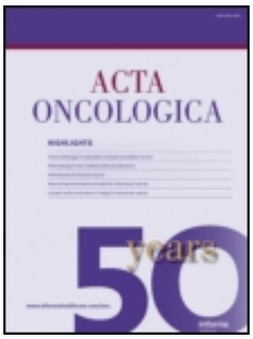

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Pituitary Adenoma Neuronal CHoristoma – The PANCH syndrome

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reactions, had the drug administered via catheter with a subcutaneous port system, introduced through the subclavian vein. We speculate that administering the drug through the catheter into the right atrium could result in uneven concentrations in the pulmonary vascular bed, and the transiently increased concentrations in lung capillaries could trigger an overwhelming inflammatory response resulting in severe infusion reaction.

In conclusion, although the pathogenesis of infusion reactions after cetuximab remains elusive, re-administration of the drug may be safe in an ICU setting.

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References

[1] Patel DD, Goldberg RM. Cetuximab-associated infusion reactions: Pathology and management. Oncology 2006;20: 1373–97.

[2] Helbling D, Borner M. Successful challenge with the fully human EGFR antibody panitumumab following an infusion reaction with the chimeric EGFR antibody cetuximab. Ann Oncol 2007;18:963–4.

[3] O'Neil BH, Allen R, Spigel DR, Stinchcombe TE, Moore DT, Berlin JD, et al. High incidence of cetuximab-related infusion reactions in Tennessee and North Carolina and the association with atopic history. J Clin Oncol 2007;25:3644–8.

[4] Melichar B, Cerman J, Malírová E. Successful management of infusion reaction accompanying the start of cetuximab therapy. Support Care Cancer 2007;15:445–9.

[5] Melichar B, Kalabova H, Urbanek L, Malirova E, Solichova D. Serial urinary neopterin measurements reflect the disease course in patients with epithelial ovarian carcinoma treated with paclitaxel/platinum. Pteridines 2007;18:1–7.

[6] van der Kolk LE, Grillo-Lopez AJ, Baars JW, Hack CE, van Oers MHJ. Complement activation plays a key role in the side-effects of rituximab treatment. Br J Haematol 2001;115: 807–11.

[7] Lenz HJ. Management and preparedness for infusion and hypersensitivity reactions. Oncologist 2007;12:601–9.

[8] Schwartzberg LS, Stepanski EJ, Fortner BV, Houts AC. Retrospective chart review of severe infusion reactions with rituximab, cetuximab, and bevacizumab in community oncology practices: Assessment of clinical consequences. Support Care Cancer 2008;16:393–8.

Pituitary Adenoma Neuronal CHoristoma – The PANCH syndrome

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To the Editor

Pituitary Adenoma Neuronal CHoristoma (PANCH syndrome) is a rare pathological entity of pituitary with unknown clinical impact.

Case history

Pituitary adenomas are common benign brain tumours that are easily diagnosed on biopsy specimens by typical morphological features. However, rarely, they may have divergent differentiation, necessitating the use of immunohistochemistry (IHC) for better characterization.

A 43-year-old lady presented with progressive weight gain, enlargement of extremities and hirsutism of 2 years duration. MRI scan showed focal enlargement of the pituitary gland suggestive of a macroadenoma (Figure 1). There was no mass effect on the optic pathway and her endocrine profile was normal (T3-2.5 nmol/l, T4-129.1 nmol/l, TSH-0.24 mIU/ml, Prolactin-4.48 ng/ml, GH-2.15 ng/ml, and Cortisol-5.45 ug/dl). She underwent trans-sphenoidal excision of the lesion via a sublabial, rhinoseptal approach with uneventful post-operative recovery. Conventional light microscopy suggested pituitary adenoma with extensive neuronal metaplasia (Figure

