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REVIEW ARTICLE

METASTATIC MENINGIOMA IN SUDANESE PATIENTS CLINICAL CASES

*⁴Lamyaa A. Elhassan, ^{2,5}Alsadig Gassoum, ²Sawsan A.H. Aldeaf, ^{1,2}Mohamed A. Arbab and ³Ahmed M. Elhassan

¹Department of Surgery Faculty of Medicine University of Khartoum, Sudan

²National Center for Neurological Sciences, Sudan

³Institute of Endemic Diseases University of Khartoum, Sudan

⁴School of Medicine Ahfaad University for Women, Sudan

⁵Faculty of Medicine Moghtarbeen University, Sudan

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ABSTRACT

Introduction: Meningiomas are usually globular in shape and well-demarcated neoplasms. They have a wide dural attachment and invaginate into the underlying brain tissue without invading it. According to (WHO) classification, there are three grades, grade 1 that are benign, grade II which are intermediate and grade III that are malignant. However, atypical (WHO grade II) and anaplastic (WHO grade III) meningiomas show a more aggressive biological behavior with a high risk of local recurrence and a less favorable prognosis.

Case 1: A 28 years young man, a farmer, was presented to our referral clinic with history of 4 months duration of headache, generalize convulsions that associated with loss of consciousness.

Case 2: 17 years old female child, an other wise healthy before, presented to neurosurgery clinic with history of slowly progressing headache and Rt scalp swelling for one year duration. Neurological examination is unremarkable.

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INTRODUCTION

Meningiomas are usually globular in shape and well-demarcated neoplasms. They have a wide dural attachment and invaginate into the underlying brain tissue without invading it. According to (WHO) classification, there are three grades, grade 1 that are benign, grade II which are intermediate and grade III that are malignant. However, atypical (WHO grade II) and anaplastic (WHO grade III) meningiomas show a more aggressive biological behavior with a high risk of local recurrence and a less favorable prognosis (New, Aronow, and Hesselink 665-75). In most cases, meningiomas can give stereotypic symptoms in specific locations; however, these stereotypic symptoms are not pathognomonic for meningiomas. Patients with meningioma may present with vascular manifestations such as stroke or transient ischemic attack, although, this presentation is very rare still it should be considered (Bouchez *et al.*, 251-62). Much has been written on the pathology of the meningiomas, and especially on their occasional malignancy. Very few contemporary surgeons have given the incidence of recurrence in their material. (Hoessly and OLIVECRONA 614-26), Hoessly thought that with his parasagittal meningiomas the recurrence rate was under 10%; (GRANT 25-35) reported 13 delayed deaths due to tumor

growth in 219 cases, all after “incomplete removals . This is a crucial distinction. It is generally agreed that some meningiomas are clinically malignant, as witnessed by rapid growth, invasive tendency, and, rarely, extra cranial metastasis.

Case 1

A 28 years young man, a farmer, was presented to our referral clinic with history of 4 months duration of headache, generalize convulsions that associated with loss of consciousness. Few weeks before his presentation, the patient developed blurring of vision and decrease visual acuity that end in bilateral blindness. MRI brain was done and showed huge Lt parieto occipital mass with thick irregular wall and associated edema effacing the adjacent lateral ventricle, (Case 1). Patient underwent surgical resection, intraoperative; we encountered highly vascular tumor, invading bone and underlain dura. Sub total excision of the tumor was performed on the part over the transverse sinus left behind. Microscopically the tumor composed of spindle shaped cells with plump nuclei and high nucleus cytoplasmic ratio. There were 6 mitosis/ 10 power fields, tumor cells were positive for Epithelial membrane antigen (EMA) and progesterone receptor. Immunostaining of Ki 67 antigen and p53 protein, showed labeling index of 11-15%, Fig. (2, 3, 4, 5, 6). Patient discharged after 10 days post operative in a good condition. The patient was given fractionation radiotherapy.

