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Localized Amyloidosis in Basal Cell Carcinoma: A Single-Center Experience

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Abstract

Objective: Basal cell carcinoma (BCC) is the most common skin cancer. In the literature, studies investigating amyloid deposition in this tumor are limited. This study aimed to evaluate the frequency of amyloid deposition using histochemical Congo red staining in BCC cases and to analyze its association with clinicopathological parameters.

Methods: A total of 243 patients with histopathologically confirmed BCC who had excisional biopsy specimens between January 1, 2022, and November 30, 2024, were included from the pathology archives. The presence of amyloid was assessed by Congo red staining and confirmed by apple-green birefringence under polarized light microscopy.

Results: The mean age of the patients was 64.0±13.3 years, and 58.4% were male. The most frequent tumor localization was the head and neck region (78.2%). Histologically, the nodular subtype was the most common (91.4%). Amyloid deposition was detected in 26.3% of cases, exclusively in the nodular subtype. Amyloid positivity was significantly higher in the older age group ($p = 0.003$). Moreover, a significant association was observed with higher Clark level ($p < 0.001$) and recurrence ($p = 0.045$), whereas no significant correlation was found with sex, localization, ulceration, or peritumoral inflammation.

Conclusion: This study demonstrated that amyloid deposition in BCC may be associated with certain clinicopathological parameters. The findings suggest that amyloid deposition may not only represent a histopathological feature but also a potential prognostic marker. Larger prospective studies incorporating immunohistochemical analyses are needed to better elucidate this relationship.

Keywords: Basal cell carcinoma, amyloid, localized cutaneous amyloidosis, Congo red

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Bazal Hücreli Karsinomda Lokalize Amiloidoz Varlığı: Tek Merkez Deneyimi

Öz

Amaç: Bazal hücreli karsinom (BHK) en sık görülen deri kanseridir. Literatürde bu tümörde amiloid birikimine ilişkin sınırlı sayıda çalışma bulunmaktadır. Bu çalışmada, bazal hücreli karsinom olgularında histokimyasal Kongo red boyası ile amiloid birikiminin sıklığını araştırmak ve klinikopatolojik parametrelerle ilişkisini değerlendirmek amaçlanmıştır.

Yöntemler: Çalışmaya, 1 Ocak 2022 – 30 Kasım 2024 tarihleri arasında Sivas Cumhuriyet Üniversitesi Tıbbi Patoloji Anabilim Dalı arşivinde histopatolojik olarak bazal hücreli karsinom tanılı ve eksizyonel biyopsi örneğine sahip 243 hasta dahil edilmiştir. Amiloid varlığı, Kongo red boyası ve polarizan mikroskopta elma yeşili çift kırılma ile değerlendirilmiştir.

Bulgular: Hastaların yaş ortalaması 64.0±13.3 yıl olup, %58,4'ü erkekti. Tümörlerin en sık yerleşim yeri baş-boyun bölgesiydi(%78,2). Histolojik olarak en sık nodüler tip (%91,4) izlendi. Amiloid birikimi olguların %26,3'ünde saptandı ve bu birikim yalnızca nodüler tipte gözlendi. Amiloid pozitiflik oranı ileri yaş grubunda anlamlı derecede daha yüksekti (p=0,003). Ayrıca Clark seviyesi (p<0,001) ve nüks varlığı (p=0,045) ile anlamlı ilişki bulunurken, cinsiyet, lokalizasyon, ülserasyon ve peritümöral inflamasyonla ilişki saptanmadı.

Sonuç: Bu çalışma, bazal hücreli karsinomda amiloid birikiminin klinikopatolojik parametrelerle ilişkili olabileceğini göstermiştir. Bulgular, amiloid varlığının yalnızca histopatolojik bir bulgu değil, aynı zamanda olası bir prognostik belirteç olabileceğini düşündürmektedir. Bu ilişkinin daha iyi anlaşılması için geniş serili prospektif ve immünohistokimyasal çalışmalar gereklidir.

Anahtar kelimeler: Bazal hücreli karsinom, amiloid, lokalize deri amiloidozu, Kongo red.

