of CG of the third ventricle.¹² Ultrastructural study of chordoid gliomas lends support for their glial derivation. The presence of focal basal lamina formation and of microvilli in most cases suggests the possibility of ependymal derivation. The more definitive ultrastructural features of ependymoma, such as cilia and desmosomal junctions are usually lacking.⁴ However, an occasional case with few tumour cells showing isolated abnormal cilia in the vicinity of the nucleus has recently been reported.⁵ In the present case, based on negativity for ependymal features (EMA negativity and lack of microvillous structures on electron microscopy), GFAP positivity and a large number of intermediate filaments on ultrastructure, the possible origin of the tumour is astrocytic.

In summary, the index case is a 5-year-old boy with an unusual extraventricular glioma with extensive myxoid change resembling chordoid glioma, occurring in left parietotemporal region.

R Goyal, R K Vashishta

Department of Pathology, Post Graduate Institute of Medical Education & Research, Chandigarh, India

S Singhi

Department of Paediatrics, Post Graduate Institute of Medical Education & Research, Chandigarh, India

M Gill

Department of Neurosurgery, Post Graduate Institute of Medical Education & Research, Chandigarh, India

Correspondence to: Dr Richa Goyal, Department of Pathology, PGIMER, Chandigarh, India, H. No. 226, Sector 15, Chandigarh-160015, India; gotoricha@yahoo.com

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References

- 1 Brat DJ, Scheithauer BW, Staugaitis SM, et al. Third ventricular chordoid glioma: a distinct clinicopathologic entity. *J Neuropathol Exp Neurol* 1998;**57**:283–90.
- 2 Kurian KM, Summers DM, Statham PFX, et al. Third ventricular chordoid glioma: clinicopathological study of two cases with evidence for a poor clinical outcome despite low grade histological features. *Neuropathol Applied Neurobiol* 2005;**31**:354–61.
- 3 Kleihues P, Louis DN, Scheithauer BW, et al. The WHO classification of tumors of the nervous system. *J Neuropathol Exp Neurol* 2002;**61**:215–25.
- 4 Brat DJ, Scheithauer BW, Cortez SC, et al. Chordoid glioma of the third ventricle. In: Kleihues P, Cavenee WK, eds. *Pathology and genetics: tumors of the nervous system. World Health Organization Classification*. Lyon: International Agency for Research on Cancer, 2000:90–1.
- 5 Pasquier B, Peoc'h M, Morrison AL, et al. Chordoid glioma of the third ventricle. *Am J Surg Pathol* 2002;**26**:1330–42.
- 6 Castellano-Sanchez AA, Schemankewitz E, Mazewski C, et al. Pediatric chordoid glioma with chondroid metaplasia. *Pediatr Dev Pathol* 2001;**4**:564–7.
- 7 Vajtai I, Varga Z, Scheithauer BW, et al. Chordoid glioma of the third ventricle: confirmatory report of a new entity. *Hum Pathol* 1999;**30**:723–6.
- 8 Louis DN, Scheithauer BW, Budka H, et al. Meningiomas. In: Kleihues P, Cavenee WK, eds. *Pathology and genetics: tumors of the nervous system. World Health Organization Classification*. Lyon: International Agency for Research on Cancer, 2000:176–84.
- 9 Nishigaya K, Kaneko M, Ohashi Y, et al. Intracranial retroclival chordoma without bone involvement: no tumour regrowth 5 years after operation. Case report. *J Neurosurg* 1998;**88**:764–8.
- 10 Vaz RM, Pereira JC, Ramos U, et al. Intracranial cervical chordoma without bone involvement. Case report. *J Neurosurg* 1995;**82**:650–3.
- 11 Carstens PEB. Chordoid tumors; a light, electron microscopic, and immunohistochemical study. *Ultrastruct Pathol* 1995;**19**:291–5.
- 12 Reifenberger G, Weber T, Weber RG, et al. Chordoid glioma of the third ventricle: immunohistochemical and molecular genetic characterization of a novel entity. *Brain Pathol* 1999;**9**:617–26.

Metastatic placental site trophoblastic tumour in the duodenum presenting with a gastrointestinal bleed

A 31-year-old woman presented with altered bowel habits, black stools and profound weakness. Abdominal ultrasonography performed elsewhere had shown hepatic metastases. Oesophago-gastroduodenoscopy had been reported as normal. A red blood cell labelled blood pool study had shown a slow bleed at the ileocaecal junction, caecum and/or ascending colon. Except for pallor, her general condition was good. There was no supraclavicular adenopathy or ascites. She underwent lower gastrointestinal endoscopy at our hospital and was detected to have a rectal polyp. Because the size and appearance of the polyp did not correlate with the symptoms, the possibility of a separate pathology was considered. Consequently, the patient underwent an upper gastrointestinal endoscopy, which showed friable ulcerated lesions in the third part of the duodenum. The endoscopic differential diagnosis included carcinoma, lymphoma and tuberculosis.

The rectal lesion was a juvenile polyp. The duodenal biopsy specimen showed ulceration with neutrophilic infiltrates. The lamina propria contained a neoplasm composed of confluent masses of polygonal to round cells with large, vesicular nuclei. Some of the cells possessed smudged nuclei; a few multinucleate giant cells were also present. There were moderate amounts of eosinophilic cytoplasm; some cells contained clear cytoplasm.

Figure 1 Duodenal neoplasm composed of sheets of polygonal to round cells with vesicular/smudged nuclei.

