

Malignant Mixed Tumour of Cutaneous Origin – a Rare Tumour Arising from Sweat Glands

Shanu Srivastava*, IM Vora**, Kanchanmala G Ghorpade***,
Sangeeta B Kulkarni+

Abstract

Cutaneous 'Malignant mixed tumour' is a rare neoplasm. No pathognomonic signs permit its clinical diagnosis and it is usually diagnosed as cutaneous cyst. A 50 year male presented with tumour over right thigh since many years with a sudden increase in size since the past 2 months. The tumour was in subcutaneous tissue with no fixity to the underlying bone. The tumour was excised. Grossly it was 10 x 8 x 5 cm, firm, nodular and partially encapsulated. It cut with a gritty sensation. The cut surface showed a variegated appearance with opaque grey white and cystic areas. Microscopically, tubular glands resembling apocrine glands, solid nests of epithelial cells with atypia and a chondromyxoid stroma with focal calcification and ossification were seen. S100 was strongly and diffusely positive. Cytokeratin, calponin and SMA were focally positive. EMA was positive in the epithelial component and P63 was negative. The final diagnosis was 'malignant mixed tumour' of skin arising from sweat glands.

Introduction

Malignant mixed tumour of cutaneous origin is a rare neoplasm. It may arise from long standing benign lesion. No pathognomonic signs permit its clinical diagnosis and it is usually diagnosed as a sebaceous cyst.¹ The tumour arises in the dermis or superficial subcutaneous tissue. The usual sites are face and extremities. Microscopically, it has two components - an epithelial and a mesenchymal component. The epithelial component is seen as glands or as sheets of epithelial cells. The glands may show either apocrine or eccrine differentiation. The features suggestive of malignancy are seen in the epithelial component in the form of cytological atypia, increased mitotic rate, necrosis and invasion.¹ The stromal component on the other hand is

benign and myxomatous with cartilaginous differentiation.

Case Report

A 50 year male presented with tumour over right thigh since many years. There was a sudden increase in size since the past 2 months. Physical examination showed a large mass in the subcutaneous tissue, which was fixed to the skin but not to the underlying bone. No lymphadenopathy was present. A 10 x 8 x 5 cm tumour was excised along with an elliptical flap of skin.

Pathological findings

Grossly the tumour was partially encapsulated and nodular. It cut with a gritty sensation. The cut surface was variegated with opaque grey white and cystic areas. The consistency was firm (Fig. 1).

Microscopic examination disclosed a biphasic pattern. There were solid sheets of polygonal cells with homogeneous and eosinophilic cytoplasm and a centrally placed nucleus. The cell membranes of individual cells were distinctly seen. The cells showed cellular atypia in the form of hyperchromasia, cellular pleomorphism and a mitotic count of 1 to 2 mitosis per high power field. The outermost cells of these nests of polygonal cells became progressively less

*Lecturer; **Professor; ***Professor and Head;
+Associate Professor; Department of Pathology, Terna Medical College, Nerul, Navi Mumbai.



Fig.1 : Tumour of size 10 x 3 x 5 cm. External surface is nodular. Cut surface is variegated with opaque grey-white and cystic areas.

cohesive as they merged with chondroid matrix. Also seen were tubular branching glands lined by double layer of cells (Fig. 2). Decapitation secretion could not be seen. The stroma appeared benign. It was chondromyxoid with focal calcification and ossification. At places pseudocartilaginous areas were present.

Immunohistochemistry revealed strong and diffuse positivity with S100. Cytokeratin, calponin and smooth muscle actin were focally positive.

Epithelial membrane antigen was positive in the epithelial component (Fig. 2) while P63 was negative. This concludes that the tumour has epithelial, myoepithelial as well as mesenchymal components with low proliferative activity. The final diagnosis was 'malignant mixed tumour' of cutaneous origin arising from the sweat glands.

