CD10 for the distinct differential diagnosis of basal cell carcinoma and benign tumours of cutaneous appendages originating from hair follicle

Demet Sengul[1], Ilker Sengul[2], Muzeyyen Hesna Astarci[3], Huseyin Ustun[3], Gamze Mocan[4]

[1]Department of Pathology, Prof. Dr. A. Ilhan Ozdemir State Hospital, Giresun, Turkey
[2]Department of General Surgery, Giresun University Faculty of Medicine, Giresun, Turkey
[3]Department of Pathology, Ankara Education and Research Hospital, Ankara, Turkey
[4]Division of Cytology, Department of Pathology, Hacettepe University Faculty of Medicine, Ankara, Turkey

Aims: Differential diagnosis between the group of trichoadenoma, trichofolliculoma, trichoepithelioma, trichoblastoma and basal cell carcinoma has been creating some difficulties for the pathologist and the clinicians, particularly in the presence of small specimens.
Material and methods: A total of 30 cases of benign tumours of cutaneous appendages originating from the hair follicle and 30 cases of basal cell carcinoma were retrieved from the archives deposited from 2004 to 2008.
Results: The expression of CD10 in both tumours was graded from [0] to [2+] for each case. The immunoreactivity of CD10 was comparatively examined among the groups and each subgroup. The stromal CD10 immunopositivity of benign tumours of cutaneous appendages originating from the hair follicle was stronger than the other ($p = 0.003$) regarding both the numerical and the degree of expression. However, peripheral CD10 of basal cell carcinoma was stronger than the other for [1+] immunopositivity ($p = 0.03$). It was exact opposite for [2+] ($p = 0.013$). Besides, central CD10 immunopositivity and CD10 reactivity for the subgroups was not significant.
Conclusions: CD10 may be very useful for the differential diagnosis between them particularly in the small and superficial biopsies and it may be even a life-saving method in some selected cases.

Key words: hair follicle, immunohistochemistry, differentiation, cutaneous adnexal neoplasms.

Introduction

Firstly Headington classified benign tumours of cutaneous appendages originating from the hair follicle (BTCOHF) as germ layer hamartomas of the hair follicle, tumours of the hair follicle originating from the germ layer, tumours originating from the external layer of the hair follicle (trichilemmoma) and tumours originating from perifollicular mesenchyme in 1976 [1]. Ackerman *et al.* [2, 3] criticised those that were included in eight textbooks of dermatopathology in 2001. Recently, the World Health Organization (WHO) classified BTCOHF as two main subgroups: benign and malignant ones in the year of 2003 [1].

Differential diagnosis between BTCOHF such as trichoepithelioma (TE), trichoblastoma (TB), trichofolliculoma (TF), trichoadenoma (TA) and basal cell carcinoma (BCC) may be very difficult and bothersome for the clinician and the pathologist. The characteristic histopathology of TE is multiple horn cysts including concentrically laminated keratin or a less structured hyaline material. But the histological features of slowly growing, fresh-coloured, soli-

tary papules, commonly reticulated cyst in the dermis surrounded by a mantle of cells or partly nodules usually located on the face which are very similar in both TE and BCC. Histological texture of both lesions consists of nests of basaloid cells inside the dermis [4, 5]. Trichoepithelioma and TB involve specialized tumour-associated stroma separating the nodules from the tumour and segregating the tumour epithelium from the enclosing dermis. Inversely, a specialized mesenchymal component is deficient for BCC. Besides, grooving of tumour epithelium and reticular dermis is present for that malignant tumour [6]. Trichofolliculoma is a rare tumour in any part of the skin [4] and a follicularly differentiated hamartoma mostly appearing during adulthood [7] without any sex predilection [8, 9]. Trichoadenoma is also a rare tumour, first determined in 1958 by Nikolowski [10] and seen as a nodular lesion usually on the face and buttocks [11, 12].

Basal cell carcinoma accounts for 65-75% of all skin tumours [13] and is the most common malignancy worldwide in white people [14]. The keratinous pattern of BCC resembles external stem sheath of the hair follicle rather than epidermis. The tumour may also have a follicular origin [15, 16]. Basal cell carcinoma is a low grade malignant tumour [17] and rarely metastasizes having the incidence of 0.0028% – 0.55% [18, 19]. Its recurrence for 5-year survival is 5% [19] and may show local invasion, especially the infiltrative and morpheic type [20]. So, it requires total excision and follow-up [5].