INTRODUCTION

Basal cell carcinoma (BCC) originates from the basal layer of the epidermis or adnexal basal cells and accounts for approximately 70–80% of all skin tumors[1,2]. It most frequently arises on sun-exposed areas, particularly the face and neck, with ultraviolet (UV) radiation considered the predominant etiological factor. Other etiological agents include prolonged arsenic exposure, ionizing radiation therapy, human immunodeficiency virus (HIV) infection, and immunosuppressive drugs[3]. BCC is typically a slow-growing neoplasm with a very low metastatic potential (<0.01%)[2,4]. According to the World Health Organization's (WHO) 2023 Classification of Skin Tumors (5th edition), BCC is categorized into nodular, superficial, micronodular, infiltrative, morpheaform (sclerosing), fibroepithelial, pigmented, and basosquamous subtypes. The diagnostic gold standard is histopathological examination, which commonly reveals a proliferation of uniform basaloid cells with hyperchromatic nuclei[3].

Amyloid is a proteinaceous material composed of fibrils that accumulate in the extracellular space. Histopathologically, amyloid appears as homogeneous, amorphous, eosinophilic extracellular deposits on hematoxylin and eosin (H&E) staining. However, this appearance is not specific, as it may be confused with hyaline material, sclerotic collagen, fibrin, or necrotic debris. For this reason, special stains are required for definitive identification. The most reliable method is Congo red staining, which demonstrates apple-green birefringence under polarized light[5]. Amyloid may accumulate in either systemic or localized forms. Localized cutaneous amyloidosis is classified into primary forms (e.g., lichen and macular amyloidosis) and secondary forms, the latter usually associated with neoplastic processes[6].

Amyloid deposition in BCC was first described in 1930[7,8]. It has been suggested that apoptotic keratinocytes contribute to amyloid deposition through the transformation of tonofilaments into amyloid fibrils with a beta-pleated sheet configuration[1]. The association between amyloid deposition and histological subtypes of BCC, peritumoral inflammatory intensity,

ulceration, perineural invasion, recurrence, and other prognostic parameters remains unclear.

The aim of this study is to investigate amyloid deposition in basal cell carcinoma (BCC) using histochemical Congo red staining and to evaluate its association with clinicopathological parameters. In view of the limited literature on this topic, our study systematically and in greater detail examines the histopathological findings and explores their relationships with clinical data. Accordingly, we anticipate that the results will provide new insights into the biological behavior and potential prognostic significance of amyloid deposition in BCC.

METHODS

This study was designed as a retrospective, descriptive analysis. The study included cases that had a histopathological diagnosis of basal cell carcinoma (BCC) between 1 January 2022 and 30 November 2024 in the archives of the Department of Medical Pathology and for whom slides from excisional surgical specimens were available.

The inclusion criteria were a histopathologically confirmed diagnosis of basal cell carcinoma (BCC) and the availability of an excisional specimen containing sufficient tumor tissue. The exclusion criteria included cases with inadequate biopsy material, missing clinical data and cases in which two pathologists could not reach a diagnostic consensus.

Clinical data for all eligible patients (age, sex, lesion localization, and presence of recurrence) were obtained from the hospital information system. Histopathological variables (tumor subtype, Clark level, perineural invasion, ulceration, tumor size, peritumoral inflammatory intensity, and amyloid deposition) were abstracted from the final pathology reports. All slides for all cases were reviewed by two pathologists. Diagnostic confirmation was performed, and any histopathological parameters required for the study but missing from the original pathology report were re-evaluated and recorded. Regarding recurrence follow-up, only cases with data available at our center were included; patients followed at external institutions were not considered.

For each case, histologic slides were reviewed, and the slide and paraffin block that best represented the tumor mass and contained areas suspicious for amyloid-like extracellular material were selected. From the selected paraffin blocks, 4-µm-thick sections were cut onto positively charged slides. Histochemical Congo red staining was performed using a fully automated special-stains platform (Ventana BenchMark Special Stains; Roche Diagnostics, Germany) with a commercial kit. Sections from histologically confirmed renal amyloidosis served as positive controls in each run. The presence of amyloid was assessed using a Nikon Eclipse 80i microscope (Nikon, Japan), and positivity was confirmed by the demonstration of apple-green birefringence under polarized light.

