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Original Article

Management of incidental dermatofibrosarcoma protuberans: A single center 5-year experience

Insidental olarak saptanan dermatofibrosarkom protuberans tedavisi: Tek merkez 5 yıllık deneyimlerimiz

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Abstract

Aim: Dermatofibrosarcoma protuberans (DFSP) is a rare skin tumor. The diagnosis is challenging because it is usually hard to differ DFSP from other skin lesions. Distant metastasis is rare, but local recurrence is common.The aim of this study is to review the treatment algorithms in DFSPs that are detected incidentally and to increase awareness about this rare tumor.

Material and Methods: 17 patients who underwent excision of epidermal lesions which were considered to be benign and were diagnosed with DFSP, at department of general surgery between 2012 and 2017, were analyzed.

Result: A total of 17 patients were analyzed retrospectively. No recurrence was detected in any of ourpatients.

Conclusion: Awareness of this rare entity is important for diagnosis and management of the disease.

Keywords: dermatofibrosarcoma protuberans; mesenchymal tumor; mohs micrographic surgery (MMS)

Öz

Amaç: Dermatofibrosarkoma protuberans (DFSP) nadir görülen bir deri tümörüdür. Teşhis zordur çünkü DFSP'yi diğer deri lezyonlarından ayırmak genellikle zordur. Uzak metastaz nadirdir ancak lokal nüks yaygındır. Bu çalışmanın amacı incidental olarak saptanan DFSP'lerde tedavi algoritmalarını gözden geçirmek ve bu nadir tümör hakkında farkındalığı artırmaktır.

Gereç ve Yöntemler: 2012-2017 yılları arasında genel cerrahi kliniğinde benign olduğu düşünülenerek eksizyonu yapılan ve DFSP tanısı alan 17 hasta retrospektif olarak incelendi.

Bulgular: Toplam 17 hasta geriye dönük olarak analiz edildi. Hastalarımızın hiçbirinde nüks saptanmadı.

Sonuç: Bu nadir antitenin farkında olunması hastalığın tanı ve tedavisi için önemlidir.

Anahtar kelimeler: dermatofibrosarkom protuberans; mezenkimal tümör; mohs mikroskopik cerrahi (MMS)

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Introduction

Dermatofibrosarcoma protuberans (DFSP) is a very rare mesenchymal skin tumor. In the latest World Health Organization (WHO) 2013 classification, these tumors were classified as moderately malignant myofibroblastic tumors [1]. Although it was first described by Darier and Ferrand, the no men clature was made by Hoffman [2].

It constitutes less than 1% of all malignancies and approximately 1% of all soft tissue sarcomas. The incidence is between 0.8 and 4.5 permillion [3-7]. Although congenital cases have been reported, they usually occur in the 3rd decade [8-10]. Although it may have a nodular appearance, such as slow-growing hypertrophic scars, it can also occur without any symptoms, such as soft tissue sarcomas. Hematogenous or lymphatic spread is very rare in DFSPs. Despite showing only local growth; it has an aggressive behavior all patterns that progress to the dermis, subcutaneous tissue, and finally to muscles as finger-like extensions. There is no difference between races, but pigmented DFSP, also known as Bednar tumor, is more common in the black race [11]. Although the difference between the genders is not provento be significant, there are different studies showing that women or men are slightly more common [8,12].

Approximately 10-15% of cases can turn into spindle cell carcinoma similar to adult fibrosarcoma. Such cases are associated with increased mitotic activity and loss of CD34 expression. In tumors that develop fibrosarcomatous transformation, the local recurrence rate is similar to ordinary dermatofibrosarcoma, but distant metastasis is observed in approximately 13% of these patients [13].

Diagnosis is madeby skin biopsy in cases which have been suspected before; however, since the early clinical symptoms of DFSP are nonspecific, diagnosis is difficult. Therefore, it is also frequently encountered in excisions of skin lesions which were considered to be benign.

Standart treatment is surgical excision. Mohs surgery or wide local excision may be preferred. The most challenging part of the management is to achieve local control. Because DFSP originates from the dermis, it invades the collagen bundles and deep connective tissue radially, and therefore it is very difficult to obtain a clean surgical border. Local recurrence has been reported in approximately 50% of studies [14].