CD10 is a 100 kDa type II cell surface zinc metalloproteinase that is known as neutral endopeptidase, enkephalinase and common acute lymphoblastic leukaemia antigen (CALLA) which is included in inactivation of various biologically active peptides [21, 22]. Firstly it is determined as CALLA antigen and expressed on neoplastic cells such as Burkitt's and follicular lymphomas, lymphoblastic cell and chronic myelocytic leukaemias. It is also expressed on the surface of a wide variety of normal cells, such as fibroblasts, brush border of renal epithelial cells and enterocytes, glomeruli, myoepithelial cells of breast, bile canaliculi, hair follicles, eccrine and sebaceous glands [21, 23, 24]. For the skin, dermatofibromas, dermatofibrosarcoma protuberans, and melanomas as well as tumours and periadnexal mesenchymal cells in normal dermis are detected as the immunoreactivity areas of CD10 [25, 26].

CD10 focally stains papillary mesenchymal body, peritumoral stroma, and peripherally palisading cells. However, CD10 diffusely stains basaloid epithelium but not peritumoral stroma in BCC [5, 27, 28].

In the differential diagnosis of BTCOHF and BCC, histopathological character may not be enough and may be troublesome for pathologists, particularly in small biopsy specimens. So, immunohistochemical techniques can be useful for distinct diagnosis [29].

For these reasons, we compared staining patterns of BTCOHF (TE, TB, TF, TA) and BCC by using an immunohistochemical marker of CD10 in the current study which is the first one in literature to our knowledge as a group of BTCOHF.

Material and methods

Case selection

The investigation conforms to the principles outlined in the appropriate version of 1964 Declaration of Helsinki and approval of the present study was received from the Ethics Committee of Ankara Education and Research Hospital.

A total of 30 cases of BTCOHF (21 TE [Group 1a], 70%; 5 TB [Group 1b], 16.6%; 2 TA [Group 1c], 6.7%; 2 TF [Group 1d], 6.7%) and 30 cases of BCC were retrieved and analysed from the archives of the Department of Pathology at Ankara Education and Research Hospital where the cases of BTCOHF had been deposited between 2004 and 2008. The punch biopsies and incision biopsies, not enclosing the neighbouring epidermis and dermis were not included in the study.

Basal cell carcinomas were classified as nodular (18 cases, Group 2a, 60%), superficial (4 cases, Group 2b, 13.3%), infiltrative (2 cases, Group 2c, 6.7%) and mixed (6 cases, Group 2d, 20%) containing two or more types together. The staining patterns of CD10 are comparatively evaluated between both groups of BTCOHF and BCC totally and between all subgroups which belong to the same main group.

Immunohistochemistry

For the immunohistochemical evaluation, formalin (10% solution; pH 7.0-7.6) fixed, paraffin-embedded tumoral tissues were prepared. Then, a pair of 4 μm sections were placed on slides covered with poly-L lysine for each case. The original HE stained slides were detained for comparison with immunostained sections. The tissue sections were dried for 12 hours in a 37°C oven and then deparaffinized with xylene and rehydrated through graded alcohols. Antigen retrieval was performed by heating them under pressure with EDTA (ScyTek Laboratories, Logan, Utah, USA) for 9 min. The sections which were placed in the aforementioned solutions for 20 min at room temperature were then taken into phosphate-buffered saline (PBS) solution. Endogenous peroxidase was inhibited by incubation with 1% H_2O_2 for 15 min. After washing of samples in PBS, they were incubated with Ultra V Block

Table I. The mean age and sex of both groups

	BCC n = 30	BTCHOF n = 30	p
AGE	64.60 ±10.89	61.43 ±14.43	0.342
SEX			
Female	12 (40%)	16 (53.3%)	0.602
Male	18 (60%)	14 (46.7%)	

(ScyTek Laboratories, Logan, Utah, USA) for hindering non-specific binding. Each pair of the sections was incubated for 2 hours with mouse monoclonal antibody of anti-CD10/ CALLA (neutral endopeptidase) (Ab-2 clone 56C6 Neomarkers Fremont, CAS, USA) as a primary antibody at room temperature. Additional four-time washing with PBS was performed and it was followed by biotinylated UltraTek Anti-Polyvalent antibody (ScyTek) for 20 min as a second antibody. They were washed again in PBS and added to DAB (ScyTek Laboratories, Logan, Utah, USA) chromogene/substrate KIT for 5 min. The sections were counterstained with HE, then dehydrated in alcohols and cleared in xylene, and lastly, balsam was performed onto them and coverslip was mounted. Sections of the small intestine for CD10 were used for positive controls (i6000 automatic staining system Biogenex) which were performed for each case.

