

Pituitary Adenocarcinoma: Report of One Case and Review of Literature

Sui AiXia, Li YiTong, Guo WenChang, Ye ZhanYing, Pang BaoGen, Yang JunJie, Xu YongBing

Sui AiXia, Li YiTong, Yang JunJie, Xu YongBing, Department of Oncology, Hebei General Hospital, Shijiazhuang 050051, China
Guo WenChang, Ye ZhanYing, Pang BaoGen, Department of Neurosurgery, Hebei General Hospital, Shijiazhuang 050051, China

Conflict-of-interest statement: The author(s) declare(s) that there is no conflict of interest regarding the publication of this paper.

Open-Access: This article is an open-access article which was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: <http://creativecommons.org/licenses/by-nc/4.0/>

Correspondence to: Sui AiXia, Department of Oncology, Hebei General Hospital, Shijiazhuang 050051, China.
Email: allen_sui77@sina.com
Telephone: +86-8598-8741

Received: May 14, 2018

Revised: June 5, 2018

Accepted: June 8, 2018

Published online: August 12, 2018

ABSTRACT

Pituitary adenocarcinomas are rare tumors, accounting for only 0.1-0.2% of pituitary tumors. The onset age was mainly between the ages of 30 to 50, and there was no obvious gender difference. There is low morbidity for pituitary adenocarcinoma, but high mortality because high malignancy and extra pituitary metastasis. In this paper, we reported 1 case of pituitary adenocarcinoma, who was diagnosed in Hebei General Hospital.

Key words: Pituitary adenocarcinoma; Sellar; Ki-67; Radiotherapy

© 2018 The Author(s). Published by ACT Publishing Group Ltd. All rights reserved.

Sui AX, Li YT, Guo WC, Ye ZY, Pang BG, Yang JJ, Xu YB. Pituitary Adenocarcinoma: Report of One Case and Review of Literature. *International Journal of Radiology* 2018; **5(1)**: 188-190 Available from: URL: <http://www.ghrnet.org/index.php/ijr/article/view/2336>

CASE REPORT

A 62-year-old woman presented with a 2-week history of intermittent dizziness and headache, who was advised hospitalization at January 15, 2015. There was no amenorrhea, lactation, acromegaly, polyuria and palpitations. There was no obvious abnormality on laboratory evaluations including complete blood count, biochemistry, analysis of tumor markers, pituitary function tests or endocrinology examination. Magnetic resonance imaging (MRI) scan showed an abnormal signal ($2.4 \times 1.9 \times 1.3$ cm) in the sellar with invasion of the right cavernous sinus (Figure 1). Six days later, the patient underwent transsphenoidal craniotomy for tumor resection. The tumor was grey-white, tough and modest blood supply. Since some of tumor tissues have invaded into the right cavernous sinus, subtotal resection was achieved. Histopathologic examination revealed a widely infiltrative neoplasm invading the overlying mucosa and showing a high mitotic activity (Figure 2). There are positive for growth hormone (GH) and prolactin (PRL), but negative for other pituitary hormones. About 70 percent regions are strong positive for p53 and Ki-67. All these data support the diagnosis of pituitary adenocarcinoma. Patient with intermittent dizziness and nausea was admitted again a month later. Intensity modulated radiation therapy was used to radiate the sellar to 56 Gy in 28 fractions and the gross tumor to 66 Gy in 33 fractions with concurrently oral temozolomide (TMZ) 50 mg bid. Two weeks after the radiation, MRI showed tumor size shrank, but metastasized to brain, spinal cord and meninges, resulting in significant compression of the brainstem. She died after a 4-month follow-up.

REVIEW OF LITERATURE AND DISCUSSION

Primary pituitary adenocarcinoma is rare, and only about 100 cases have been reported in recent ten years. Due to low morbidity, the clinical features, diagnosis criteria and treatment protocols of the disease are lack of unified standard. Here, we summarize the literature on pituitary adenocarcinoma.



Figure 1 MRI scan showed an abnormal signal ($2.4 \times 1.9 \times 1.3$ cm) in the sellar with invasion of the right cavernous sinus. a) axial b) sagittal c) coronal.

