts to differentiate between plasma cell tumors of the sellar region and other intrasellar processes and pituitary adenomas are of importance, as the course and treatment of these disease entities are different.

The differential diagnosis of an intrasellar or parasellar mass mimicking a pituitary adenoma is broad and complex (see Table 2). In the largest series of sellar masses reported to date [8], 47/1937 cases were misdiagnosed as pituitary adenomas pre-operatively.

The radiologic and clinical appearance of myelomatous disease involving the pituitary fossa is, in the absence of systemic manifestations of disease, similar to that of patients affected by non-functioning invasive pituitary adenomas. The characteristics of 20 previously reported cases and the current case are summarized in Table 3. Unfortunately, details of 2 cases are unavailable [8].

The mean age of patients at presentation was 56 years, ranging from 34 to 75 years. The oldest case reported was that of an Asian woman with multiple myeloma presenting for the first time as a parasellar syndrome with cranial nerve palsies [9]. There was a slight preponderance of females (62%) compared to males (38%). Similar to our case, all other cases presented with either headache and (or) visual compromise. The predominant presenting complaints were headaches (71%), diplopia (52%), visual loss (19%), facial numbness (10%), bloody nasal discharge (5%), eye pain (5%), craniofacial pain (5%) and photophobia (5%). Cranial nerve involvement was present in all but 3 cases at presentation [10–12]. The most common cranial nerve involved was cranial nerve VI (57%), followed by cranial nerves III (38%), IV (14%), V<sub>1</sub> (10%), I (5%) and VIII (5%).

Our findings of a well preserved anterior pituitary function are in agreement with the majority of reported cases. The preoperative endocrine assessment of these cases demonstrated a well preserved pituitary function. Pituitary function

**Table 2** Intrasellar masses masquerading as Pituitary Adenomas reported in the literature [1, 8, 23–34]

Primary Intracranial Tumors / Cysts
Craniopharyngioma
Pituitary Carcinoma
Schwannoma
Hamartoma of Rathkes cleft cyst
Mature Teratoma
Mixed tumor of the hypothalamus
Arachnoid cyst
Epidermoid cyst
Mucocoele
Primary Cranial Tumors
Chordoma
Multiple Myeloma/Plasmacytoma
Intra-osseous Meningioma
Intracranial granular cell tumor
Germinoma
Metastatic Tumors
Vascular Anomalies
Giant aneurysm of the carotid artery
Sellar hemangiopericytoma
Intrasellar vascular malformation
Infectious
Pituitary tuberculoma
Abscess of the pituitary gland
Intrasellar Cysticercosis
Inflammatory
Lymphocytic Hypophysistis
Sarcoidosis

was entirely normal in 13 cases. Information regarding pituitary function was not provided in some case reports. Only one patient [13] had evidence of pituitary dysfunction with evidence of an impaired cortisol response to insulin induced hypoglycemia, and a borderline low response of TSH to TRH. Another patient had evidence of concomitant primary hypothyroidism with an appropriately elevated TSH level [9].

Regarding imaging characteristics, all cases had evidence of sellar expansion and radiological evidence of destruction of the sellar floor. In cases of marked radiological pituitary fossa destruction, it is unusual to find well preserved anterior pituitary function. Normal or minimally disturbed anterior pituitary function in the setting of pituitary fossa destruction suggests that the primary lesion may lie outside the pituitary fossa [13]. Imaging studies revealed variable extrasellar tumor extension in all but one patient who did not have a CT head performed however in that particular case there was radiologic evidence of bony destruction of the sellar floor by plain X-ray [14]. Suprasellar expansion occurred in 7 cases (33%) and 57% had expansion into the parasellar region and the sphenoid sinus. As bony destruction can be seen with either a pituitary adenoma or a non-pituitary mass, this does not differentiate between the two entities.