Evaluation and statistical analysis

All specimens were observed under a light microscope and the number of immunopositive tumour cells and stromal cells were evaluated using a scale of [0] to [2+] as follows: [0] negative (< 10% positive cells); [1+] regionally positive (10-50% positive cells); [2+] diffusely positive (> 50% positive cells). For the statistical analysis, SPSS – 13.0 was used, which is a computer statistical programme. All the data were expressed as means ± standard errors of means (SEM). Student's t-test in the analysis of numerical variants, Pearson chi-square and Fisher's exact tests for comparing the rational data were used at the suitable areas. Pearson correlation analysis was used for the relationship between the numerical data and p-value less than 0.05 was considered as significant for all the tests.

Results

Patients with BTCOHF (14 females and 16 males) ranged in age from 26 to 74 years (median 61.43 ±14.43) and patients with BCC (12 females and 18 males) ranged in age from 34 to 85 years (median 64.60 ±10.89) (Table I). While both tumour groups were localised on the head region, BTCOHFs were mostly detected on the nasal area and BCCs – on the eye circumference (Tables II, III).

Stromal, peripheral and central expression of CD10 in both tumours were graded from [0] to [2+] for each case. 19 of 30 cases (63.3%) of BCC and 11 of 30 cases of BTCOHF (36.7%) had stromal [0] immunoreactivity. 8 of 30 cases (26.7%) of BCC and 4 of 30 cases of BTCOHF (13.3%) had stromal [1+] immunoreactivity. 15 of 30 cases of BTCOHF (50%) and 3 of 30 cases (10%) of BCC had stromal [2+] immunopositivity. All degrees of stromal immunoreactivity of BTCOHF were significantly stronger than BCC concerning both numerical and degree of expression (p = 0.003) (Figures 1, 2). 14 of 30 cases of BTCOHF (46.7%) and 3 of 30 cases (10%) of BCC had peripheral [1+] immunoreactivity. The peripheral expression of CD10 for BCC was significantly stronger than BTCOHF regarding

Table II. Localisations for BTCHOFs

	Forehead	Nose	Eyebrow	Periorbital	Nasolabial	Back	Preauricular	Scalp	Lip	Cheek
TE	1	5	2	3	5	2	0	2	1	0
TB	0	1	1	0	0	0	1	1	0	1
TA	0	1	0	1	0	0	0	0	0	0
TF	0	1	0	1	0	0	0	0	0	0

Table III. Localisations for BCC

	Forehead	Nose	Eyebrow	Periorbital	Nasolabial	Back	Preauricular	Scalp	Lip	Cheek
Nodular	2	3	2	4	1	0	0	3	1	2
Superficial	1	1	0	0	0	1	0	1	0	0
Infiltrative	0	0	0	0	0	0	1	0	0	1
Mixed	1	0	0	4	0	0	0	0	1	0

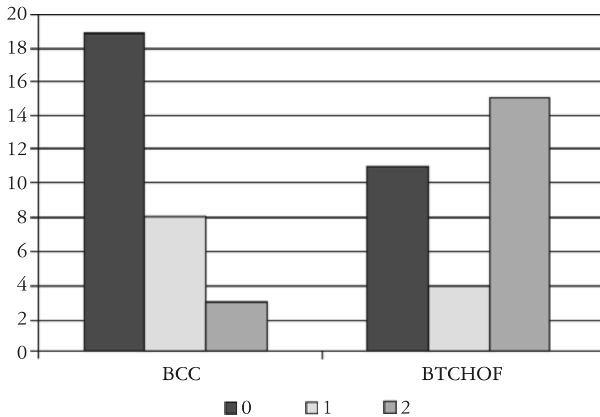

Fig. 1. The comparison of the stromal staining of patterns of CD10 for BTCHOF and BCC