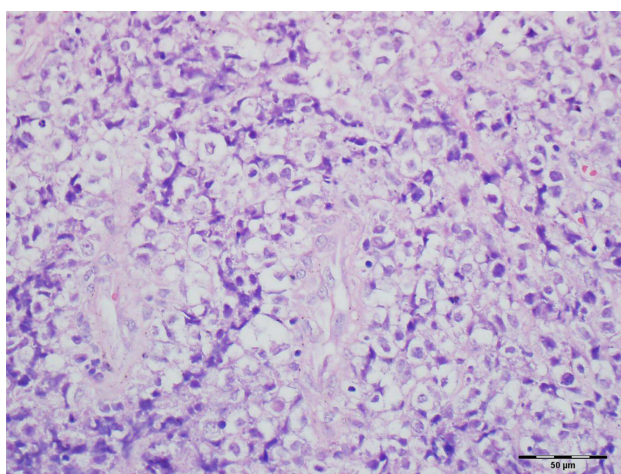


Figure 2 Tumor cells were round or polygonal with atypia, moderate cytoplasm and a high mitotic rate (H & E $\times 400$).

1. Clinical features

The clinical manifestations of pituitary adenocarcinoma are lack of specificity. Common clinical manifestations include limb hypertrophy, amenorrhea and galactorrhea caused by excessive secretion of pituitary hormones. In addition, the patients also suffered from headache, vision loss, visual field disturbance and cranial nerve paralysis due to tumor compression to surrounding nerve tissue^[1]. The corresponding symptoms of metastatic lesions occur when the tumor metastasizes to the brain, spinal cord, meninges, liver, bone, lymph nodes, lung and other parts. In this case, the first symptoms of patient includes intermittent dizziness, headache and diplopia, which is similar manifestations as cerebral infarction.

2. Diagnosis

The diagnosis of pituitary adenocarcinoma requires the support of pathology and imaging. However, early diagnosis of pituitary adenocarcinoma is difficult because of no obvious difference in symptom, imaging or histopathology among pituitary adenocarcinoma, atypical pituitary adenocarcinoma and invasive pituitary adenocarcinoma^[2].

Scheithauer found that the Ki-67 index of invasive pituitary adenocarcinomas is much higher than that of non-invasive ones^[3], so the Ki-67 index was used to evaluate the mitotic and proliferative activity of pituitary adenocarcinoma. Therefore, pituitary adenocarcinomas patients needed to be closely followed when Ki-67 index $> 3\%$ and p53 positive, especially Ki-67 index $> 10\%$. If Ki-67 index $> 20\%$, the patients were recommend the diagnosis of pituitary

adenocarcinoma in situ^[4,5].

In this case, the imaging showed that the patient has pituitary occupies space lesion, so diagnosed as pituitary adenocarcinomas combined with pathological p53 and Ki-67 strongly positive. Subsequent multiple metastases confirmed the accuracy of the diagnosis.

3. Treatment

The treatment of pituitary adenocarcinomas includes surgery, radiotherapy, chemotherapy, hormone therapy, molecular targeted therapy and other methods.

Surgical resection: Surgery is the preferred treatment for pituitary adenocarcinoma, which can provide immediate relief of compressive symptoms and aid definitive diagnosis. However, surgery is difficult to completely remove the tumor because of easy metastasis. So, pituitary adenocarcinoma has the high tendency to relapse after surgery^[6].

Radiation therapy: Radiotherapy is administered to prevent regrowth in subtotaly resected pituitary carcinomas or to slow growth of expanding sellar tumor with no chance of surgery. So, radiotherapy can only improve symptoms, but no decisive effect on the prognosis. The patient of pituitary adenocarcinomas showed a significant reduction of tumor after the radiation, which confirmed the effectiveness of radiotherapy. Although the patient finally died of distant metastasis. It has also been suggested that the radiotherapy did not only lower proliferation index, but also reduce metastasis toward central nervous system^[7].

Chemotherapy: It is now believed that temozolomide (TMZ) should be considered the only drug chemotherapy drug that may control pituitary adenocarcinoma and its metastasis. Nineteen patients with pituitary adenocarcinomas received oral TMZ for 2~24 cycles. Hormonal and tumoral responses were reported in 11/16 (69%) secreting tumors, and tumoral response in 1/3 (33%) non-secreting tumor^[8].

Molecular targeted therapy: Heaney reported the level of pituitary tumor transforming gene (PTTG) expression was lower in normal pituitary than that in pituitary adenocarcinoma^[9]. Immunohistochemical results suggested that there was a positive correlation between PTTG expression and cell proliferation markers such as Ki-67. Further studies demonstrated that PTTG was associated with cell proliferation in mouse transgenic models^[10]. At present, although the mechanism is not fully understood, many experiments in vitro and vivo have confirmed that the expression of PTTG is related to the proliferative status of pituitary adenocarcinoma cells, which provides