

## A case of pyoderma gangrenosum in a dog successfully treated with prednisolone alone

*Een geval van pyoderma gangrenosum bij een hond succesvol behandeld met enkel prednisolone*

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### ABSTRACT

In this case report, a markedly painful ulcerative dermatitis consistent with pyoderma granulosum is reported in a 2.5-year-old entire female Maltese dog. The dog had a nasal stridor and irregular ulcers with raised inflammatory borders involving the lumbar area, tail and the hindlimbs. The lesions did not respond to antibiotics. Histopathologic features include deep crateriform ulcerations with massive infiltrations of neutrophils beneath and adjacent to the ulcers. Treatment with prednisolone (Kela Laboratories, Sint-Niklaas, Belgium) alone resulted in the resolution of nasal signs and all skin lesions.

### SAMENVATTING

In deze casuïstiek wordt een opvallend pijnlijke, ulceratieve dermatitis overeenstemmend met pyoderma gangrenosum beschreven bij een 2,5 jaar oude, intacte, vrouwelijke maltezer. De hond had een nasale stridor en vertoonde onregelmatige ulceraties met verheven inflammatoire randen op de lenden, staart en achterste ledematen. Er was geen respons op diverse antibiotica. Het histopathologisch beeld werd gekenmerkt door diepe kratervormige ulceraties met een uitgesproken neutrofiel infiltraat, onderliggend en aan de randen van de verzweringen. Een behandeling met enkel prednisolone (Kela Laboratories, Sint-Niklaas, Belgium) resulteerde in een complete regressie van de nasale symptomen en huidletsels.

### INTRODUCTION

Pyoderma gangrenosum (PG) is a rare, destructive, reactive sterile inflammatory skin disease (Gross et al., 2005; Miller et al., 2013). Only three cases have been reported in dogs in the peer-reviewed literature (Bardagi et al., 2007; Simpson et al., 2013). It is one of the group of neutrophilic dermatoses in dogs that also includes subcorneal pustular dermatosis and Sweet's syndrome. Canine PG lesions are described as multifocal, markedly painful, hemorrhagic pustules and nodules, which breakdown to form discharging irregular ulcers with elevated undermined borders. Lesions typically involve the trunk, particularly the dorsum, but also the face, the limbs, the tail base and tail. Mucosal involvement has not been reported. The phenomenon of pathergy or Koebner phenomenon (new lesions forming in response to minor trauma) and the clinical finding of cribriform scarring, minor human diagnostic

criteria, have been documented in two dogs (Simpson et al., 2013). Dogs may be febrile and depressed. In humans, PG may either be idiopathic or associated with underlying systemic disease, or more rarely, has been linked to many kinds of surgery and various drugs (Ruocco et al., 2009). Canine PG has been associated with preceding polyarthritis in one dog (Bardagi et al., 2007), and considered idiopathic in two dogs (Simpson et al., 2013). PG is diagnosed by compatible clinical features, with exclusion of other (infectious) causes of ulcerative disease, and by supportive histopathology (characteristic large crateriform ulcers with massive infiltrations of neutrophils beneath and adjacent to the ulcers) (Gross et al., 2005; Ruocco et al., 2009). The determination of whether there is an associated disorder is mandatory (Ruocco et al., 2009). Reported effective treatments in the dog include oral prednisolone in conjunction with ciclosporin (Bardagi et al., 2007) or with azathioprine (Simpson et al., 2013).

In the present report, the clinical aspects of PG in a Maltese dog and the successful treatment with prednisolone alone are described.

### CASE DESCRIPTION

A 2.5-year-old, entire female Maltese dog was presented with a five-week history of a painful and progressive skin eruption that started on the dorsal trunk and a five-day history of nasal stridor and sneezing. Treatment prior to presentation included an unknown course of oral cephalexin, followed by an unknown course of oral doxycycline (Ronaxan, Merial Belgium, Diegem) combined with oral meloxicam (Meloxoral, Le Vet B.V., Oudewater, the Netherlands). No further therapeutic information was available. Five days prior to admission, the dog had received orally 3 mg/kg verapamil (Verapamil, Bayer Austria GmbH, Vienna, Austria) given once a day. The previous medical history revealed two antecedents of back pain, respectively two months and one month before the onset of the skin condition, which had been treated each time with five-day courses of meloxicam orally 0.1 mg/kg once daily. On several occasions, the dog had travelled with the owners to Spain.

Upon physical examination, the skin lesions were so painful that the dog had to be muzzled. When muzzled, 'bubble blowing' (nasal formation of air bubbles) was observed (Figure 1). Rectal temperature was 39.2°C. The peripheral lymph nodes were normal on palpation. There were only few skin lesions present. A cluster of lesions -some of them had coalesced- involved the dorsal lumbar region. Solitary and more extensive lesions were present on the dorsal proximal part of the tail and on the lateral aspects of both tarsi. The skin lesions were irregular ulcers covered by crusts. The ulcers had an elevated purulent border and a necrotic base (Figure 2), or had a margin surrounded by a halo of bright erythema. An extensive crusted lesion on the tail was discharging hemorrhagic exudate and its proximal margin revealed hemorrhagic pustulation (Figure 3). Differential diagnoses included deep bacterial pyoderma, fungal infection, leishmaniasis, vasculitis and pyoderma gangrenosum.