Discussion

Mixed tumour (Pleomorphic adenoma) are well characterized in salivary glands, however they are very rare at other sites like soft tissue and skin. Most of these tumours arise in the extremities. Grossly they are well circumscribed and lobulated.² Microscopically malignant mixed tumour must be differentiated from Extra Skeletal Mesenchymal Chondrosarcoma (ESMC). In ESMC, the cartilaginous component in the stroma is usually benign but some may show low-grade chondrosarcoma. The stroma of malignant mesenchymal tumour is however

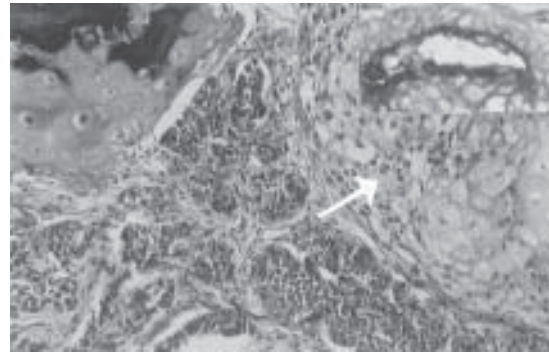


Fig. 2 : Biphasic tumour composed of epithelial cells arranged in sheets and tubules lined by double layer of cells and stroma which is chondromyxoid with focal calcification. Pseudocartilaginous areas are also seen (arrow). HE X400 Inset shows EMA positivity in epithelial component.

always benign. Another differentiating point is seen in the cells surrounding the cartilaginous stroma which are primitive looking in ESMC. Immunopositivity for epithelial or myoepithelial components (EMA, calponin, SMA and cytokeratin) suggest malignant mixed tumour.^{3,4} S100 positivity however is seen in both these tumours. Hassab, Naby *et al* found that the polygonal cells resembling epithelial cells in the stroma of cutaneous mixed tumour stain for S100 protein and keratin, but are negative for actin favouring the hypothesis that they are epithelial rather than myoepithelial.⁵ However in our case they showed positivity with actin, which was similar to the study of Iglesias *et al* suggesting a myoepithelial nature of the cells.⁶ Eccrine differentiation is suggested by a single layer of cells lining the glands. Apocrine differentiation is suggested by double layer of cells lining tubular branching glands with decapitation secretion of the inner layer.³

However Requena *et al* found that tubular branching pattern in cutaneous mixed tumours is considered an expression of

apocrine differentiation even in the absence of decapitation secretion.³ Immunostains do not help in differentiation between apocrine and eccrine types.⁵ In our case decapitation secretion was not seen. Features suggesting malignancy in a mixed tumour are increased cellularity with, numerous or even focal mitosis, which may be seen only focally, cellular atypia, necrosis and invasion. The invasion can be lymphatic, vascular or in the surrounding subcutaneous tissue.¹

References

1. Harrist TJ, Aretz TH, Mihon MC, *et al.* Cutaneous malignant mixed tumour. *Arch Dermatol* 1981; 117 : 719- 24.
2. Jason L, Hornick MD, Christopher DM Fletcher. Myoepithelial tumours of soft tissue. *Am J Surg Pathol* 2003; 27 : 1183-96.
3. Luis Requena, Evaristo Sanchez Yus, Danial J, Santa Cruz. Apocrine type of cutaneous mixed tumour with follicular and sebaceous differentiation. *Am J Dermatopathol* 1992; 14 (3) : 186-94.
4. Banerjee SS, Harris M, Eyden BP, *et al.* Chondroid syringoma with hyaline cell change. *Histopathology* 1993; 22 : 235-45.
5. Hussien M. Hassab- EL- Naby MD, Sam Tam MS, HT (ASCP), *et al.* Mixed tumours of the skin. *Am J Dermatopathol* 1989; 11 (5) : 413-28.
6. Iglesias FD, Forcelledo FF, Sanchez TS, *et al.* Chondroid syringoma. A histochemical study of 15 cases. *Histopathology* 1990; 17 : 311-8.

A PEACE-FUL SOLUTION TO COPD EXACERBATION

Treatment with long-acting inhaled anticholinergic agents of β agonists, alone or with inhaled corticosteroids, improves lung function.

Exacerbations are a particular problem in COPD, because they provoke distress in patients, invoke health-care costs, and have high mortality rates in hospitalized patients. All the treatments decrease the risk of exacerbation, as do influenza and pneumococcal vaccination.

1500 mg carbocisteine daily was accompanied by a reduction in mean exacerbation frequency from 1.35 to 1.01 events per year.

However, some issues need clarification. We know little about the causes of exacerbation in this population and especially whether carbocisteine selectively prevented specific types of events, as has been suggested for other treatments.

Paul Albert, Peter Calverley, The Lancet, 2008; 371 : 1975-76.