Cases were categorized into two groups according to the presence or absence of amyloid detected by Congo red (present/absent). The dependent variable of the study was histochemically detected amyloid deposition. The independent variables were age, sex, tumor size, lesion localization (face, scalp/other head, neck, trunk, upper extremity, lower extremity), tumor subtype (nodular, infiltrative, superficial), peritumoral inflammatory intensity (mild, moderate, severe), perineural invasion (present/absent), ulceration (present/absent), Clark level (1–5, 1: epidermis, 2: papillary dermis,3: filling of papillary dermis, 4: reticular dermis, 5:subcutis)[3], and recurrence (present/absent).

The study received approval from the Institutional Ethics Committee (Approval No. 2024.12/72), was conducted in accordance with the Declaration of Helsinki, and used strict anonymization safeguards; written informed consent was obtained from all participants.

Statistical Analysis

All analyses were performed using SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were calculated

for all variables. The normality of continuous variables was assessed with the Kolmogorov–Smirnov and Shapiro–Wilk tests. Variables conforming to a normal distribution are presented as mean ± standard deviation, whereas non-normally distributed variables are presented as mean (minimum–maximum). Categorical variables are summarized as n (%). Between-group comparisons were conducted using the chi-square (χ^2) test or Fisher's exact test for categorical variables, and the t-test or Mann–Whitney U test for continuous variables, as appropriate to distributional assumptions. To identify factors associated with amyloid positivity, variables that reached significance at $p < 0.05$ in univariate analyses were entered into a multivariable logistic regression model (enter method). Regression results are reported as the regression coefficient (B), standard error (SE), Wald statistic, p-value, and odds ratio [Exp(B)] with 95% confidence intervals (CI). Model classification performance was evaluated using the classification table. A two-sided $p < 0.05$ was considered statistically significant for all tests.

RESULTS

A total of 243 patients were included in this study. The mean age of the cohort was 64.0±13.3 years (range: 22–96 years). Of these, 58.4% were male (n = 142) and 41.6% were female (n = 101).

With respect to anatomical localization, the most frequent site was the facial region, accounting for 78.2% of cases (n = 190). This was followed by the scalp (n = 31, 12.8%), neck (n = 5, 2.1%), trunk (n = 6, 2.5%), upper extremity (n = 6, 2.5%), and lower extremity (n = 5, 2.1%).

Histopathological subtype distribution revealed that the nodular subtype predominated (n = 222, 91.4%), while infiltrative (n = 14, 5.8%) and superficial subtypes (n = 7, 2.9%) were less frequent.

Peritumoral inflammation (PTI) was distributed relatively evenly across categories: mild (n = 82, 33.7%), moderate (n = 79, 32.5%), and severe (n = 82, 33.7%). Perineural invasion (PNI) was identified in 7.8% of cases (n = 19). Ulceration was observed in 44.9% of tumors (n = 109).

Regarding Clark invasion levels, stage 4 was the most frequent (n = 166, 68.3%), followed by stage 3 (n = 43, 17.7%). Less frequently, stage 5 (n = 28, 11.5%) and stage 2 (n = 6, 2.5%) were observed, while no cases of stage 1 were encountered.

Amyloid deposition was identified in 26.3% of cases (n = 64) using Congo red staining and polarized light microscopy (Figure 1 and 2). Tumor recurrence was present in 15.6% of patients (n = 38). The mean tumor size was 1.10±0.85 cm (range: 0.1–7.0 cm). Detailed findings are presented in Table I.

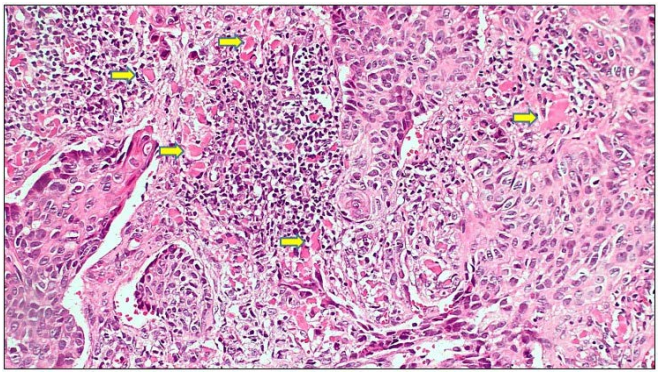

Figure 1. Basal cell carcinoma with localized amyloidosis. Tumor showing hyperchromatic nuclei, eosinophilic cytoplasm, peripheral palisading, and stromal retraction, with scattered deposits of amorphous eosinophilic amyloid (yellow arrows) (H&E, ×200).

