Metastatic basal cell carcinoma of the spine

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A rather progressive case of basal cell carcinoma of the left zygoma which subsequently metastasized to spine and brain resulting in mortality is presented. Surgical management of spinal metastases should be prompt and radical.

Introduction

Clinically, basal cell carcinomas range from erythematous skin tan or plaque to smooth or ulcerative papules, nodules, subcutaneous nodules or deep ulcerations. The behaviour of the tumour is variable, the majority following a rather slow progressive course, a few progressing to mutilation, and fewer still to metastasis.

Basal Cell Carcinoma (BCC) is generally speaking very rare in Blacks. It is, however, seen in albinos and Caucasians that live in the West African sub region. This report highlights the importance of suspicion of the lesion and at the same time adds to the number of cases so far reported in the literature.

Case Report

Mrs AK, University College Hospital, Ibadan, No. 388797, a 44 year old Russian lady, was first seen in the Plastic Surgery Unit of University College Hospital in 1986 when she presented with a left zygomatic nodular lesion which on excision and local flap cover was confirmed to be a BCC. Shortly after this initial excision, she developed a recurrence in the same area. This was excised and a temporal fascial flap based on the middle temporal artery branch of the superficial temporal was transferred and covered with a skin graft.

The area was subsequently irradiated. She, however, developed progressive trismus and lower motor neurone facial nerve palsy on the left side as well as telangiectatic local skin changes. In 1992, an ulcerative skin change was noticed again in the left zygoma.

The area was radically excised and the ipsilateral temporalis muscle based on a terminal branch of the internal maxillary artery was transposed and covered by a split skin graft. Histology confirmed morphea type basal cell carcinoma.

Around the time of surgery, she experienced sharp pain radiating from the back to anterior abdomen on the right side. X-ray investigation revealed a collapsed LI vertebra. A diagnosis of spinal tuberculosis (TB) was entertained particularly as she had had contact with a tuberculous patient previously. She was then started on anti TB treatment (rifampicin and INH) pending further results of investigation to confirm TB.

Her lower limbs progressed to paralysis and at neurosurgical review, an impression of metastatic spinal basal cell carcinoma was made. She had a surgical exploration by posterior approach and debulking of the metastasis. She, however, died a month post operatively from cerebral metastasis.

Discussion

Basal cell carcinoma (BCC) orginates in the pluripotential epithelial cells of the epidermis and hair follicles. It occurs almost exclusively in Caucasians, the typical patient being fair haired, fair skinned and blue eyed, who spends a great deal of time out doors and sunburns easily. "Few cases of BCCs reported in Black patients[1] as well as BCCs that develop in non exposed areas in Whites my represent a different pathogenesis from BCC caused by ultraviolet light exposure. This may possibly be immune in nature. Habets *et al*[2] suggested that the immune response to the presence of skin cancer is primarily T cell mediated, and that T cells play a major role in the body's defence against the proliferation of BCC.

The commonest histologic type of BCC is the nodular, followed by the superfical types. The morphea type as in the case being reported though uncommon has the ability to synthesize type 4 collagenase which enhances its aggressive behaviour.

Robins and Albiom[3] noted that BCCs are more likely to recur in young women, a fact attributed to the conservative margin taken as an attempt to give these patients a good cosmetic result. Approximately one third of all incompletely excised BCCs will recur. Those in certain anatomic sites such as periorbital and perinasal regions often recur even after adequate treatment[4]

The hypothesis that tumour proximity to major lymphatic systems or large calibre blood vessels may be of significance in consideration of metastatic BCC is supported by this case. Hence massive tumour size, ulceration, or history of multiple recurrences may not be absolute prerequisites.[5]

About 300 cases of metastatic BCC have been documented in the literature. This constitutes an estimated incidence of 0,1pc[6]. When it metastasizes, it is usually to lymph nodes, lungs and bones. This case, however, constitutes the second report in literature of cord compression due to a metastatic BCC.

Of all the modalities of treatment which include electrodesiccation with curettage,[7] cryosurgery, intralesional interferon injection,[8] radiotherapy, photodynamic therapy,[9] and surgical excision; surgical excision is the most effective for large primary lesions which as in this case may require reconstruction of a large defect after radical excision.

Metastatic BCC is more difficult to treat. When it is discovered, the average survival is only eight to 10 months. Surgical treatment of spinal metastases should be radical. Immediate surgical decompression and stabilization should be indicated

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before complete paralysis develops. Once complete paralysis has set in, extensive lesions should be treated by a combined anterior and posterior approach.[11]

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Myasthenia gravis (MG): a preliminary report

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Eighteen cases of MG admitted to the Neurology Unit, 7th April Hospital, Benghazi, Libya, over a period of three years October 1991 to December 1994 were reviewed. The female to male ratio was 2,6:1 (13 females and five males). The mean age of presentation was 13,3 years later for male patients compared to females (mean age of presentation in females was 26,5 years and in males 39,8 years). The average time interval between the onset of symptoms and diagnosis was 2,5 years.

At the time of diagnosis 94,5pc (17 cases) of the cases had generalized MG and 5,5pc (one case) had ocular symptoms only. In 11,1pc (two cases) of patients an association with thyroid disorder was observed. Repetitive nerve stimulation (RNS) test was abnormal in 83pc (15 cases) of our cases.

All cases were initially treated with anticholinesterase and 22,2pc (four cases) also additionally required steroid therapy. Thymectomy was performed on eight cases, four of which had thymus hyperplasia. None of our cases had any thymoma.

Of these eight cases, one case (12,5pc) had complete remission, five cases (62,5pc) were doing well with a reduced dose of anticholinesterase and ± steroids. However, two cases (25pc) required intermittent plasmapharesis and immunosuppressants in addition to anticolinesterase and steroids. These patients are obviously being followed up for long term outcome.

Introduction

MG was first described by Willis in 1672 and the clinical picture was well recognized by the end of the 19th century. Further landmarks were Mary Walker's observation in 1934 of improvement with anticholinesterase, Blalock's reported intial success with thymectomy in 1940 and the role of receptor antibodies by Patrick and Lindstrom in 1973.[1,2] It is an organ specific auto-immune disease, caused by an antibody mediated assault on the acetylcholine receptors (AchR) at the neuromuscular junction. Binding of these antibodies to AChR leads to a reduction in the number of active receptors brought about either by functional block of the receptors; by increased rate of degradation or by complement mediated lysis resulting in impaired neuromuscular transmission.[3,4]

Although it is an auto-immune disease, the heterogeneity of the disorder argues against a single etiologic factor. The thymus is abnormal in most patients and must play a role in the pathogenesis. It may occur at any age and its prevalence is variably estimated at five to nine per 100 000 population.[5] It is characterized by weakness of skeletal muscles, which increases with muscle activity. The current treatment of MG includes anticholinesterase, steroids, thymectomy, immunosuppressants and plasma exchange.

The present paper is the first ever study of MG reported from Libya, and reviews the clinical features, therapeutic aspects and our experience with a small number of cases (18 cases).

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Materials and Methods

Eighteen cases of MG admitted to the Neurology Unit, 7th April Hospital, Benghazi, Libya over a period of three years (October 1991 to December 1994) were reviewed. The data was taken