*Corresponding author: Elhassan

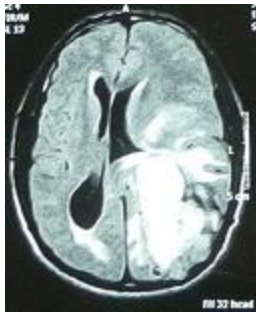
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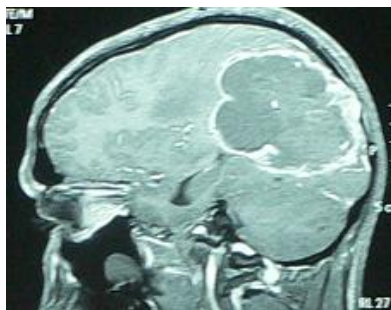
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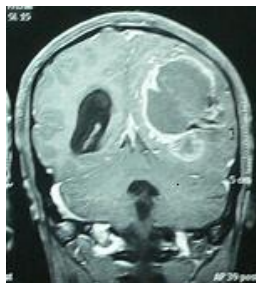
B



C



D



E

Case. 1: (A,B, and C), Axial MRI sequencing T₁, T₂ and FLAIR showed large Lt parieto occipital Lobe lesion, heterogeneous in T₁, hyper intense in T₂ with effacement of the adjacent ventricular system and finger like projection edema (D and E) sagittal and coronal MRI sequence T₁ with contrast respectively, showed irregular capsular enhancement