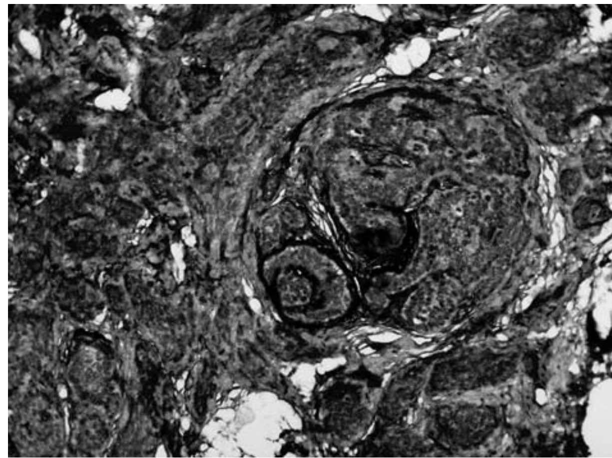

Fig. 2. [2+] stromal expression of CD10 for TE (original magnification, 10 × 20)

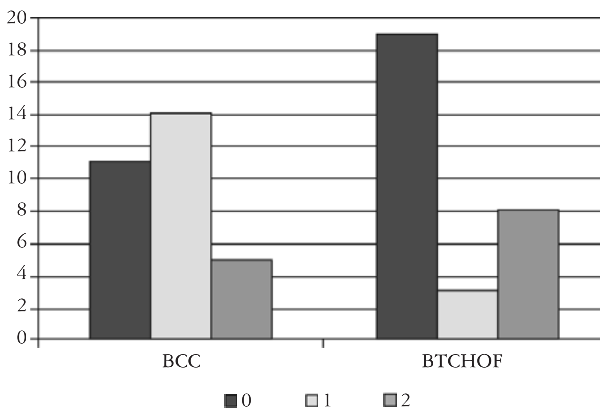

Fig. 3. The comparison of peripheral staining patterns of CD 10 for BTCHOF and BCC

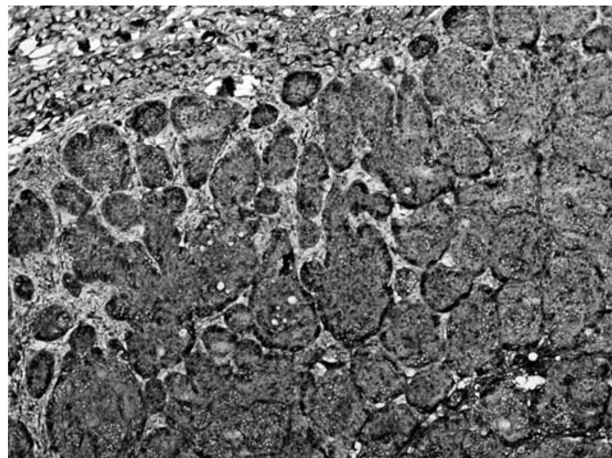

Fig. 4. [2+] peripheral expression of CD10 for TE (original magnification, 10 × 20)

as [1+] immunopositivity ($p = 0.03$). On the contrary, 8 of 30 cases (26.7%) of BTCOHF and 5 of 30 cases of BCC (16.6%) had peripheral [2+] immunoreactivity. The peripheral [2+] immunoreactivity of BTCOHF was stronger than BCC, but not significant (Figures 3, 4). Central expression of CD10 was not significant for both groups.

CD10 expression of the subgroups of both groups is summarized in Tables IV and V and there was no significant difference between the subgroups.

Discussion

A large number of and various contributory laboratory techniques have been researched on the purpose of the correct differentiation between BCC and BTCOHF because their treatment and prognosis are also dissimilar. But they are continuing to be a diagnostic challenge, especially in a small and superficial biopsy.

CD10 is a very useful marker for the differential diagnosis of leukaemia and lymphoma [21] and has been used in the differential diagnosis of various epithelial and mesenchymal neoplasias recently. Renal cell carcinoma, hepatocellular carcinoma, urothelial carcinoma, and prostate carcinoma; endometrial stromal sarcoma, schwannoma, and stromal cells of ovarian epithelial cells express CD10 [30-32]. Ogawa *et al.* detected CD10 reactivity with p53 in stromal cells of colorectal tumours and Iwaya *et al.* assigned CD10 positivity in breast tumours, and then evaluated it for an important prognostic factor for recurrence and survival [32-34]. Similarly, Kazuhiro *et al.* decided in their study that CD10 positivity may be assigned as a marker for estimation of recurrence or invasion in BCCs. They observed CD10 immunoreactivity in tumour cells for less aggressive subtypes and in stromas for more aggressive subtypes [35, 36]. In the study of Pham *et al.* stromal expression of CD10 in TE and cellular expression of CD10 in BCC were significant. They correlated it with stromal features, patterns of the tumour development and discrepancy in host reac-