Figure 2 High power view of the tumour cells.

Mitotic figures were scarce. There was no desmoplasia, haemorrhage, necrosis or calcification. The tumour cells expressed cytokeratin (1:50) strongly and diffusely and were negative for CD45 (1:50) and CD30 (1:20) (all antibodies from Dakocytomation, Carpinteria, CA, USA).

Initially, a diagnosis of a high grade undifferentiated carcinoma involving the duodenum was considered. However, the unusual clinical features of a massive gastrointestinal bleed occurring in an otherwise healthy woman and the presence of hepatic metastases without abdominal adenopathy on ultrasonography were contradictory to a histological diagnosis of a high grade epithelial neoplasm. Moreover, there were some unusual morphological features: the bulk of the neoplasm was deep in the lamina propria and there was no dysplasia in the overlying epithelium. Further, the smudged cells raised the possibility of a trophoblastic neoplasm (figs 1 and 2). Further investigation showed a serum β -human chorionic gonadotropin (β -HCG) level of 11 000 mIU/ml. The slides were re-evaluated and a diagnosis of placental site trophoblastic tumour (PSTT) was made. The tumour cells expressed β -HCG focally and human placental lactogen diffusely (both Dakocytomation, Ontario, Canada; predilute, kindly done by Dr R Chetty). The patient was re-interviewed and we learnt that she had had an abortion recently. However, she refused further investigation or treatment and died a month later.

Primary duodenal choriocarcinoma as seen at autopsy and PSTT involving the duodenum and retroperitoneum in a known case of PSTT have been reported earlier.^{1,2} However, we are unaware of any reports of metastatic PSTT presenting primarily as a duodenal lesion. Choriocarcinoma usually shows haemorrhagic areas, with focal expression of human placental lactogen and strong expression of β -HCG in the tumour cells. PSTTs possess sheets of tumour cells with diffuse human placental lactogen and focal β -HCG expression. The moderate rise in serum β -HCG levels in patients with PSTT, as in our case, correlates with the focal expression of β -HCG in the tumour cells.^{3,4} The cells of epithelioid trophoblastic tumour are smaller than those of a PSTT and form cords and nests but no sheets; moreover, epithelioid trophoblastic tumour cells express human placental lactogen only focally. Differentiating between choriocarcinoma and PSTT is of importance because the former are exquisitely chemosensitive. PSTTs are relatively resistant to chemotherapy,

though a few long term remissions to etoposide-methotrexate-actinomycin-D or cisplatin-etoposide treatment have been reported.³

There are many lessons to be learnt from this unfortunate patient. Pathologists must always consider, especially in an unusual clinical setting, the possibility of a trophoblastic neoplasm, a potentially curable disease, before labelling a tumour as a high grade carcinoma or an incurable disease. For physicians, it underscores the fact that sound clinical judgement followed by good communication with the pathologist is the key to a correct diagnosis. Finally, it is a reminder that it is mandatory to take an obstetric history, even in apparently non-obstetric cases.

Sanjay A Pai

Department of Pathology, Manipal Hospital, Bangalore, India

Dinesh Kini

Department of Gastroenterology, Manipal Hospital, Bangalore, India

Ravi P Deo, Poonam U Patil

Department of Oncology, Manipal Hospital, Bangalore, India

Correspondence to: Dr Sanjay A Pai, Department of Pathology, Manipal Hospital, Airport Road, Bangalore 560017, India; sanjayapai@gmail.com

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References

- 1 **Matthews TH**, Heaton GE, Christopherson WM. Primary duodenal choriocarcinoma. *Arch Pathol Lab Med* 1986;**110**:550–2.
- 2 **Ben-Arie A**, Piura B, Biran H, *et al*. Metastatic placental site trophoblastic tumors: clinical aspects of two unique cases. *Acta Obstet Gynecol Scand* 2001;**80**:672–3.
- 3 **Shih IM**, Kurman RJ. The pathology of intermediate trophoblastic tumors and tumor-like lesions. *Int J Gynecol Pathol* 2001;**20**:31–47.
- 4 **Hui R**, Maritza M, Parkash V. Gestational trophoblastic diseases: recent advances in histopathologic diagnosis and related genetic aspects. *Adv Anat Pathol* 2005;**12**:116–25.
- 5 **Behtash N**, Ghaemmaghami F, Hasanzadeh M. Long term remission of metastatic placental site trophoblastic tumor (PSTT: case report and review of literature). *World J Surg Oncol* 2005;**3**:34.

CORRECTIONS

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Figure 4 of an article in the September issue should have been published in colour (Fukuzawa R, Heathcott RW, More HE, *et al*. Sequential *WT1* and *CTNBN1* mutations and alterations of β -catenin localisation in intralobar nephrogenic rests and associated Wilms tumours: two case studies. *J Clin Pathol* 2007;**60**:1013–16). Figure 4 is published in colour on our website at <http://jcp.bmj.com/supplemental>.

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An article in the September issue was published with an incorrect title (Stój A, Rudzki Z, Stachura J, *et al*. The *JAK2* V617F mutation in Philadelphia-negative chronic myeloproliferative disorders. *J Clin Pathol* 2007;**60**:1070–1). The correct title should be: The *JAK2* V617F mutation is frequently present in buccal swabs from patients suffering from Philadelphia-negative chronic myeloproliferative disorders, who carry the mutation detected in bone marrow or peripheral blood cells.