Fig.2. showed ki 67 labeling index 11-15% in atypical metastatic meningioma

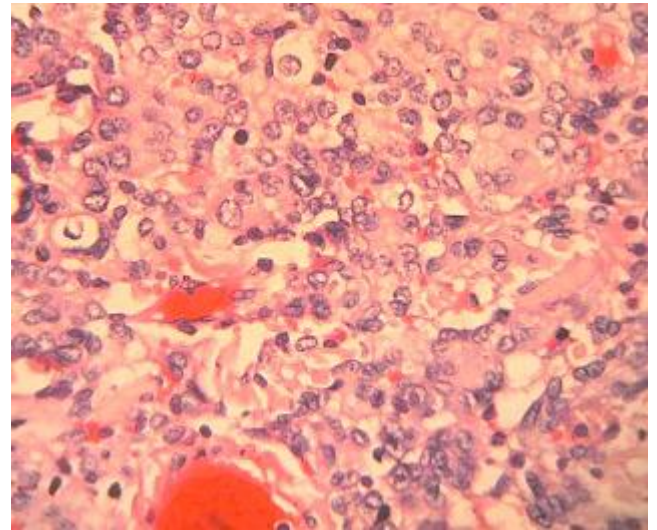


Fig.3. showed H&E of atypical meningioma

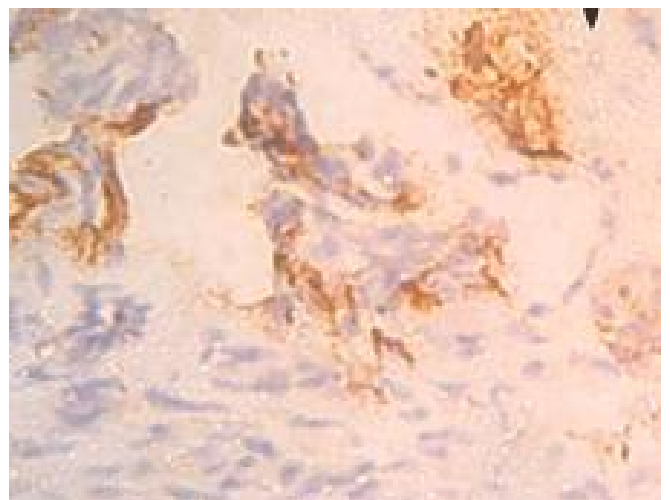


Fig.4 showed positive EMA in meningioma

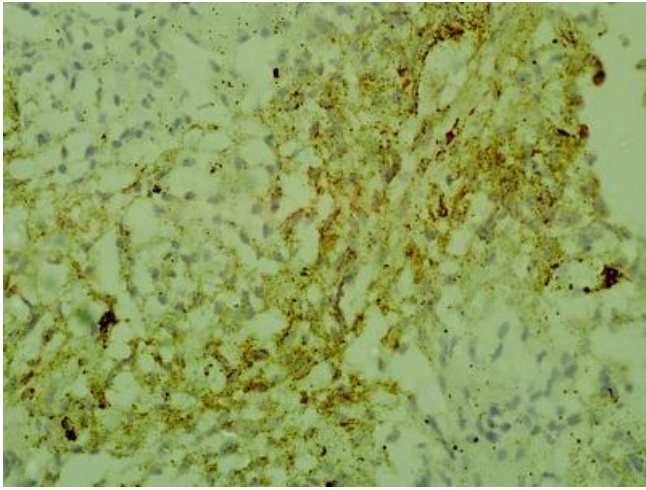


Fig. 5. showed p53 labeling index 11-15% in meningioma

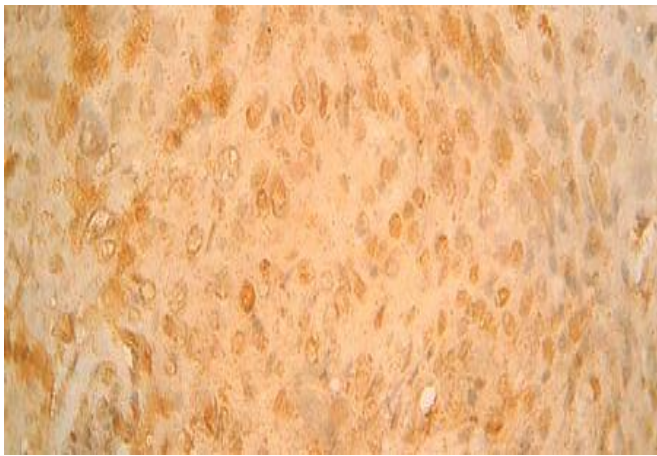
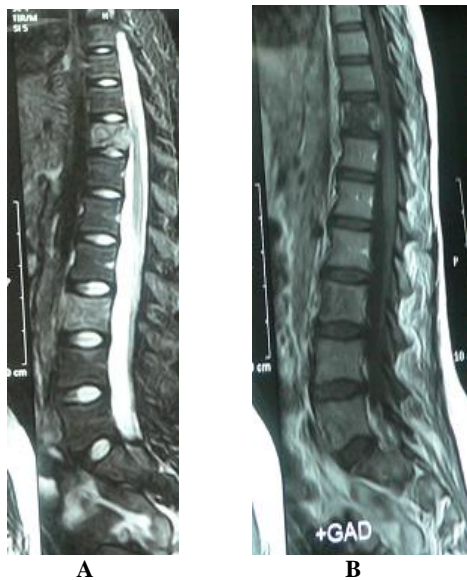


Fig. 6. showed p53 labeling index 16-20% in meningioma



Case 2. (A and B) MRI examination showed T₂ and T₁ with contrast respectively, there is significant changes involving the vertebra bodies D₁₁, L₃ and S₁, it is representing as low signal in T₁ and heterogeneous enhancement after contrast, it is high signal in T₂, there is some posterior expansion of D₁₁ causing spinal cord compression

Few months after, the patient was presented again with symptoms suggested of local recurrence, with features of Lt occipital scalp swelling, headache, vomiting and Lt Lateral cervical swelling. Local examination of cervical swelling, showed 5x3 cm mass, attached to under line tissue, and located in the posterior triangle of the neck. MRI brain showed recurrence of the tumor. The patient underwent surgical debulking to minimize the intracranial symptoms and elevated intracranial pressure, with excision of the cervical swelling. Microscopically, the occipital lesion showed same histology of the first one, but the tumor from the cervical showed, meningiothelial and spindle cells with dark nuclei, and no lymphoid tissue was seen. Macroscopic and microscopic examinations revealed that, the aforementioned mass was consisted of transitional meningioma. Patient discharge in a good condition and was planed for brain radiotherapy and chemotherapy but he died at home before receiving any palliative treatment.