**Table 3** Characteristics of cases with plasmacytomas mimicking pituitary adenomas

Case	Age and sex	Clinical symptoms	Cranial nerve palsy	CT scan expansion	Progression to myeloma (Mths)	Treatment	Reference
1	75 F	Headache Diplopia Photophobia	III & IV, VI (R)	Sphenoid sinus R Parasellar	Yes (0)	Rad Rx Chemo	9
2	44 M	Headache Visual loss (L)	None	Sphenoid sinus Suprasellar	Yes (1)	Unknown	10
3	34 F	Craniofacial pain	None	Sphenoid sinus Suprasellar	No	Rad Rx	11
4	53 F	Headache	None	Sphenoid sinus Parasellar (L) Suprasellar	No	Rad Rx	12
5	66 M	Headache Diplopia	VI (L)	Parasellar (R and L)	No	Rad Rx	13
6	65 M	Headache Diplopia	III (L & R) VI (R)	X-ray & carotid angiogram – sellar floor destruction & R sided sellar mass	Yes (3)	Rad Rx	14
7	62 F	Headache Visual loss	I	Sphenoid sinus Suprasellar	Yes (5)	Rad Rx	15
8	42 M	Headache Blurry vision	III & VI (L partial)	Sphenoid sinus Parasellar (L)	Yes (3)	Rad Rx Chemo	16
9	42 M	Headache Left Eye Pain Bloody nasal discharge	III VI (L partial)	Sphenoid sinus Parasellar (L) Suprasellar	Yes (3)	Rad Rx	17
10	47 M	Ptosis Retrobulbar pain	III & IV (L)	Parasellar (L)	Yes (0)	Unknown	18
11	51 F	Diplopia	?	Sphenoid sinus	No	Rad Rx Chemo	19
12	66 F	Headache Decreased hearing	VIII (L)	L petrous bone	No	Rad Rx	20
13	62 M	Headache Diplopia	VI (L)	Sphenoid sinus	Yes (0)	Unknown	35
14	58 F	Headache Facial numbness (R) Diplopia	V (R partial) VI (L) VII (partial)	Sphenoid sinus Parasellar Suprasellar	No	Rad Rx	36
15	57 M	Headache Diplopia	VI (L)	Suprasellar	Yes (0)	Rad Rx Chemo	37
16	50 F	Diplopia	VI (R partial)	Sphenoid sinus	No	Rad Rx	38
17	64 F	Diplopia	III, IV & VI (R & L)	Sphenoid sinus	Yes (22)	Rad Rx	39
18	73 F	Headache Visual loss Diplopia	III & VI (L)	Sphenoid & L parasellar sinuses	Yes (0)	Unknown	40
19	47 M	Diplopia	III (L)	Parasellar	Yes (18)	Rad Rx Chemo	41
20	57 F	Headache Visual loss	VI (R)	Sphenoid sinus Parasellar (R)	No	Rad Rx	42
21	57 F	Headache Facial numbness (R)	V (R partial)	Sphenoid sinus Parasellar	Yes (0)	Rad Rx Chemo Autologous stem cell transplant	Our case

Details of 2 cases by Sautner et al. [8] are unavailable.

Adapted from Mandagere et al.

R = right, L = left, Rad Rx = Radiation treatment, Chemo = Chemotherapy, ? = unavailable.

The difficulty in making a histologic diagnosis was highlighted in 5 cases that were originally reported as pituitary adenomas based on histology, but were later correctly diagnosed as myelomatous disease, after clinical expression of the occult disease or the use of electron microscopy [15–19]. These cases clearly demonstrate that the analysis of an atypical pituitary adenoma should include routine electron microscopy and immunohistochemical stains. In addition, the presence of monoclonal immunoglobulin on immunostaining distinguishes plasmacytomas from lymphocytic hypophysitis, which consists of a polyclonal B cell lineage and is high on the differential diagnosis of a sellar mass. All patients underwent surgical removal or biopsy of the mass except in one case [9] where multiple myeloma was diagnosed simultaneously and the patient received local radiation to the intracranial mass and conventional chemotherapy, with good clinical outcome. In the present case, the diagnosis of multiple myeloma was made after transphenoidal surgery in the absence of any clinical or biochemical evidence of a lytic bone disorder on presentation.