In this study, our aim is to review the treatment algorithms in DFSPs that are detected incidentally and to increase awareness about this rare tumor.

Material and Methods

Our study began with the approval of the ethical committee of our institute. Between 2012 and 2017, patients who underwent mass excision and were diagnosed with DFSP in our center were evaluated retrospectively. Patients previously diagnosed with dermatofibrosarcoma and had re-excision, and those diagnosed with biopsy before excision, were excluded from the study. Patients were compared in terms of age, gender, tumor localization, surgical margin status, re-excision status, surgical margin status after re-excision, immunohistochemical markers, mitotic activity in 10 hpf, Ki67 proliferation index, follow-up times, whether they received radiotherapy and complications related to radiotherapy. All patients were called for clinical control and the presence of recurrence was investigated. Distance larger than 0.1 cm was considered as clean surgical margin. Statistical analysis was performed using SPSS v22 statistical program. This study was carried out in accordance with the Declaration of Helsinki and was approved by the local ethics committee and informed consents were taken from all participants.

Results

Between 2012-2017, 17 patients who had mass excision and were diagnosed as dermatofibrosarcoma protuberans incidentally were evaluated retrospectively. The clinicopathological features of the patients are shown in Table 1. When the gender distribution was analyzed, it was determined that 9 of 17 patients were male and 8 were female. The average age at the time of diagnosis was 43.88 (21-72). The tumor was on the back in 5 patients (29.4%), on the forearm in 4 patients (23.5%), on the anterior chest wall in 3 patients (17.6%), on the shoulder in 3 patients (17.6%), and on the leg in 2 patients (11.8%). Surgical margin positivity was detected in 12 patients (70.6%) after the first resection. Clean surgical margins were obtained in 5 patients (29.4%) after the first resection. Re-excision was performed in 12 patients with surgical margin positivity. However, 8 of these 12 patients (66.6%) were found to have clean surgical margins, while 4 patients (33.3%) were not able to achieve clean surgical margins. The treatment was completed with radiotherapy in 4 patients who had positive margins after re-excision. Radiation-induced dermatitis was observed in 2 patients receiving radiotherapy, while no complications occurred in other patients. While CD34 was positive in all patients, we have detected Factor 13A in 6 patients (35.3%), p53 in 2 patients (11.8%), CD99 in 2 patients (11.8%), and vimentin in

2 patients (11.8%) (Table 2). The mitotic activity in 10 hpf and Ki 67 proliferation index did not pass the homogeneity test. The median value for the number of mitosis was 4.00, and the median value for the Ki 67 proliferation index was 7.00. Clinically and radiologically, no recurrence was observed in any of the patients called for control in October 2019. Radiological recurrence control was done by ultrasonography. Our average follow-up time was 23.9 months. This study was approved by the local ethics committee, all procedures were carried out in accordance with the 2013 Helsinki Declaration and informed constents were taken from all participants.

Table 1.

Statistics	Age	Mitosis	Ki 67	Following time
Mean	43,88	4,71	10,12	24,59
Median	43,00	4,00	7,00	17,00
Std. Deviation	13,656	4,469	16,035	15,879
Minimum	21	1	1	6
Maximum	72	20	70	61

Discussion

Dermatofibrosarcoma protuberans (DFSP) is a very rare mesenchymal skin tumor. Although it has been reported that it is slightly more common in women in some studies, it has been shown in many studies that it is seen in both sexes equally as well as in our study [4]. It may appear nodular, such as a slowly growing hypertrophic scar, or appear without any symptoms, like soft tissue sarcomas. In addition, atrophic plaque or morphea-like appearance may cause delay in diagnosis [15]. Although its dimensions vary during diagnosis, it can be detected in sizes ranging from 0.5 cm to 12 cm [4]. Many morphological variants have been defined, and the pigment variant is called the "Bednar Tumor" [16]. It usually shows only local growth but has an aggressive growing pattern such as giving finger like extensions into the dermis, subcutaneous tissue and finally muscle tissue [8]. In 60% of cases, tumor cells extend parallel to the epidermis [8,9].