Case 2

17 years old female child, an other wise healthy before, presented to neurosurgery clinic with history of slowly progressing headache and Rt scalp swelling for one year duration. Neurological examination is unremarkable. MRI brain was done and revealed, Rt occipital well enhance lesion with extra cranial extension. Patient underwent surgical excision, and the tumor was totally removed. Patient discharged 10 days post operative in a good condition. Microscopically the tumor was composed of whorls and bundle of spindle shaped cells with dark nuclei exhibiting scattered mitosis. The tumor is cellular in places where the cells are rounded with small dark nuclei and scanty cytoplasm, cell were positive for Epithelial membrane antigen (EMA) and progesterone receptor. Ki 67 and p53 protein immunostaining was done, labeling index 11-15%, and 16-20%, was identified within the positive cell nuclei of Ki 67 antigen, and P53 protein respectively. The diagnosis of atypical meningioma, was based on increase cellularity, mitosis and abnormal nuclear cytoplasm ratio. Patient was given 20 sessions of radiotherapy and after which she retained back to school. 6 months later she presented with history of headache, vomiting, convulsions. MRI brain, showed recurrence on the previous site of surgery. Patient underwent surgical excision and the microscopic examination showed features of atypical meningioma. Patient was received radiotherapy as palliative care. few weeks later, she presented at the emergency unit, with history of nocturnal back pain, lower limbs pain and unsteady gait, there is defective sphincter control, and impair level of consciousness. Clinical examination showed an ill patient, lower limbs examination revealed hypertonia and exaggerated reflexes, power grade 3. There is huge occipital swelling. CT brain, showed Huge extra cranial tumor fumingating through the previous route of surgery with intracranial extension. MRI dorsal and lumbosacral spine were done and revealed changes involving multiple vertebral bodies D₁₁ and L₃, there is expansion of D₁₁ causing compression at this level (Case 2). Biopsy was taken from the spine tumor, and histopathology showed meningiothelial cells. Patient was planed for whole brain radiotherapy and chemotherapy but she died at hospital without receiving any palliative care.

DISCUSSION

Meningioma is one of the most frequently encountered tumors of the central nervous system that arise from arachnoid cap cells. They form a heterogeneous group of mostly benign tumors with slow growth, however malignant meningioma shows local invasion, involving the venous sinuses, dura mater, skull scalp and brain (Fukushima *et al.*, 517-24). Local recurrence rate of meningioma even after complete resection vary from 9-32% (Fukushima *et al.*, 517-24). Atypical meningiomas have increase mitotic figure, with the 3 of the following :increase cellularity, small cell with high nucleous cytoplasm ratio, prominent nucleoli, sheet like growth pattern and focal necrosis. (Perry *et al.*, 2046-56) The mitotic figure in atypical meningioma has been define as 4 or more per high power field, the above criteria have been shown to correlate with the recurrence. Taking the radiographic indicators for meningioma aggressiveness, such as Mushrooming, non homogenous enhancement, peri tumoral edma, cyst like, indistinct tumor brain border, all these have been proposed as a clue about the malignancy and aggressive behavior (Perry *et al.*, 2046-56) In our cases the MRI showed aggressive characteristic indicators which are, multilobulated mushrooming like lesion, no obvious dura base with feature of intra axial lesion (GBM) and finger like projection edema. High labeling index of Ki 67 antigen and p53 protein was associated with the present cases. Metastatic meningioma was very rare with an incidence of 1 in 1,000 meningiomas, lung was the most frequent site for metastasis of meningiomas (3, 4). (Enam *et al.*, 1172-77). However the reported metastatic rate of atypical meningiomas was 5%. In our cases metastatic meningiomas were constituted 3.9%. Histopathological diagnosis of metastatic meningioma is difficult (Baisden, Hamper, and Ali 291-94). The most common sites of metastases of meningiomas are the lungs (60%), followed by the abdomen and liver (34%), cervical lymph nodes (18%), long bones, pelvis and skull (11%), pleura (9%), vertebrae (7%), CNS (7%), and mediastinum (5%) (Kepes *et al.*, 312-16). In the present of metastasis, the survival rate is poor with the median survival rate less than 2 years after primary tumor diagnosis. More over, there is quite variable latency period between the diagnosis of intracranial meningioma and the metastasis, (Miller *et al.*, 763-66). The lesion usually exhibit multiple local recurrence before it metastasized and this what had been observed in our two patients. Meningioma may disseminated through haematogeneous, ymphatics or cerebro spinal fluid (New *et al.*, 267-76), Some other mode of metastasis spread the passage through the venous channels and through the vertebral venous system, the tumor may reach the spinal cord and vertebral body and this may suggest metastasis in our patients.

In the period of seven years we operated 405 meningioma patients, grade 1 meningioma constituted 77.3%, grade 11 costituted 18.6% and grade 111 constituted 4.1%, atypical meningioma constituted 12.6%, there was only two metastatic tumor of atypical variant. In the present cases, the anatomical site of metastasis is usual but with bad prognosis, both patients were died shortly after diagnosis of metastasis.

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