The majority of reported cases similarly progressed to multiple myeloma or had occult myeloma at the time of presentation. Biochemical data suggestive of myelomatous disease was present in 4 patients, at the time of initial assessment. The majority of cases had no known plasma cell tumor diagnosis at the time of sellar biopsy. The diagnosis of myelomatous disease in these cases was made following sellar mass biopsy. Despite careful endocrine workup, neurological and radiological assessment, the majority of cases except for one case [9], were diagnosed at pathology after transphenoidal resection. In our case, myelomatous disease was not clinically apparent on initial presentation. Both total protein and albumin levels were low; there was no evidence of renal insufficiency, hypercalcemia, anemia, bone pain or pathologic fractures. The time between initial diagnosis of the intrasellar plasmacytoma and first manifestations of systemic myeloma in 11 cases was variable (0–22 months). The longest disease free survival is 9 years following diagnosis of a solitary intrasellar plasmacytoma [20]. The clinical outcome is poor, as the majority of patients progress to systemic multiple myeloma over a variable time period. Determining whether a plasmacytoma is solitary is important prognostically. Over 50% of patients with an apparently solitary plasmacytoma of bone will develop multiple myeloma within 10 years; 10% have either a local recurrence of the plasmacytoma or another solitary plasmacytoma [21].

Traditional treatment modalities have included radiation therapy for local or residual disease and chemotherapy for systemic myeloma. Radiation therapy directed towards the sellar lesion was administered to 71% after initial surgery and histopathological diagnosis of a plasma cell tumor. Plasma cell tumors are typically radiosensitive however a potential

exists for treatment failure with radiation therapy in the setting of incomplete resection. Systemic chemotherapy was administered to 29% following the diagnosis of multiple myeloma. In contrast to previously reported cases, our patient was treated with autologous bone marrow stem cell transplantation, in addition to conventional treatment. Survival post autologous transplantation is superior to that associated with standard therapies, with a median survival of about 5 years reported [22].

In a large transphenoidal surgical series of 911 patients with sellar masses, the majority of sellar masses were either hormone secreting or non-secretory pituitary adenomas (91%); only 9% of intra-sellar masses were of non-pituitary origin [1]. The most common clinical presentation of patients with non-pituitary masses included signs of anterior pituitary hormone deficits (37%), hyperprolactinemia (28%) and DI (6%). These particular clinical deficits are common at presentation in both pituitary adenomas and non-pituitary lesions and do not help in differentiating between the two entities. This is in contrast to the series of patients who presented with plasmacytomas; in fact only 1 of 23 cases reported had significant evidence of anterior pituitary hormone dysfunction. Similar to the current series of patients, no patient with a pituitary adenoma in the Freda series presented with DI; therefore confirming that intra-sellar myelomatous lesions may mimic pituitary adenomas in clinical presentation. In the Freda series of non-pituitary sellar masses, visual loss and cranial neuropathy (III, IV, VI) were identified in 24% and 20% respectively on presentation. In contrast, patients with intrasellar plasmacytomas in this series had visual loss and cranial neuropathy, 31% and 66% respectively, on presentation.

Our case illustrates several important features of myelomatous disease in the pituitary fossa: (1) it can mimic a “non-functional” invasive pituitary adenoma in clinical presentation and imagery; (2) cranial nerve involvement may help to distinguish it from a benign adenoma; (3) anterior pituitary function may be well preserved despite significant sellar destruction. Based on our case and review of the world's literature, well preserved pituitary function associated with destruction of the pituitary fossa and the sudden development of cranial neuropathies are clues that the primary lesion may be an intrasellar plasmacytoma.

It is rare for myelomatous disease to present for the first time clinically as a sellar mass and masquerade as a non-functional invasive pituitary macro-adenoma. In the absence of overt systemic symptoms or signs of myeloma, pre-operative diagnosis is impossible. Clinical presentation, radiologic features and light microscopic features may be indistinguishable from a chromophobe pituitary adenoma, highlighting the importance of immuno-histochemical staining or the use of electron microscopy in atypical cases. In patients with intrasellar lesions that erode surrounding bone,

plasma cell tumors must be considered even in the absence of other characteristic findings of this disease. In contrast to other intrasellar and parasellar masses, patients with intrasellar plasmacytomas are more likely to have cranial neuropathies and normal anterior pituitary function in the setting of significant bony destruction of the sella.

