FROM DIABETES INSIPIDUS TO SELLAR XANTHOGRANULOMA – A “YELLOW BRICK ROAD” DEMANDING TEAM-WORK

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Abstract

Xanthogranulomas are inflammatory lesions exceptionally rarely occurring in the sellar region. Sellar xanthogranulomas (SXG) result from secondary hemorrhage, infarction, inflammation or necrosis upon existing craniopharyngioma (CP), Rathke's cleft cyst (RCC) or pituitary adenoma (PA), or represent a stage in xanthomatous hypophysitis evolution. "Pure SXG" are independent of a preexisting lesion.

A 70 year old male patient, laryngeal cancer survivor, presented with central diabetes insipidus (CDI). MRI revealed an intra-suprasellar mass of uncertain origin. Transsphenoidal surgery resulted in an efficient lesion resection with maximal pituitary sparing. Pathological report has confirmed SXG without conclusive identification of preexisting sellar lesion. Age at presentation and gender were atypical for SXG. The most frequent presenting signs of SXG were absent.

Most SXG are initially misdiagnosed as CP, RCC or PA. Preoperative clinical and radiological uncertainty may impact operative planning. Differentiating from CP is crucial, due to divergent operative target goals and prognosis. Intraoperative frozen section analysis could guide surgical extensiveness. Close collaboration must include endocrinologist, neuroradiologist, neurosurgeon and pathologist. Quantity and quality of provided tissue are essential for avoiding bias in pathohistological analysis of cystic or heterogenous lesions. Awareness is needed of new pathological entities in the sellar-parasellar region. SXG should be considered in differential diagnosis of CDI-causing sellar lesions.

Key words: pituitary, sellar mass, polyuria-polydipsia, cholesterol granuloma, transsphenoidal surgery.

INTRODUCTION

Xanthogranulomas (XG) are inflammatory

lesions, primarily affecting the skin, hallmarked by xanthoma cells – lipid laden histiocytes and cholesterol clefts causing gross yellow-color (1). Intracranial XG are infrequent (1.6-7%) most often involving choroid plexus (2). Sellar region xanthogranulomas (SXG) are exceptionally rare (3). SXG prevalence in large surgical cohorts was 0.6-2% in adults, reaching 10% in children (4-7). Mean presenting age was 36.6 years, with a female predominance of up to 60% (3).

Rarity of SXG obscures full understanding of their etiology, diagnostics, management and prognosis. Preoperative distinction from more common sellar lesions is challenging. An incidentally discovered sellar mass most likely represents a pituitary adenoma (PA) in 83%, a Rathke's Cleft Cyst (RCC) in 19-53%, another cyst in 20%, craniopharyngioma (CP) in 15-27%, meningioma in 6-15% or a metastasis in 5% (8). Central diabetes insipidus (CDI) is often idiopathic (13%), iatrogenic after neurosurgery (13%) or associated with one of sellar-parasellar lesions: germ cell CNS tumors (in 23%), CP (in 19%), inflammation (in 9%), metastasis (in 6%), RCC (in 4%) or rarely with a PA (in 2%)(9). SXG can be complicated by CDI in up to 30%, but rarity of SXG makes it an overall exceptionally rare cause of CDI often not considered in differential diagnosis. A total of only 9 SXG adults associated with CDI at presentation have been reported in literature (2-4, 10-12).

Presenting SXG imaging may simulate many other sellar lesions: CP, PA, RCC, cysts, pituitary abscess or intracranial aneurysm. In addition, MRI findings may evolve over the course of SXG progression. SXG are most often intra-suprasellar (67%), with suprasellar origin, less frequently only suprasellar (22%) and rarely exclusively intrasellar (11%)(3, 4). Suprasellar extension is more prevalent in children (92%) including

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hypothalamic involvement in 46% (7). Cavernous sinus invasion is universally absent (3,13,14). In most cases a T1-weighted (T1w) hyperintensity is described (in 83%) - probably corresponding to cystic content and cholesterol clefts. T2-weighted (T2w) intensity is heterogeneous in 43% - particularly as necrosis and hemorrhage occur (3, 4). A fluid-debris level may appear at the lesion's depending portion, marking interface between high signal xanthogranulomatous component and hypointense necrotic debris (15). Contrast enhancement is most often peripheral, but may be homogenous or heterogeneous. Calcifications are mostly absent in adults, but are found in up to 38% children (3, 7, 13, 14). MRI stalk thickening is in some studies observed in up to 83% (4).