Table IV. The comparison of staining patterns of the subgroups of BCC with CD10

	Nodular (n = 18)	Superficial (n = 4)	Infiltrative (n = 2)	Mixed (n = 6)	P
Stromal staining with CD10					
0	14 (77.8%)	3 (75%)	1 (50%)	1 (16.7%)	
1	3 (16.7%)	1 (25%)	1 (50%)	3 (50%)	0.156
2	1 (5.6%)	–	–	2 (33.3%)	
Peripheral staining with CD10					
0	8 (44.4%)	–	1 (50%)	2 (33.3%)	
1	7 (38.9%)	3 (75%)	1 (50%)	3 (50%)	0.768
2	3(16.7%)	1 (25%)	–	1 (16.7%)	
Central staining with CD10					
0	13 (72.2%)	3 (75%)	2 (100%)	3 (50%)	
1	4 (22.2%)	1 (25%)	–	2 (33.3%)	0.846
2	1 (5.6%)	–	–	1 (16.7%)	

Table V. The comparison of staining patterns of the subgroups of BTCHOF with CD10

	TE (n = 21)	TB (n = 5)	TA (n = 2)	TF (n = 2)	
Stromal staining with CD10					
0	6 (28.6%)	2 (40%)	2 (100%)	2 (100%)	
1	2 (9.5%)	3 (60%)	–	–	0.081
2	13 (61.9%)	–	–	–	
Peripheral staining with CD10					
0	12 (57.1%)	3 (60%)	2 (100%)	2 (100%)	
1	3 (14.3%)	–	–	–	0.717
2	6 (28.6%)	2 (40%)	–	–	
Central staining with CD10					
0	17 (81%)	4 (80%)	2 (100%)	2 (100%)	
1	1 (4.8%)	–	–	–	0.972
2	3 (14.2%)	1 (20%)	–	–	

tions [5, 37]. However, Costache *et al.* suggested that CD 10 was useless for displaying stromal reactivity [38].

In the current study, we evaluated the stromal, peripheral and central immunoreactivity of CD10 in BTCOHF and BCC grading from [0] to [2+] for each case. Stromal expression of CD10 for BTCOHF was significantly stronger than that of BCC regarding both the numerical and the degree of expression. Contrary to this, peripheral expression in BCC was significantly stronger than that of BTCOHF concerning [1+] immunopositivity. However, peripheral [2+] reactivity of CD10 in BTCOHF was stronger than the other, but not significant. Central expression of CD10 was not significant for both groups. In our study, stronger stromal staining in BTCOHF and stronger peripheral staining in BCC with CD10 were observed and these results were similar to Pham *et al.*'s ones [39]. Additionally, the outcome of staining of the same localizations supported the hypothesis of follicular differentiation of both groups, despite the degree of immunopositivity was different from each other.

There was no significant difference between the subgroups of both groups concerning CD10 immunoreactivity. When we analysed the subgroups of each group, we detected that the mixed type of BCC developing in nodular [+] infiltrative pattern had a tendency to obtain the stromal staining more densely according to the other subtypes of BCC. This

result was supporting the study of Yada *et al*. [32], i.e. when capability of invasion increases in BCC, the tendency of stromal staining with CD10 may become possible.

In conclusion, we have determined in this study that CD10 may be useful for the differential diagnosis between BTCOHF and BCC as an immunohistochemical marker and it may solve a dilemma for the clinicians and the pathologists particularly in the small and superficial biopsies. In our opinion, studying with CD10 may even be a life-saving method for the selected cases. For all that, a limited number of the cases was the handicap of our study. However varied markers, dissimilar techniques, and a larger multicentred series are necessary for the distinct discriminations.

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Address for correspondence

Ilker Sengul, General Surgery, MD, Asst. Prof.
The Founder Chairman of the Department of General Surgery
Vice Dean's Office
Deanery of Giresun University Faculty of Medicine
28100 Giresun
Turkey
Vice-chancellor's office: +90 454 310 10 00
Deanery: +90 454 214 14 96
GSM: +90 530 885 00 50
fax: +90 454 310 10 16
e-mail: dr.ilker52@mynet.com