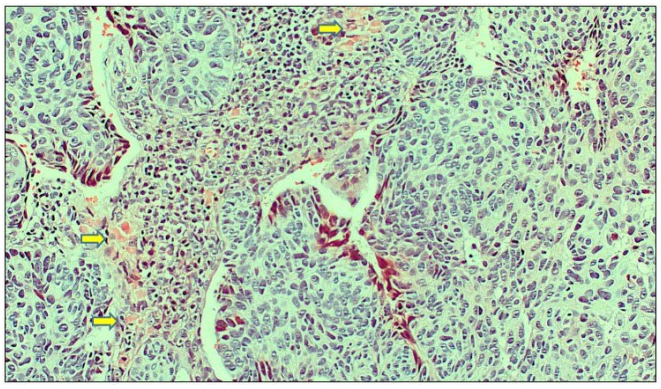

Figure 2. Basal cell carcinoma with localized amyloidosis. Amyloid deposits exhibiting pink–orange congophilic staining (yellow arrows) (Congo red, ×200).

Table I: Demographic and Clinical Characteristics of the Patients

Variables (n=243)	n %
Sex	
Female	101 41.6
Male	142 58.4
Localization	
Face	190 78.2
Scalp/Other cranial sites	31 12.8
Neck	5 2.1
Trunk	6 2.5
Upper extremity	6 2.5
Lower extremity	5 2.1
Tumor Subtype	
Nodular	222 91.4
Infiltrative	14 5.8
Superficial	7 2.9
Peritumoral Inflammation	
Mild	82 33.7
Moderate	79 32.5
Severe	82 33.7
Perineural Invasion	
Absent	224 92.2
Present	19 7.8
Ulceration	
Absent	134 55.1
Present	109 44.9
Clark Level	
1	0 0.0
2	6 2.5
3	43 17.7
4	166 68.3
5	28 11.5
Amyloid Deposition	
Absent	179 73.7
Present	64 26.3
Recurrence	
Absent	205 84.4
Present	38 15.6
Age (Mean±SD, max-min)	64.02± 13.28 (22-96)
Tumor Size (cm)	1.10±0.85 (0.1-7.0)

The associations between amyloid deposition and various clinical and histopathological parameters are summarized in Table II. No statistically significant relationship was found between amyloid positivity and sex, tumor localization, degree of peritumoral inflammation (PTI), or the presence of ulceration ($p > 0.05$).

In contrast, significant differences were observed in certain parameters:

-Tumor subtype was significantly associated with the presence of amyloid ($p = 0.016$).

-Perineural invasion (PNI) was more frequently observed in amyloid-negative cases ($p = 0.007$).

-Clark invasion level demonstrated a statistically significant difference between the groups ($p < 0.001$).

-The presence of recurrence was significantly associated with amyloid deposition ($p = 0.045$).

In addition, the mean age was significantly higher in the amyloid-positive group ($p = 0.003$). Although the mean tumor size was larger in amyloid-positive cases, the difference did not reach statistical significance ($p = 0.098$).