Table 2.

		Count	Row N %	Column N %	Table N %
Gender	Male	9	100,0%	52,9%	52,9%
	Female	8	100,0%	47,1%	47,1%
Localization	Dorsum	5	100,0%	29,4%	29,4%
	Chest	3	100,0%	17,6%	17,6%
	Shoulder	3	100,0%	17,6%	17,6%
	Leg	2	100,0%	11,8%	11,8%
	Forearm	4	100,0%	23,5%	23,5%
Surgical Margins (SM)	Negative	5	100,0%	29,4%	29,4%
	Positive	12	100,0%	70,6%	70,6%
Re-excision	No	5	100,0%	29,4%	29,4%
	Yes	12	100,0%	70,6%	70,6%
SM After Re-excision	Negative	8	100,0%	66,6%	66,6%
	Positive	4	100,0%	33,3%	33,3%
Radiation Therapy	No	13	100,0%	76,5%	76,5%
	Yes	4	100,0%	23,5%	23,5%
CD34	Negative	0	0,0%	0,0%	0,0%
	Positive	17	100,0%	100,0%	100,0%
Factor 13A	Negtive	11	100,0%	64,7%	64,7%
	Positive	6	100,0%	35,3%	35,3%
p53	Negative	15	100,0%	88,2%	88,2%
	Positive	2	100,0%	11,8%	11,8%
CD99	Negative	15	100,0%	88,2%	88,2%
	Positive	2	100,0%	11,8%	11,8%
Vimentin	Negative	15	100,0%	88,2%	88,2%
	Positive	2	100,0%	11,8%	11,8%
Recurrence	No	17	100,0%	100,0%	100,0%
	Yes	0	0,0%	0,0%	0,0%
Complication	No	15	100,0%	88,2%	88,2%
	Yes	2	100,0%	11,8%	11,8%

In the early stages, DFSPs can be mixed with lipomas, epidermalcysts, keloid tissue or nodular fasciitis [17]. In advanced disease, pyogenic granuloma, Kaposi's sarcoma and other soft tissue sarcomas should also be considered. There are also studies showing that it can develop in traumatized tissues or scars that have under gone multiple surgical procedures [18]. Hematogenous or lymphatic spread is very rare in DFSPs [17]. DFSP is microscopically characterized by diffuse infiltration of dermis and subcutaneous tissue. The tumor grows among fibrous septi and infiltrates adipose tissue, creating the typical honeycomb look. The atypia is minimal and mitotic rate is low [15]. Increased mitotic activity, necrosis and fibrosarcomatous changes; are indicators of aggressive behavior and poor prognosis [19].

Immunohistochemically, vimentin, CD34, apolipoprotein D, nestin and sometimes EMA can be detected in tumorcells. Mostly, desmin, S100 protein, stromelysin III, tenascin and keratin are negative. Infibrosarcomatous DFSPs, CD 34 loss and increased TP53 expression can be demonstrated [19,20].

Genetically, DFSP is characterized by a COL1A1- PDGFB gene fusion in most cases. The promoter and variable portions of the collagen 1A1 (COL1A1) gene are combined with exon 2 of the platelet-derived growth factor beta (PDGFB) gene, causing irregular regulation of PDGFB protein [21,22]. At the chromosome level, gene fusion is caused by the exchange of substances between the chromosome bands 17q21 (COL1A1) and 22q13 (PDGFB). This exchange can be seen as balanced or unbalanced t (17; 22) or as one or more super numerary ring chromosomes [14,23,24]. These ring chromosomes, which may contain many copies of fusion genes or other parts of the arms of 17q and 22 q chromosomes, are more common in elderlypatients [25]. The other form is more common in children [26,27]. In rare cases, fusion of PDGFB with other chromosomal regions has been demonstrated. COL6A3-PDGFB fusion was demonstrated in the DFSP of the breast. This fusion, like COL1A1-PDGFB fusion, activates the PDGFB receptor [28,29].