## References

1. Freda PU, Wardlaw SL, Post KD (1996) Unusual causes of sellar/parasellar masses in a large transphenoidal surgical series. *J Clin Endocrinol Metab* 81:3455–3459
2. Clamp JR (1967) Some aspects of the first recorded case of multiple myeloma. *Lancet* 2:1354–1356
3. Richardson PG, Kassarian A, Jing W (2004) Case records of the Massachusetts General hospital. Case 38 – 2004: A 40-year old man with a large tumor of the skull. *N Engl J Med* 351(25):2637–2645
4. Silverstein A, Doniger D (1963) Neurologic complications of myelomatosis. *Arch Neurol* 9:102–110
5. Cushing H (1932) Intracranial tumors; notes upon a series of two thousand verified cases and the surgical mortality percentages pertaining there to. Springfield, IL: Thomas.
6. Spaar FW (1980) Paraproteinemias and multiple myeloma. *Handbook of clinical Neurology* vol 39; North Holland, Amsterdam, pp. 131–180
7. Clarke E (1954) Cranial and intracranial myelomas. *Brain* 77:61–68
8. Sautner D, Saeger W, Ludecke DK (1993) Tumors of the sellar region mimicking pituitary adenomas. *Exp Clin Endocrinol* 101:283–289
9. Kanoh T, Okuda T, Hayashi M, Yumoto Y (1996) Multiple myeloma presenting as parasellar syndrome and cranial nerve palsies. *Rinsho Ketsueki* 37(3):260–264
10. Estopinan V, Riobo P, Fernandez G, Varela C (1985) Plasmacitoma intrasellar simulando un adenoma hipofisario [letter]. *Med Clin (Barc)* 20:128
11. Jacquet G, Vuillier J, Viennet A, Godard J, Steimle R (1991) Plasmocytome solitaire stimulant un adenoma hypophysaire. *Neurochirurgie* 37:67–71
12. Mandagere KA, Schimke RN, Kyner JL, Bhatia PS (1998) An unusual sellar mass—solitary plasmacytoma. *Endocr Pract* 4:382–386
13. Evans PJ, Jones MK, Hall R, Scanlon MF (1985) Pituitary function with a solitary intrasellar plasmacytoma. *Postgrad Med J* 61:513–514
14. Sanchez JA, Rahman S, Strauss RA, Kaye GI (1977) Multiple myelomasquering as a pituitary tumour [letter]. *Arch Pathol Lab Med* 101:55–56
15. Poon MC, Prchal JT, Murad TM, Galbraith JG (1979) Multiple myeloma masquerading as a chromophobe adenoma. *Cancer* 43:1513–1515
16. Harrison LB, Schnall S, Cardinale FS, Farber LR (1987) Multiple myeloma presenting as a pituitary tumor [letter]. *Int J Radiat Oncol Biol Phys* 13:653–654
17. Bitterman P, Ariza A, Black RA, Allen WE III, Lee SH (1986) Multiple myeloma mimicking pituitary adenoma. *Comput Radiol* 10:201–205
18. Hornedo J, Calvo F, Aramburu P et al (1982) Plasmacitoma extramedular Ig D lambda simulando un adenoma de hipofisis. *Med Clin (Barc)* 79:377–379
19. Bindal AK, Bindal RK, Van Loveren H, Sawaya R (1995) Management of intracranial plasmacytoma. *J Neurosurg* 83:218–221
20. McLaughlin DM, Gray WJ, Jones FGC, Mirakhor M, McCance DR, Sheridan B, Atkinson AB (2005) Plasmacytoma: An unusual cause of a pituitary mass lesion. A case report and a review of the literature. *Pituitary* 7(3):171–177
21. Holland J, Trenkner DA, Wasserman TH, Fineberg B (1992) Plasmacytoma: treatment results and conversion to multiple myeloma. *Cancer* 69:1513–1517
22. Attal M, Harousseau JL, Stoppen AM et al (1996) A prospective, randomized trial of autologous bone marrow transplantation and chemotherapy in multiple myeloma. *N Engl J Med* 335:91–97