Table II: Comparison of Demographic and Clinicopathological Parameters According to Amyloid Deposition

Variables (n=243)	n %	Amiloid (-) n %	Amiloid (+) n %	p *
Sex				
Female	101 41.6	74 41.3	27 42.2	0.906
Male	142 58.4	105 58.7	37 57.8	
Localization				
Face	190 78.2	140 78.2	50 78.1	
Scalp/Other sites	31 12.8	22 12.3	9 14.1	
Neck	5 2.1	4 2.2	1 1.6	
Trunk	6 2.5	5 2.8	1 1.6	0.935
Upper extremity	6 2.5	5 2.8	1 1.6	
Lower extremity	5 2.1	3 1.7	2 3.1	
Tumor Subtype				
Nodular	222 91.4	158 88.3	64 100.0	
Infiltrative	14 5.8	14 7.8	0 000.0	**0.016**
Superficial	7 2.9	7 3.9	0 000.0	
Peritumoral Inflammation				
Mild	82 33.7	62 34.6	20 31.3	
Moderate	79 32.5	56 31.3	23 35.9	0.780
Severe	82 33.7	61 34.1	21 32.8	
Perineural Invasion				
Absent	224 92.2	160 89.4	64.0 100.0	**0.007**
Present	19 7.8	19 10.6	00.0 000.0	
Ulceration				
Absent	134 55.1	99 55.3	35 54.7	0.932
Present	109 44.9	80 44.7	29 45.3	
Clark Level				
1	0 00.0	0 00.0	0 00.0	
2	6 2.5	6 3.4	0 00.0	
3	43 17.7	41 22.9	2 3.1	
4	166 68.3	109 60.9	57 89.1	**< 0.001**
5	28 11.5	23 12.8	5 7.8	
Recurrence				
Absent	205 84.4	146 81.6	59 92.2	**0.045**
Present	38 15.6	33 18.4	5 7.8	
Age (Mean±SD, max-min)	64.02± 13.28 (22-96)	62.53±13.55 (22-96)	68.19±11.60 (33-90)	**0.003**
Tumor Size (cm)	1.10±0.85 (0.1-7.0)	1.03±0.74 (0.1-4.5)	1.29±1.06 (0.3-7.0)	0.098

A p-value < 0.05 was considered statistically significant.

Table III: Multivariable logistic regression analysis.

Variables	B	p	OR Exp B	%95 CI
Age	0.035	0.012	1.036	1.008-1.064
Clark level				
1	-2.595	0.001	0.075	0.016-0.355
2	-2.174	0.019	0.114	0.019-0.695
3	0.0226	0.701	1.253	0.396-.3970
Recurrence	1.124	0.037	3.078	1.072-8.837

B: Beta coefficient, OR: Odds Ratio, CI: Confidence Interval

The multivariable logistic regression model included variables that were significant in univariate analyses (age, tumor subtype, perineural invasion, Clark level, and recurrence). The regression results are presented in Table 3. Accordingly, age, Clark level, and recurrence were significantly associated with amyloid positivity. For age (B = 0.035; OR = 1.036; p = 0.012), each one-year increase was associated with an approximately 3% higher odds of amyloid positivity, suggesting that age may be an independent risk factor. Lower Clark levels demonstrated a protective effect, whereas recurrence significantly increased the risk. Tumor subtype and perineural invasion were not significantly associated with amyloid positivity in the multivariable model.

DISCUSSION

This study investigated amyloid deposition in BCC cases using Congo red staining and evaluated its association with clinicopathological parameters. In our series,

amyloid deposition was observed in 26.3% of cases and was significantly more frequent in the nodular subtype of BCC. Moreover, amyloid positivity showed statistically significant associations with advanced age, recurrence, and higher Clark levels, as well as with the absence of perineural invasion. These findings suggest that the presence of amyloid may have potential implications for the biological behavior and prognostic features of BCC, in line with the limited number of similar studies available in the literature.

Although reports on amyloid deposition in BCC are scarce, the incidence has been reported to range between 8% and 75%. The majority of series indicate that amyloid deposition is most frequently detected in nodular subtypes, while it is rarely observed in superficial or morpheaform types. A male predominance and higher incidence in patients over the age of 60 have also been emphasized, with the head and neck region being the most common site of occurrence[1,4,9,10]. Our findings align with trends reported in the literature: amyloid deposition is particularly prominent in nodular cases, and patterns related to age, sex, and head–neck localization suggest a non-random distribution within the clinicopathological context. The interstudy variability in reported rates is likely attributable to heterogeneity in sampling (representativeness of selected blocks, number of sections examined) and methodological approaches. Given the potential for microscopic, focal distribution of amyloid, it should be underscored that single-block/single-section assessment may limit sensitivity; although we sought to mitigate bias by selecting the section with the greatest tumor burden, this limitation cannot be fully eliminated. Prospectively, multi-block/multi-section protocols and standardized assessment criteria (with immunophenotypic support where appropriate) may more reliably map subtype-specific biology and reduce the observed variability.