Intreatment, excision of the skin and subcutaneous tissue with distant surgical margins is recommended [30]. If there is muscle or bone invasion; resection of these tissues are also recommended to obtain negative surgical margins [31]. There currence rate is related to the width of the resection [32]. In some studies, recurrence rates have been shown to be under 5% in those with a clean margin of 5 cm or more [31]. Mohs surgery, also known as Mohs micrographic surgery (MMS), is the name of the method in which the tumor is gradually removed into thin layers and examined. This process continues until there are not any tumor cells in the samples taken. It can be done in one session as an out patient procedure. Reasonable tissue excision ,which is the basic principle of MMS, reduces scar tissue an deliminates the need for future surgical or medical treatment [33,34]. The average lesion length at the time of diagnosis ranges from 4.4 cm to 4.9 cm in different studies. The average wound area is 21.7 cm2 in Mohs surgery and 63.4 cm2 in wide local excision [35-37].

Imantinib mesylate is an oral tyrosine kinase inhibitor. It can be used in adults for recurrent, unresectable and metastatic disease. It prevents the binding of ATP to the PDGF-beta receptor, a tyrosine kinase, by competitive inhibition. This; slows kinase activity, limits tumor growth and provides apoptosis. Patients with t (17; 22) translocation respond better to imatinib and therefore this translocation should be investigated prior to treatment. This translocation can be detected by the FISH (fluorescent in situhybridization) or reverse transcription polymerase chain reaction (RT-PCR)methods. Imatinib has side effects such as indigestion, edema, fatigue, anemia, and skin rash. Most of the patients which have translocation respond well to imatinib mesylatetherapy. In studies, the response to imatinib treatment is about 65%. The duration of treatment is variable. Some sources recommend 6 months of treatment, but this can be extended if needed. Alternatively, radiotherapy can be used in unresectable or recurrent tumors. In addition, adjuvant radiotherapy can reduce the risk of local recurrence [38-39]. Radiotherapy combined with surgery should be considered in the presence of a positive or inadequate surgical margin, in cases of recurrence, or if extensive surgical excision will have unacceptable cosmetic or functional out comes [40,41].

One of the limitations of our study is the absence of translocation testing. Chemotherapy had not been tried in any of our patients, but successful results had been achieved with radiotherapy.

The number of patients in our study is 17. Since this is a relatively small group, more studies on incidental cases will be a guide for what lesions should be suspected.

Conclusion

Performing a biopsy before excision in patients with a skin lesion and suspection of dermatofibrosarcoma protuberance is important in order to achieve clean surgical margins and to prevent re-excision. In this way, chemotherapy and radiotherapy treatments that may need to be given additionally can be prevented. Therefore, increasing our awareness about

DFSP will increase the success in local control, which is the most challenging part in the treatment of this disease.

