Juvenile–onset, Severe Peripheral Edema in Miniature Dachshunds

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Abstract: Four miniature Dachshunds with juvenile onset, severe peripheral edema are described. All dogs developed edema at 3 to 5 months old at the pinnae, tail and it was observed at the muzzle, periocular area, extremities, and prepuce in some. Physical examination and blood examinations included complete blood count, and serum chemistry analysis showed no evidence suggesting a primary cause of edema in three cases. Case 4 showed temporary, moderate hypoalbuminemia, but no clinical and laboratory findings to lead underline disorders were found. Histopathologic findings revealed pale-staining collagen losing its fibrillar character with extensive edema in the dermis in all dogs. Based on these findings, congenital vascular disturbance in the skin was suspected and the edema was reduced after administration with tocopherol nicotinate and/or carbazochrome sodium sulfonate hydrate.

Key words: congenital, edema, miniature Dachshund

要 約: 若齢で発症した著しい末梢浮腫を呈したミニチュア・ダックスフンド 4例を報告した。浮腫は 4か月齢で耳介と尾に生じ、一部では口吻、眼周、四肢、包皮にもみられた。4例は身体検査および血液検査で浮腫の原因と考えられる特記すべき異常は認められなかった。症例 4では中程度の低アルブミン血症が一過性にみられたが、基礎疾患を示唆する臨床像および検査所見はみられなかった。皮膚病理組織像は真皮全層に広範な浮腫と本来の原線維構造を失った淡い膠原線維がみられた。これら所見より皮膚の先天的血管機能異常が予想され、浮腫はニコチン酸トコフェロールやカルバゾクロムスルホン酸ナトリウムの投与後に軽減した。

キーワード: 先天的要因、浮腫、ミニチュア・ダックスフンド

Introduction

Edema is defined as an abnormal collection of water in the interstitial space. Approximately 60 to 70% of lean body weight is water, about two thirds of which is contained in the intracellular space and one third in the extracellular space. Most extracellular water is contained in the interstitium, a matrix of connective tissue and gel-like substrate that forms the area between the vascular compartment and the cell membrane. Edema occurs when this relationship is disturbed, and there are various causes including increased hydrostatic pressure, reduced plasma osmotic pressure, lymphatic obstruction, sodium retention, and inflammation. In general, edema is a secondary feature of many clinical presentations, and also a non-diagnostic feature of any inflammatory dermatoses. This report describes 4 miniature Dachshunds with juvenile-onset, severe peripheral edema without any significant primary causes.

Case Report

Case 1: a 3-month-old, intact female miniature Dachshund presented at Oda Animal Hospital with marked edema on the pinnae, muzzle, extremities, and tail, as well as pruritus at the pinnae, and hyperpigmentation, alopecia, and scaling on the tail (Fig. 1a). No evidence suggesting systemic circulation disorders was found during careful physical examination. Complete blood count, serum chemistry analysis, urine examination and X-ray examination were unremarkable. Biopsy specimens were taken from the extremities and tail.

Case 2: a 7-month-old, intact female miniature Dachshund presented at Marcy Animal Hospital with edema and alopecia on the pinnae and tail. The owner noticed the symptoms when she was 3 months old. The puppy was in good health aside from skin lesions. No evidence suggesting systemic circulation disorder was found during careful physical examination. Complete blood count and serum chemistry analysis were unremarkable. Biopsy specimens were taken from the pinnae and tail.

Case 3: a 4-month-old, intact female miniature Dachshund presented at Veterinary Medical Teaching Hospital, Nippon Veterinary and Life Science University with pruritus and marked edema on the pinnae, muzzle, periocular areas, extremities, and tail, as well as hyperpigmentation, alopecia, and scaling on the pinnae (Fig. 1b). The severity of the edema varied daily. No evidence suggesting systemic circulation disorder was found during careful physical examination. Complete blood count and serum chemistry analysis were unremarkable. Biopsy specimens were taken from the pinnae and tail.

Case 4: a 5-month-old, intact male miniature Dachshund presented at Harley Animal Hospital with pruritus and marked edema and alopecia on the pinnae, muzzle, periocular area, extremities, tail, and prepuce, as well as hyperpigmentation and scaling on the pinnae, vertex, and tail (Fig. 1c). The puppy was in good health aside from skin lesions. No evidence suggesting systemic circulation disorder was found during careful physical examination, and edema was observed only in peripheral areas. Complete blood count and serum chemistry analysis were unremarkable, except for moderate hypoalbuminemia (Alb: 1.6 g/dl). Biopsy specimens were taken from the head, pinnae and tail.