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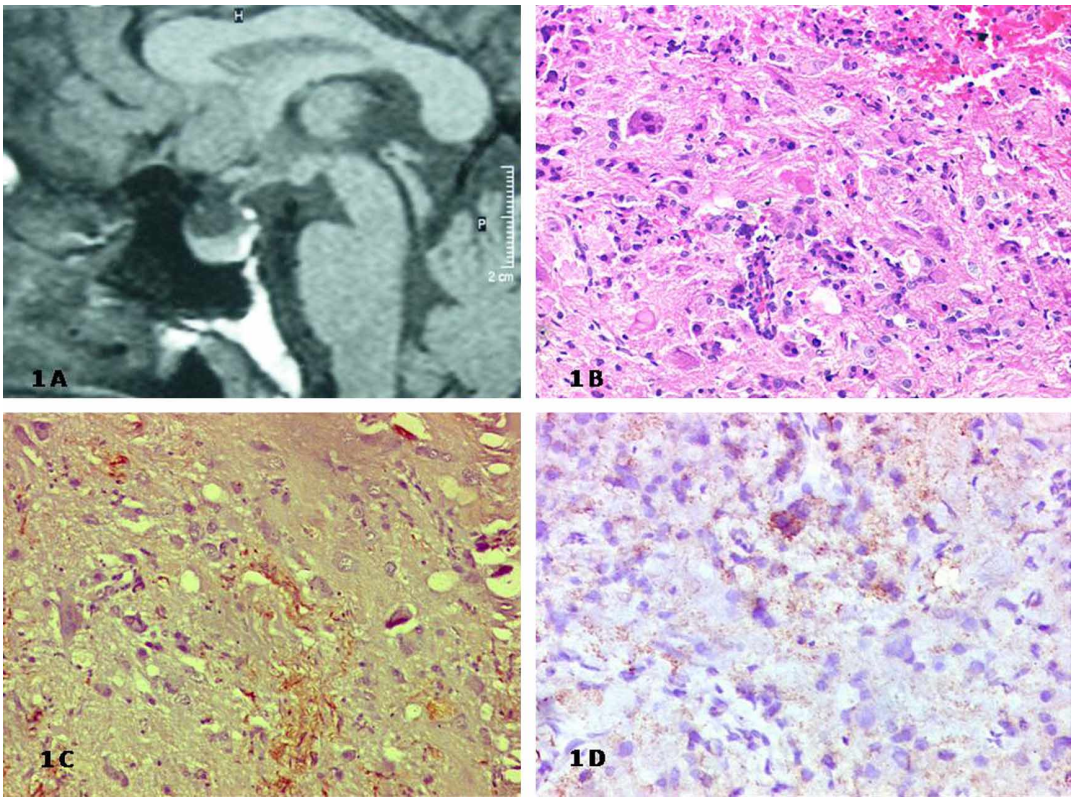

Figure 1. A) Sagittal T1 weighted non-contrast MRI showing hypointense lesion in the pituitary; B) Conventional light microscopy (H & E) of the lesion showing pituitary epithelial cells admixed with bipolar ganglion cells in a glial background (×10); C) Photomicrograph showing synaptophysin positivity D) and background GFAP staining suggestive of glio-neuronal differentiation (×10).

2). On IHC, the tumor had cytokeratin (CK), glial fibrillary acidic protein (GFAP), and synaptophysin positivity highlighting the adenoma component, gliofibrillary background, and metaplastic neuronal cells respectively (Figure 3a,b). The overall picture was suggestive of 'Pituitary Adenoma Neuronal CHoristoma' syndrome. As there was no residual disease on imaging, and the lesion essentially non-secretory, she was kept on surveillance imaging. She had significant symptom relief following surgery and continues to remain asymptomatic on follow-up.

Discussion

The differential diagnoses of pituitary adenoma include a variety of intracranial neoplasms that arise in the sellar-suprasellar region such as pilocytic astrocytoma, craniopharyngioma, ganglioglioma,

Table I. Literature review of glioneuronal differentiation of pituitary tumour (PANCH).

Author [ref]	Year	Patients	Comment
Puchner MJ [5]	1995	4	Chronic over-stimulation by hypothalamic releasing hormones produce hormone secreting pituitary adenomas
Saeger W [2]	1997	5	4 cases of GH secreting and 1 case of adenoma tissue with gangliocytoma
Baysefer A [4]	1997	3	PANCH with Cushing's disease
Sharma MC [6]	1999	2	Review of hypothesis of divergent differentiation
Kurosaki M [7]	2002	6	Ganglion cell lesions with synaptophysin, neurofilament and GHRH positivity. Hypothesized that intrasellar gangliocytoma promoted the growth of the pituitary adenoma
Kontogeorgos G [8]	2006	7	Neurofilament protein in pituitary adenomas suggests a common origin for neuronal and pituitary adenoma cell elements in gangliocytomas
Nasr C [9]	2006	1	Hypothalamic gangliocytomas producing GHRH associated with pituitary adenomas causing acromegaly
Serri O [10]	2008	1	Prolactin secretory adenoma with PANCH