The pathogenesis of amyloid deposition in BCC remains uncertain, and it is still debated whether the amyloid is truly tumor-derived. Multiple mechanisms have been proposed. The most widely accepted hypothesis is that during apoptosis of keratinocytes, degenerated tonofilaments undergo structural transformation into fibrillar proteins, which subsequently accumulate extracellularly as amyloid. Since BCC develops slowly, with tumors reported to reach 1 cm in diameter over approximately six months[2], prolonged exposure to etiological agents may further promote amyloid deposition[1,4,5,11]. In addition, the peritumoral inflammatory microenvironment has been suggested as a facilitating factor in this process[12]. Collectively, these findings indicate that amyloid deposition in BCC is not attributable to a single source but rather represents a multifactorial process shaped by tumor cell death, stromal response, and immune mechanisms.

Our results further demonstrated significant associations between amyloid positivity and specific histopathological parameters. Amyloid was most frequently observed in nodular subtypes, consistent with previous studies reporting a strong link between this subtype and amyloid deposition, whereas none of the superficial or infiltrative BCCs in our cohort exhibited amyloid. Although amyloid deposition has been reported across different BCC subtypes in the literature, it is considerably less frequent in non-nodular types[4]. The mean age of amyloid-positive cases in our series was significantly higher than that of amyloid-negative cases, supporting earlier reports describing increased amyloid incidence in patients over 60 years of age. Male predominance and a predilection for the head and neck region in our series were also in agreement with prior studies[1,9,13-15].

From a histopathological perspective, amyloid positivity demonstrated an inverse association

with perineural invasion, as none of the amyloid-positive cases exhibited perineural invasion. To the best of our knowledge, this observation has not been emphasized in previous literature and may reflect a relationship between amyloid deposition and less aggressive histological features. Similarly, higher Clark levels were more frequent in amyloid-negative cases, suggesting a possible inverse correlation between deep invasion capacity and amyloid deposition. A comparable trend was observed for recurrence. Taken together, our findings imply that BCC cases exhibiting adverse histopathological prognostic factors may demonstrate lower rates of amyloid deposition. While ulceration was observed more frequently in amyloid-negative cases in our cohort, no statistically significant relationship was established, consistent with earlier reports[4]. Peritumoral inflammation also showed no significant association with amyloid status. We believe that future studies assessing the composition and density of inflammatory cell populations may provide a more detailed understanding of this relationship. These results collectively indicate that amyloid deposition in BCC should not be regarded merely as a passive histopathological phenomenon but may represent a parameter influencing tumor biology and clinical outcome.

The strengths of this study include a relatively large sample size for assessing amyloid deposition in BCC and a comparative analysis of the findings with clinicopathological parameters; moreover, the single-center design and use of standardized histochemical methods enhance data reliability. Nevertheless, several limitations should be acknowledged: the retrospective design, the evaluation of only a single section from a single block in each case, and the absence of immunohistochemical characterization of amyloid subtypes. Notably, the single-section approach may under-detect focal amyloid deposits and thereby reduce apparent sensitivity; to mitigate this, for each case we selected the slide and paraffin block that best represented the tumor and examined the corresponding section. In addition, because routine screening data for systemic amyloidosis were unavailable, this variable could not be included in the study.

CONCLUSION

Our study demonstrates that amyloid deposition in BCC is associated with specific clinicopathological characteristics and provides valuable data that contribute to the limited literature on this topic. Our findings suggest that amyloid deposition may not merely represent a passive histopathological feature but could potentially serve as a prognostic marker.

Ethical approval: The study received approval from the Institutional Ethics Committee (Approval No. 2024.12/72), was conducted in accordance with the Declaration of Helsinki, and used strict anonymization safeguards; written informed consent was obtained from all participants.

Conflict of Interest: The authors declared no conflicts of interest.

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