In all cases, histopathologic findings were basically the same; they revealed pale-staining collagen losing its fibrillar character with extensive edema and diffuse infiltration of mononuclear cell at the dermis (Fig. 2). Pyoderma, demodicosis, dermatophytosis, Malassezia dermatitis, and juvenile cellulitis were ruled out based on clinical features and histopathologic examination. In three cases there were no abnormalities suggesting systemic circulation disorder, liver and kidney dysfunctions, and protein exsorption enteropathy as causes for ascites and pleural effusion as well as edema. Case 4 showed only moderate hypoalbuminemia, even though the dog’s general condition was quite good. Appropriate food had been fed, and no complaint regarding urine and stool presented. The dog has grown without any obstruction. Hypoalbuminemia was reevaluated one and half year later, and the level was within normal (2.3 g/dl, reference range: 2.3–3.6 g/dl). Urinalysis was also performed at the same moment, and no proteinurea was found. In this case, association of hypoalbuminemia with the onset or aggravation of the edema was not completely ruled out, but it was validated that the same etiology to other cases presented.

All dogs were treated with tocopherol nicotinate (Juvela N, Eisai Co., Ltd., Tokyo, Japan) at a dose of 50–100 mg once or twice daily to enhance peripheral circulation and vascular function. In cases 1 and 3, carbazochrome sodium sulfonate hydrate (Adona, Mitsubishi Tanabe Pharma Co., Osaka, Japan) at a dose of 7.5–10 mg twice daily was also administered for the same therapeutic strategy. Their efficacy was observed in all cases, but...
symptoms did not completely disappear and relapsed mainly in the most severe areas. In case 1, edema was completely resolved with concurrent use of tocopherol nicotinate and carbazochrome sodium sulfonate hydrate, even though secondary lesions such as hyperpigmentation and alopecia remained. In case 2, the condition improved with tocopherol nicotinate, but exacerbation was noted after 9 months withdrawal of the agent. The dog showed edema, hyperpigmentation and alopecia on the tail, and mild edema and alopecia on the pinnae and bridge of the nose (Fig. 1d). Some claws became deformed and fell off. Then, tocopherol nicotinate was administered again, and edema became less apparent. In case 3, both tocopherol nicotinate and carbazochrome sodium sulfonate hydrate were administered as initial treatment, and edema was reduced. However, anti-pruritic agents such as antihistamine and glucocorticoids were required to manage some scratching behavior at the edema. Those agents were effective with
occasional usage rather than continuous usage for pruritus, but not edema itself. Case 4 was treated with antihistamine for pruritus initially, and it seemed to be effective for pruritus, but not edema. Then tocopherol nicotinate was added for the remaining edema, and this was apparently reduced. Based on these findings, it is suggested that tocopherol nicotinate and carbazochrome sodium sulfonate hydrate are likely to be effective to reduce the edema.

Discussion

We reported 4 miniature Dachshunds with severe peripheral edema characterized by juvenile onset. This edema was not related to any systemic circulation disturbance, even though hydrostatic pressure, and plasma osmotic pressure were not objectively evaluated. In addition, no primary inflammatory dermatoses were recognized in these dogs. To the best of our knowledge, there have been no reports describing severe peripheral edema in juvenile miniature Dachshunds. Based on these findings, congenital vascular disturbance in the skin was clinically suspected and evaluated therapeutically. We used tocopherol nicotinate and carbazochrome sodium sulfonate hydrate as an etiologic therapy. Tocopherol nicotinate (Juvela N, Eisai Co., Ltd., Tokyo, Japan) dilates the vessels of the peripheral vascular system to increase the blood flow and relieves symptoms related with hypertension or peripheral circulation disturbance\(^1\). Carbazochrome sodium sulfonate hydrate (Adona, Mitsubishi Tanabe Pharma Co., Osaka, Japan) acts on the capillaries to inhibit an increase in vascular permeability, and enhances the vascular resistance\(^5\). In all cases, some efficacy was observed, even though symptoms did not completely disappear.

Histopathologically, this edema was characterized by pale-staining collagen losing its fibrillar character with extensive edema at the dermis, which was compatible with ischemic dermatopathy. This term is used to group multiple vasculopathic syndromes by similar clinical and histopathologic characteristics\(^3, 4\). The pathogenesis is not well elucidated, but both the clinical and histopathologic features correlate with lesions likely to result from long-term lack of cutaneous vascular sustenance. Cutaneous hypoxia can lead to follicular atrophy and chronic skin changes such as scaling and hyperpigmentation. Lesions occur over bony prominences due to susceptibility to trauma, and on the distal extremities where collateral circulation does not allow appropriate vascular sustenance\(^3, 4, 8, 9\). This has been reported in Shetland sheepdogs, Collies, and related crossbreeds, Chow Chow, Beauceron shepherds, Welsh corgi, Lakeland terrier, German shepherd dog, and Kuvasz\(^3, 4, 8\). While miniature Dachshunds are described in the textbook, we were not able to find any case reports in the literature. Although edema is not a typical feature of ischemic dermatopathy, various clinical signs were possibly seen in each breed\(^5\). It is considered that edema represents ischemic vasculopathy in miniature Dachshunds.

In the present dogs, the severity of the edema was various and changeable. This might be explained by both the degree of vascular anomaly and acquired external and internal factors such as unstable function during the growth stage, environment factors such as temperature, mechanical stress, autonomic stimulation due to psychogenic stresses, hormones such as estrus, and drugs that lead to changes of blood flow. For investigation of the etiology, further careful follow-up is warranted in these cases and additional cases with pedigree analysis.

References