neurocytoma and germ cell tumours [1]. These tumours arise from diverse cellular lineages and have different management principles and prognosis. PANCH is a rare tumour in this region of uncertain cellular origin [2]. Histogenesis of glial and neuronal metaplasia in pituitary adenomas has been debated [3] suggesting that they arise from embryonal pituitary cell rests or have common hypothalamic origin. It is also hypothesized that sparsely granulated growth hormone (GH) producing adenoma cells can differentiate to the neuronal lineage [4]. Morphologically, the tumour is composed of chromophobe pituitary adenoma with varying ganglionic/neuronal component with or without neuropil.

PANCH has been described only as isolated reports or small case series (Table I), with vast majority being hormone secreting pituitary tumours with consequent increase in serum hormone levels [6,7,10]. GH and adrenocorticotropic hormone secreting tumours have a tendency towards glial differentiation [3,5,9]. They usually present with intracellular inclusion bodies like Crooke's hyaline change. Only few non-secretory PANCH syndrome cases have been reported including this one.

As in other pituitary tumours, surgery is the cornerstone of management of these tumours. Completely excised tumours, with no evidence of residual tumour on post-operative imaging, should be kept on close observation. In patients with gross residual disease, or progression on surveillance imaging, not amenable to further safe resection, definitive radiotherapy should be offered to improve outcome.

In summary, non-secretory pituitary adenoma presenting with glioneuronal differentiation (PANCH) is an extremely rare entity with an unknown clinical course.

References

[1] Halbauer J, Mészáros I, Dóczi T, Kajtár P, Pajor L, Kovács K, et al. Rare sellar region tumors. Pathol Oncol Res 2003;9:134–7.

[2] Saeger W, Lüdecke DK, Losa M. Combined neuronal and endocrine tumors of the sellar region. Pathologe 1997;18:419–24.

[3] Horvath E, Kovacs K, Scheithauer BW, Lloyd RV, Smyth HS. Pituitary adenoma with neuronal choristoma (PANCH): Composite lesion or lineage infidelity? Ultrastruct Pathol 1994;18:565–74.

[4] Baysefer A, Gezen F, Kayali H, Erdo(an E, Timurkaynak E, Celasun B. Intrasellar gangliocytoma resembling pituitary adenoma. Minim Invasive Neurosurg 1997;40:107–9.

[5] Puchner MJ, Lüdecke DK, Saeger W, Riedel M, Asa SL. Gangliocytomas of the sellar region–a review. Exp Clin Endocrinol Diabetes 1995;103:129–49.

[6] Sharma MC, Karak AK, Mahapatra AK, Sarkar C. Pituitary adenoma with neuronal choristoma: A report of two rare cases. Clin Neurol Neurosurg 1999;101:128–32.

[7] Kurosaki M, Saeger W, Lüdecke DK. Intrasellar gangliocytomas associated with acromegaly Brain Tumor Pathol 2002;19:63–7.

[8] Kontogeorgos G, Mourouti G, Kyrodimou E, Liapi-Avgeri G, Parasi E. Ganglion cell containing pituitary adenomas: Signs of neuronal differentiation in adenoma cells. Acta Neuropathol 2006;112:21–8.

[9] Nasr C, Mason A, Mayberg M, Staugaitis SM, Asa SL. Acromegaly and somatotroph hyperplasia with adenomatous transformation due to pituitary metastasis of a growth hormone-releasing hormone-secreting pulmonary endocrine carcinoma. J Clin Endocrinol Metab 2006;91:4776–80.

[10] Serri O, Berthelet F, Bélair M, Vallette S, Asa SL. An unusual association of a sellar gangliocytoma with a prolactinoma. Pituitary 2008;11:85–7.

Aggressive primary splenic CD5 positive/Cyclin D1 negative B-cell lymphoma in a patient with chronic hepatitis B virus infection

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To the editor

Primary splenic lymphomas are uncommon and represent less than 1% of all lymphomas. We describe an unusual case of aggressive primary splenic lymphoma in a patient with hepatitis B virus infection with unique morphologic and immunophenotypic

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