CASE REPORT

A 70 years-old male was referred for polyuria and polydipsia, first observed one year ago. Daily diuresis reached 15 liters. He was treated for type 2 diabetes mellitus for 8 years, being insulin dependent for a year. Three years before he was operated for laryngeal carcinoma. Ears-nose-throat controls with chest X-ray and liver ultrasound revealed no disease remnant or recurrence. At presentation he reported no current or previous severe headaches, nor visual field impairment. The examination revealed no clinical signs of hormonal excess, nor of dehydration or fluid retention. Visual field and ocular motility were unimpaired on examination. Computerized perimetry was normal. Profile skull X ray revealed marginally enlarged sella turcica without intrasellar calcifications. He was moderately obese (BMI 31.9kg/m²). No

xanthelasma, skin xanthomas nor corneal arcus were identified. Liver ultrasound revealed mild steatosis. Glycemic control was satisfactory. Liver function tests and serum lipids (off treatment) were normal. Serum angiotensin converting enzyme, calcium and phosphate were normal. Basal hormonal evaluation revealed no anterior pituitary deficiency other than marginally lowered IGF-1. Serum prolactin was normal in daily profile (Table 1). Water deprivation associated with vasopressin sensitivity test confirmed partial CDI (Table 1). As advised by endocrinologist, desmopressin (DDAVP) treatment was introduced, normalizing the fluid balance.

Pituitary MRI disclosed borderline enlarged sella and a posterior sellar lesion of 15mm with suprasellar extension but no contact with the optic chiasm. The lesion was hyperintense in T1w and of an moderate intensity in T2w images. Possible prior hemorrhage was suspected (Fig. 1 A-C).

In the view of an intra-suprasellar mass of uncertain origin, causing CDI and possible partial hypopituitarism, the patient was referred to transsphenoidal surgery (TSS) targeted at mass reduction. Perioperative hydrocortisone support was administered prophylactically. Microscopic TSS and postoperative recovery were uneventful. Intraoperatively a white-colored soft mass was observed in the posterior part of anterior pituitary filled with yellow-colored content.

Multiple yellow-colored soft-elastic tissue fragments, 10x11x3mm in cumulative size, were received for pathohistological analysis. Immunohistochemistry identified numerous macrophages (CD68+, GFAP-, S-100-, CK8/18-,

Table 1. Preoperative and postoperative basal hormonal status and water deprivation test results

Baseline anterior pituitary hormonal status at presentation		Postoperative Follow up	Normal range
fT4	12.3	10.5	(7-18) ng/L
TSH	0.67	2.4	(0.3-5.5) mIU/L
Cortisol (8 am)	417	396.5	(131-642) nmol/L
Testosterone	12.8	2.7	(8.2-34.8) nmol/L
SHBG	32	28	(9-54) nmol/L
FSH	18.7	9.9	(1.3-8.1) IU/L
LH	12.9	3.5	(1.0-5.3) IU/L
PRL (8h, 11h, 13h)	148.9...214.2...276.2		(52.2-461.1) mIU/L
PRL		153.5	
IGF-1	60.7	44.0	(69-200) ng/mL
Preoperative water deprivation test			
<i>Plasma</i>		<i>Urine</i>	
Baseline Plasma Osmolarity	296 mOsm/L	Baseline urine specific gravity	1.005
Maximal Plasma Osmolarity	305 mOsm/L	Baseline Urine Osmolarity	232 mOsm/L
		Maximal Urine Osmolarity	246 mOsm/L
		Urine Osmolarity after DDAVP	548 mOsm/L

Figure 1. MRI of sellar region – Preoperative: (A) sagittal T1w (B) coronal T1w (C) coronal T2w. – Postoperative: (D) sagittal T2w (E) coronal T1w. Arrows indicate the location of sellar mass.