Declaration of conflict of interest

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Referanslar

1. Jo VY, Fletcher CDM. WHO classification of soft tissue tumours: an update based on the 2013 4thedition. Pathology (Phila) 2014; 46: 95–104.
2. Lemm D, Mügge L-O, Mentzel T, Höffken K. Current treatment options in dermatofibrosarcoma protuberans. J Cancer Res Clin Oncol 2009; 135: 653–65.
3. Bhambri S, Desai A, Del Rosso JQ, Mobini N. Dermatofibrosarcoma protuberans: a case report and review of the literature. J Clin Aesthet Dermatol 2008; 1: 34–6.
4. Gloster HM, Jr. Dermatofibrosarcoma protuberans. J Am Acad Dermatol 1996; 35: 355–74.
5. Hong YJ, Choi YW, Myung KB, Choi HY. A case of myxoid dermatofibrosarcoma protuberans. Ann Dermatol 2011; 23: 379–81.
6. Rutkowski P, Wozniak A, Switaj T. Advances in molecular characterization and targeted therapy in dermatofibrosarcoma protuberans. Sarcoma 2011; 2011: 959132.
7. Dragoumis DM, Katsohi LA, Amplianitis IK, Tsiftsoglou AP. Late local recurrence of dermatofibrosarcoma protuberans in the skin of female breast. World J Surg Oncol 2010; 8: 48.
8. Criscione VD, Weinstock MA. Descriptive epidemiology of dermatofibrosarcoma protuberans in the United States, 1973 to 2002. J Am Acad Dermatol 2007; 56: 968-73.
9. Burkhardt BR, Soule EH, Winkelmann RK, Ivins JC. Dermatofibrosarcoma protuberans. Study of fifty-six cases. Am J Surg 1966; 111: 638-44.
10. Reddy C, Hayward P, Thompson P, Kan A. Dermatofibrosarcoma protuberans in children. J Plast Reconstr Aesthet Surg 2009; 62: 819-23.
11. Amonkar GP, Rupani A, Shah A, Deshpande R. Bednar Tumor: An Uncommon Entity. Dermatopathology (Basel) 2016; 3: 36-8.
12. Chen H, Zhou L, Xiong J et al. Clinicopathological analysis of 14 cases of early stage dermatofibrosarcoma protuberans. Zhonghua Yi Xue Za Zhi 2015; 95: 3445–8.
13. Voth H, Landsberg J, Hinz T, Wenzel J, Bieber T, Reinhard G, Höller T, Wendt- ner C-M, Schmid-Wendtner M-H. Management of dermatofibrosarcoma pro- tuberans with fibrosarcomatous transformation: an evidence-based review of the literature. J Eur Acad Dermatol Venereol 2011; 25: 1385–91.
14. Mentzel T, Pedeutour F, Lazar A, Coindre J-M. In: Fletcher CDM, Bridge JA, Hogendoorn PCW, Mertens F, editors. Dermatofibrosarcoma protuberans. Lyon: WHO Classification of Tumours of Soft Tissue and Bone; 2013. p. 77–9.
15. Fletcher CDM, Bridge JA, Hogendoorn P, Mertens F (Eds). WHO Classification of Tumors of Soft tissue and Bone. IARC, Lyon, 2013, 77-79.
16. Martin L, Piette F, Blanc P et al; French Group for Cutaneous Oncology. Clinical variants of the preprotuberant stage of dermatofibrosarcoma protuberans.Br J Dermatol 2005; 153: 932-6.
17. Angouridakis N, Kafas P, Jerjes W et al. Dermatofibrosarcoma protuberans with fibrosarco- matous transformation of the head and neck. Head Neck Oncol 2011; 3: 5.
18. McKee PH, Calonje JE, Granter S et al. Pathology of the Skin, with Clinical Correlations. Vol. 2. Philadelphia Elsevier-Mosby; 2005.
19. Llombart B, Serra-Guillén C, Monteagudo C, López Guerrero JA, Sanmartín O. Dermatofibrosarcoma protuberans: a comprehensive review and update on diagnosis and management. Semin Diagn Pathol 2013; 30: 13-28.
20. Liang CA, Jambusaria-Pahlajani A, Karia PS, Elenitsas R, Zhang PD, Schmults CD. A systematic review of outcome data for der- matofibrosarcoma protuberans with and without fibrosarcomatous change. J Am Acad Dermatol 2014; 71: 781-6.
21. Simon M-P, Pedeutour F, Sirvent N et al. Deregulation of the platelet-derived growth factor β-chain gene via fusion with collagen gene COL1A1 in dermatofibrosarcoma protuberans and giant-cell fibroblastoma. Nat Genet 1997; 15: 95–8.
22. O'Brien KP, Seroussi E, Dal Cin P et al. Var- ious regions within the alpha-helical domain of the COL1A1 gene are fused to the second exon of the PDGFB gene in dermatofibrosarcomas and giant-cell fibroblastomas. Genes Chromosomes Cancer 1998; 23: 187–93.
23. Pedeutour F, Coindre J-M, Nicolo G, Bouchot C, Ayraud N, Turc Carel C. Ring chromosomes in dermatofibrosarcoma protuberans contain chromosome 17 sequences: fluorescence in situ hybridization. Cancer Genet Cytogenet 1993; 67: 149.
24. Pedeutour F, Simon MP, Minoletti F, Sozzi G, Pierotti MA, Hecht F, Turc-Carel C. Ring 22 chromosomes in dermatofibrosarcoma protuberans are low-level am- plifiers of chromosome 17 and 22 sequences. Cancer Res 1995; 55: 2400–3.
25. Terrier-Lacombe MJ, Guillou L, Maire G et al. Dermatofibrosarcoma protuberans, giant cell fibroblastoma, and hybrid lesions in children: clinico- pathologic comparative analysis of 28 cases with molecular data - A study from the french federation of cancer centers sarcoma group. Am J Surg Pathol 2003; 27: 27–39.