23. Esposito F, Cappabianca P, Del Basso De Caro M, Cavallo LM, Rinaldi C, De Divitiis E (2004) Endoscopic endonasaltransphenoidal removal of an intra-suprasellar schwannoma mimicking a pituitary adenoma. *Minim Invasive Neurosurg* 47(4):230–234
24. Haridas A, Ansari S, Afshar F (2003) Chordoma presenting as a pseudoprolactinoma. *Br J Neurosurg* 17(3):260–262
25. Matsumoto S, Hayase M, Imamura H, Oda Y, Kikuchi H, Katayama M, Ishihara T (2001) A case of intrasellar meningioma mimicking pituitary adenoma. *No Shinkei Geka* 29(6):551–557
26. Halbauer DJ, Meszaros I, Doczi T, Kajtar P, Pajor L, Kovacs K, Gomori E (2003) Rare sellar region tumors. *Path Oncol Res* 9(2):134–137
27. Uzal MC, Kocak Z, Doganay L, Tokatli F, Caloglu M, Kilincer C (2001) Pituitary metastasis mimicking a macroadenoma from carcinoma of the larynx: a case report. *Tumori* 87(6):451–454
28. Barontini F, Ammannati F, Gagliardi R, Mauri S, Mannelli M, Mennonna P (1994) A further case of giant intrasellar carotid aneurysm mimicking a pituitary adenoma: The relevance of a multivariate approach in differential diagnosis. *Ital J Neurol Sci* 15(7):369–372
29. Morrison DA, Bibby K (1997) Sellar and suprasellar hemangiopericytoma mimicking pituitary adenoma. *Arch Ophthalmol* 115(9):1201–1203
30. Gould TJ, Johnson LN, Colapinto EV, Spollen LE, Rodriguez FJ (1996) Intrasellar vascular malformation mimicking a pituitary macroadenoma. *J Neuroophthalmol* 16(3):199–203
31. Kumar N, Singh S, Kuruvilla A (2001) Pituitary tuberculoma mimicking adenoma: Magnetic resonance imaging. *Australas Radiol* 45(2):244–246
32. Podgorski JK, Rudnicki SZ, Potakiewicz Z, Delimat L, Bolewski J (1991) A case of primary abscess of the pituitary gland. *Neurol Neurochir Pol* 25(5):683–688
33. Boecher-Schwartz HG, Hey O, Higer HP, Pernecky A (1991) Intrasellar cysticercosis mimicking a pituitary adenoma. *Br J Neurosurg* 5(4):405–407
34. Skandarajah A, Ng WH, Gonzales M, Kaye AH (2002) Lymphocytic hypophysitis mimicking pituitary macroadenoma. *J Clin Neurosci* 9(5):586–589
35. Dhanani AN, Bilbao JM, Kovacs K (1990) Multiple myeloma presenting as a sellar plasmacytoma and mimicking a pituitary tumor: report of a case and a review of the literature. *Endocr Pathol* 1:245–248
36. Juneau P, Schoene WC, Black P (1992) Malignant tumors in the pituitary. *Arch Neurol* 49:555–558
37. Kerty E, Nakstad PH (1984) Plasmacytoma masquerading as a pituitary tumor [letter]. *J Neurol Neurosurg Psychiatry* 47:99–100

38. Losa M, Terreni MR, Tresoldi M et al (1992) Solitary plasmacytoma of the sphenoid sinus involving the pituitary fossa: A case report and review of the literature. *Surg Neurol* 37:388–393
39. Urbanski SJ, Bilbao JM, Horvath E, Kovacs K, So W, Ward JV (1980) Intrasellar solitary plasmacytoma terminating in multiple: A report of a case including electron microscopical study. *Surg Neurol* 14:233–236
40. Vallat M, Vallat JM, Loubet A, Leboutet MJ, Robin A (1981) Plasmacytome a localization hypophysaire: rapport d'un cas. *Bull Soc Ophthalmol Fr* 81:355–356
41. Vaquero J, Areitio E, Martinez R (1982) Intracranial parasellar plasmacytoma. *Arch Neurol* 39:738
42. Weber J, Jaksche H (1999) Solitary plasmacytoma of the pituitary area. *Acta Neurochir (Wein)* 141(2):219–220