Synaptophysin-), rare foreign-body giant cells containing intracytoplasmic cholesterol crystals, numerous lymphocytes and rare hemosiderophages. One tissue fragment was partly covered by squamous epithelium (CK8/18+ p40+, membranous β -catenin+). Few fragments of anterior pituitary tissue were identified (CK8/18+, Synaptophysin+, preserved acinar pattern) adherent to or infiltrated by sporadic macrophages (CD68+) (Fig. 2 A-E). Histopathological analysis was concluded as XG which potentially could be a consequence of a pre-existing lesion, demarcated by small fragments of squamous epithelium of unknown origin (Rathke's cleft cyst with squamous metaplasia or papillary craniopharyngioma). The pre-existence of pituitary adenoma in the analyzed material was excluded by negative immunohistochemical staining for Synaptophysin, a neuroendocrine marker which is unequivocally observed in all pituitary adenomas.

On a six months endocrine follow-up visit, the patient was generally well, with normal fluid balance, serum sodium and urine osmolality on DDAVP. Follow up anterior pituitary status revealed normal postoperative basal cortisol and thyroid function tests, but low testosterone, low FSH and LH and low IGF-1, indicating postoperative partial hypopituitarism (Table 1). Postoperative MRI confirmed a successful mass

reduction with only a minor postero-basal sellar T1w-hypointense lesion of 3mm, no suprasellar extension and no stalk deviation (Fig. 1 D, E).

DISCUSSION

We have described a rare case of a sellar region xanthogranuloma (SXG) presenting with isolated partial central diabetes insipidus (CDI) in a 70 year old male laryngeal cancer survivor. Transsphenoidal surgery provided an efficient resection of intra-suprasellar mass with maximal pituitary sparing. Pathology confirmed SXG with a small fragment of squamous epithelium, the origin of which could not be precisely determined (Rathke's cleft cyst with squamous metaplasia vs. papillary craniopharyngioma). Age at presentation and gender were atypical for SXGs which favour young adults and women. The most frequent presenting signs of SXG were absent. Prior hypophysitis was improbable based on the absence of headaches or significant anterior pituitary deficit. Squamous epithelium fragments signalled an underlying lesion, but its origin could not be conclusively determined. Pituitary adenoma (PA) was improbable clinically and finally excluded histologically. Nuclear p40 positivity and membranous beta-catenin positivity ruled out

Figure 2. Photomicrography of pathohistological and immunochemical specimen: (A) Giant cells of foreign body type with cholesterol clefts in the cytoplasm near normal anterior pituitary (down, right, demarcated by an oval line) (HE, x400); highlighted by CD68 immunostaining (x400) (B); A fragment of epithelium (HE, x400) (C) for which squamous nature was confirmed by nuclear p40 (D) and membranous β -catenin (E) immunostaining.

craniopharyngioma (CP), leaving an unconfirmed possibility of an underlying squamous metaplasia of Rathke's cleft cyst (RCC). A possibility of a pure SXG could also not be excluded.

SXG were inaugurated as a separate entity in 1999 as Paulus *et al.* reported distinctive histological features in patients originally diagnosed as CP. (1) Understanding of these rare lesions is still limited, their origin being controversial and pathogenesis probably heterogeneous. Xanthogranulomatous reaction may result from chronic inflammation in CP, or at RCC wall, from a traumatic or spontaneous hemorrhage into RCC or other cystic lesions. PAs are associated with SXG in 0.6-2.2%, typically non-functioning macroadenomas in middle-age women, involving subclinical apoplexy, and a higher risk of hypopituitarism (16). A giant-cell granuloma may result from significant or repeated hemorrhage on the background of adeno- or infundibuloneuro-hypophysitis (17). SXG is considered by some to be a part of a xanthomatous sellar lesions spectrum, ranging from xanthomatous hypophysitis (XH), xanthogranulomatous hypophysitis to SXG (18). SXG cases described in the literature were related to CP in 32.5%, to RCC in 12.5-86%, to PA in 9.4% and

to XH in 3.8%. Isolated SXG was described in 41.8% (2,19). Pure SXG, lacking an epithelial component, is still disputed as a distinct entity. Its confirmation and delineation from other subtypes rely on adequate material being provided for histological analysis.

Clinical presentation of SXG usually involves headache (50%), visual impairment (63-67%), or hypopituitarism (3). CDI at presentation is found in 33-43% and anterior pituitary dysfunction in 67% (2,3). Our patient presented without headaches or visual disturbances (most prevalently associated with SXG) but with isolated partial CDI. Previous 9 cases of adults with SXG associated with CDI at presentation are summarized in Table 2.