26. Pedeutour F, Simon MP, Minoletti F, Barcelo G, Terrier-Lacombe MJ, Combe- male P, Sozzi G, Ayraud N, Turc-Carel C. Translocation, t(17;22)(q22;q13), in dermatofibrosarcoma protuberans: a new tumor-associated chromosome rear- rangement. Cytogenet Genome Res 1996; 72: 171–4.
27. Sirvent N, Maire G, Pedeutour F. Genetics of dermatofibrosarcoma protuberans family of tumors: from ring chromosomes to tyrosine kinase inhibitor treat- ment. Genes Chromosomes Cancer 2003; 37: 1–19.
28. Dadone-Montaudié B, Alberti L, Duc A et al.. Alternative PDGFD rearrangements in dermatofi- brosarcomas protuberans without PDGFB fusions. Mod Pathol 2018; 31: 1683– 93.
29. Dickson BC, Hornick JL, Fletcher CDM, Demicco EG, Howarth DJ, Swanson D, Zhang L, Sung Y-S, Antonescu CR. Dermatofibrosarcoma protuberans with a novel COL6A3-PDGFD 14-fusion gene and apparent predilection for breast. Genes Chromosomes Cancer 2018; 57: 437–45.
30. Khatri VP, Galante JM, Bold RJ, Schneider PD, Ramsamooj R, Goodnight JE Jr. Dermatofibrosarcoma protuberans: reappraisal of wide local excision and impact of inadequate initial treatment. Ann Surg Oncol 2003; 10: 1118-22.
31. Chang CK, Jacobs IA, Salti GI. Outcomes of surgery for dermatofibrosarcoma protuberans. Eur J Surg Oncol 2004; 30: 341- 5.
32. Fiore M, Miceli R, Mussi C et al. Dermatofibrosarcoma protuberans treated at a single institu- tion: a surgical disease with a high cure rate. J Clin Oncol 2005; 23: 7669-75.
33. Skin Cancer Foundation. Mohs surgery; 2019: https://www.skincancer.org/skin-cancer-information/mohs-surgeryAccessed 2019 Mar 19.
34. Nehal K, Lee E. Mohs surgery. In: Post TW, ed. UpToDate. Waltham (MA): UpToDate; 2018: www.uptodate.comAccessed 2019 Feb 20.
35. Valdivielso-Ramos M, Torrelo A, Campos M, Feito M, Gamo R, Rodriguez-Peralto JL. Pediatric dermatofibrosarcoma protuberans in Madrid, Spain: multi-institutional outcomes. Pediatr Dermatol 2014; 31: 676-82.
36. Zhou X, Sun D, Liu Y et al. Dermatofibrosarcoma protuberans: our 10-year experience on 80 patients[published online June 9, 2019]. J Dermatolog Treat.
37. DuBay D, Cimmino V, Lowe L, Johnson TM, Sondak VK. Low recurrence rate after surgery for dermatofibrosarcoma protuberans: a multidisciplinary approach from a single institution. Cancer 2004; 100: 1008-16.
38. Amavi AK, Dossouvi T, Padaro E, Adabra K, Dosseh ED. Management for locally advanced dermatofibrosarcoma protuberans in Togo. Bull Cancer 2018; 105: 333-4.
39. Yadav S, Verma N, Khurana N, Neogi S. Recurrent Dermatofibrosarcoma Protuberans with Pigmention and Myoid Differentiation. Sultan Qaboos Univ Med J. 2018; 18: 228-30.
40. Dagan R, Morris CG, Zlotecki RA, Scarborough MT, Mendenhall WM. Radiotherapy in the treatment of dermatofibrosarcoma protuberans. Am J Clin Oncol 2005; 28: 537–9.
41. Ballo MT, Zagars GK, Pisters P, Pollack A. The role of radiation therapy in the management of dermatofibrosarcoma protuberans. Int J Radiat Oncol Biol Phys 1998; 40: 823–7.