Preoperative differentiation of SXG from CP, RCC, lymphocytic hypophysitis, PA apoplexy or pituitary abscess is difficult. Majority of reported cases were initially misjudged as CP or PA (3, 6). Differentiation against CP is crucial due to different prognosis and surgical objectives. CP has bimodal age peaks around 5 and 50 years while SXG favors adolescents and young adults. SXG is associated with a smaller lesion size, longer preoperative history, lower incidence of calcifications and visual impairment,

Table 2. Summary of previously published adult cases (and our patient) of SXG associated with central diabetes insipidus at presentation

Author, year	Age	Sex	Clinical picture at presentation	Pituitary function at presentation	MRI at presentation	Histopathology	Ref.
Hama S, 1999	48	F	Polyuria-polydipsia	Partial hypopituitarism (preserved HPA axis)	Pituitary stalk thickening, enlarged pituitary with a cystic lesion, strong contrast enhancement, loss of posterior pituitary hyperintensity	RCC, lymphocytes, neutrophils, foamy cells, fibrosis, capillary proliferation	(10)
Sugata S, 2009	26	M	Polyuria, bitemporal hemianopsia, fatigue	Panhypopituitarism	Suprasellar mass 30mm, isointense with patchy T1w hypointensity, heterogeneously enhanced, T2w hypointense	Fibrous tissue, cholesterol clefts, multinucleated giant cells, hemosiderin deposits, no epithelial cells	(11)
Amano K, 2013	20	M	Headache	Panhypopituitarism, hyperprolactinaemia	Intra-supra sellar 12mm, T1w hyperintense, T2 iso-hyperintense, rim contrast enhancement	Xanthochromic fluid. Granulomatous tissue with cholesterol clefts, hemosiderin deposits, multinucleated giant cells, and chronic inflammatory infiltrates. Components of RCC	(2)
Amano K, 2013	68	M	Headache, Visual field impairment	Panhypopituitarism	Intra-supra sellar 23mm, T1w low-hyperintense, T2w low-hyperintense, rim contrast enhancement	Cholesterol-cleft granulomas Hemosiderin deposits. Chronic inflammation infiltrates. Calcification. Components of RCC.	(2)
Bao SS, 2014	35	M	Polyuria-polydipsia (at age of 15), severe headache, hypogonadism	Hypogonadotropic hypogonadism	Suprasellar mass 30mm, mixed T1w intensity, heterogeneous and hyperintense on T2w	Xanthogranuloma	(12)
Hernandez-Estrada RA, 2017	35	F	Polyuria, polydipsia, headache	<i>n.a.</i>	Intrasellar mass 13mm posterior pituitary gland. heterogeneous T1w and T2w hyperintensity	Cholesterol granuloma (Immunohistochemistry for b-catenin and BRAF V600E negative)	(3)
Hernandez-Estrada RA, 2017	32	F	Polyuria, polydipsia, headache, amenorrhea	<i>n.a.</i>	Sellar-suprasellar ovoid lesion 25mm T1w and T2w hyperintense	Cholesterol granuloma (Immunohistochemistry for b-catenin and BRAF V600E negative)	(3)
Hernandez-Estrada RA, 2017	40	F	Polyuria, polydipsia	<i>n.a.</i>	Sellar-Suprasellar, 12mm T1w hyperintensity, T2w heterogeneous intensity, calcifications	Cholesterol granuloma with RCC epithelium (Immunohistochemistry for b-catenin and BRAF V600E negative)	(3)
Ved R, 2018	63	F	Polyuria, polydipsia	Partial hypopituitarism, hyperprolactinemia	Intra-suprasellar 13mm, cystic, rim enhancement, T1w hyperintense, stalk thickening and enhancement, expanded sella	Necrosis, CD68+ cells (Absent: multinucleated giant cells, hemosiderin, cholesterol clefts, granuloma, squamoid epithelium, histiocytes, foamy macrophages, T and B lymphocytes, eosinophils) Numerous macrophages (CD68+, GFAP-, S-100-, CK8/18-, Synaptophysin-), Rare foreign-body giant cells containing intracytoplasmic cholesterol crystals, numerous lymphocytes and rare hemosiderophages. Squamous epithelium fragment (CK8/18+ p40+, membranous β -catenin +).	(4)
Present case	70	M	Polyuria-polydipsia	Possible partial hypopituitarism (GH deficiency)	Posterior-sellar and suprasellar lesion 15mm, T1w hyperintense, T2w intermediary intensity. Possible prior hemorrhage.		

n.a. – not available; RCC - Rathke's cleft cyst; Ref. - reference.

better resectability, and more favorable outcome than CP (7). In the case of our patient, a 70 year old cancer survivor, preoperative differential diagnosis of a CDI-causing sellar-suprasellar lesion included: a metastasis, CP, germ cell CNS tumor, RCC or a PA.

In MRI assessment of cystic sellar-parasellar lesions, potential indicators of SXG are: thin peripheral rim of low signal intensity with peripheral enhancement, suprasellar tumor epicenter, absence of calcifications and cavernous sinus invasion (4). Typical radiological signs of SXG were lacking in our patient and SXG was not considered in the radiological differential diagnosis. Preferred treatment for SXG is surgical mass-reduction and optic chiasm decompression, with pituitary stalk sparing (4). Histologically benign nature makes aggressive complete removal unnecessary (13). However, since preoperative distinction from CP is difficult, gross tumor resection often remains surgical goal for cystic sellar-parasellar lesions of uncertain origin (4). Intraoperative frozen section analysis could alleviate decision on surgical extensiveness. (2, 3). Adhesiveness to adjacent structures sometimes complicates surgery. Most reported patients were treated transsphenoidally, yet a marked suprasellar extension may demand transcranial approach (13). Providing sufficient tissue quantity and quality for histological analysis is crucial to minimize sampling bias risk particularly for cystic or heterogeneous lesions (4). Complete SXG resection was achieved in 64-71%, and subtotal in 29-36% (3,13). Visual field impairment frequently recovered postoperatively while hypopituitarism and CDI persisted (3,13). Surgical intervention early in the development of the SXG degenerative changes (adhesion to and infiltration of the normal pituitary) may contribute to reduced risk of postoperatively persisting endocrine dysfunction (20).

No recurrence or regrowth of residual lesions were observed after average follow-up of 7 years (7,13). Favorable neurosurgical outcome of SXG and low recurrence rate affirm their benign and slow growing nature. Limit of available data still invites caution and close follow-up. Treatment of our patient with transsphenoidal surgery was successful with a minor remnant on six months follow-up MRI. CDI persisted and partial hypopituitarism evolved, sparing basal cortisol or thyroid function.

Pathological diagnosis of SXG is challenging, with features overlapping or presenting in transitional forms. Although SXGs are proposed as a distinct entity, they frequently represent end-stage of other lesions, including CP, RCC, PA, granulomatous hypophysitis.

Careful search for epithelium remnants provides clues for SXG origin. Nuclear β -catenin positivity indicates a papillary CP. Nuclear p40 positivity delineates a squamous epithelium, which could result from squamous metaplasia of another epithelium (e.g. RCC). Synaptophysin positivity can demarcate remnants of PA. After excluding all differential diagnoses, SXG is diagnosed, as in our case. Macroscopical features of SXG include a fibrous capsule densely adherent to dura, bulk of tumor with non-hemorrhagic soft tissue and cystic components (3). Cholesterol clefts and lymphocytic infiltrates are nearly ubiquitously present (2). Hemosiderin deposits (indicating past hemorrhage) are described in 89%, fibrosis in 86%, foreign-body giant cells in 78%, eosinophilic necrotic debris in 59%, macrophages accumulation in 59%. Focal cyst-like epithelium is found in 35% (1, 17).

In conclusion, a 70 year old male cancer survivor, presenting with diabetes insipidus, was diagnosed with a sellar-suprasellar mass on imaging. Successful transsphenoidal operation led to diagnosis of sellar region xanthogranuloma (SXG). Age at presentation and gender were atypical for these exceptionally rare lesions favoring young adults and women. Most frequent SXG signs (headaches or visual impairment) were absent. Awareness is raised of new pathological entities in the sellar-parasellar region. SXG should be considered in differential diagnosis of CDI-causing sellar masses. Preoperative clinical and radiological uncertainty may impact peri-operative planning. Close collaboration is needed between an endocrinologist, neuroradiologist, neurosurgeon and pathologist. Quantity and quality of provided operated tissue are essential for pathohistological analysis, particularly in cystic or heterogeneous pituitary lesions.

Conflict of interest

The authors declare that they have no conflict of interest.

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Consent

Full informed consent was provided from the patient whose anonymity is preserved.

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