A complete blood count, serum biochemistry profile and cytology of ulcerated skin were performed. Multiple skin biopsies were submitted for histopathology. Cytology revealed purulent inflammation but no micro-organisms. A complete blood count showed mild neutrophilia  $16.9 \times 10^9/L$  (normal range  $6.0 - 12.0 \times 10^9/L$ ). No abnormalities were found on serum biochemistry. Leishmania infantum antibodies titres were not detected. While awaiting the histopathological findings, verapamil therapy was continued. The skin lesions had not improved at day 15. No micro-organisms were identified on histopathology on hematoxylin and eosin, and periodic acid Schiff stains. Histopathological



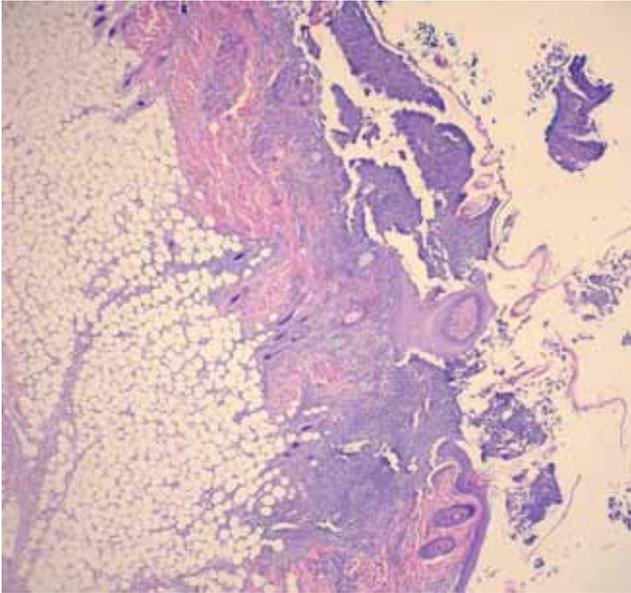
**Figure 1. Photograph of the muzzled 'bubble blowing' dog.**



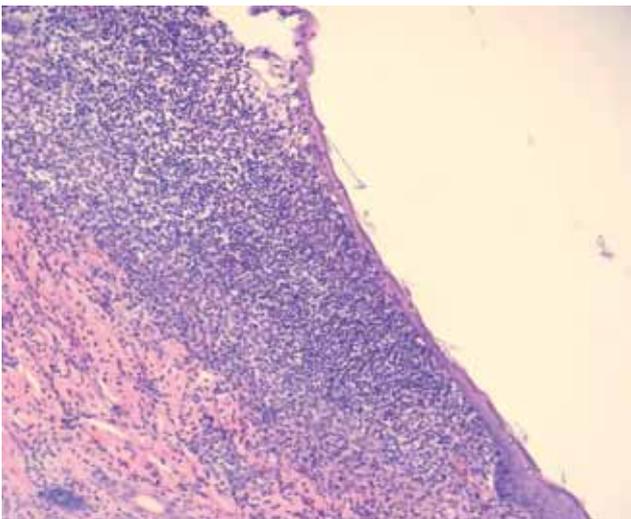
**Figure 2. Photograph of the flank. Small irregular ulcer with a raised inflammatory border and a necrotic base.**



**Figure 3. Photograph of the tail. Extensive crusted lesion discharging a hemorrhagic exudate. Inset shows hemorrhagic pustulation (arrow) arising at its proximal border.**



**Figure 4. Photomicrograph. Large dermal pustule and crateriform ulceration extending into the panniculus. Note marked neutrophilic infiltrate. Hematoxylin and eosin stain 40x.**



**Figure 5. Photomicrograph. Subepidermal neutrophilic infiltrate surrounded by mononuclear cells adjacent to an ulceration. Hematoxylin and eosin stain 200x.**

with a raised inflammatory and irregular border and a necrotic base (Ruocco et al., 2009; Simpson et al., 2013).

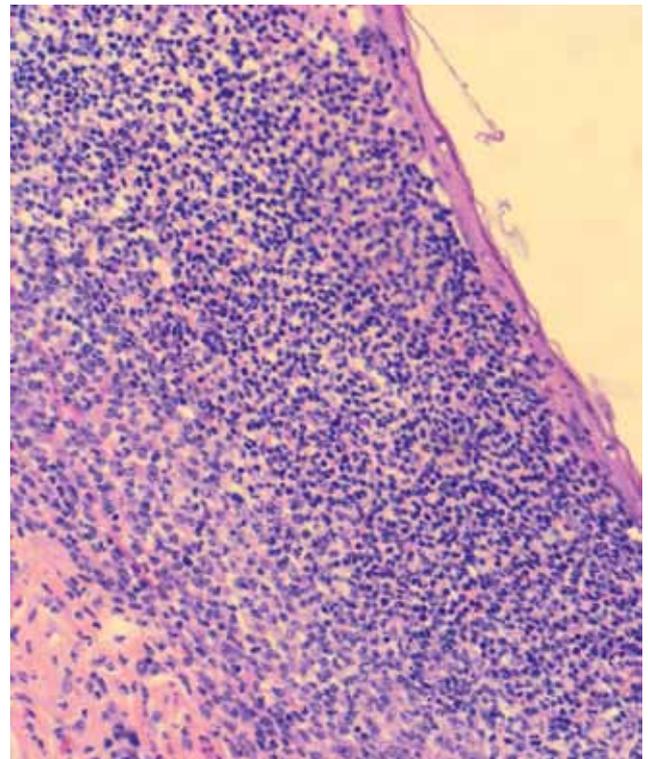
The dog in the present report had skin lesions consistent with PG. The lesions were markedly painful, irregular ulcers with an elevated inflammatory border and a necrotic base or ulcers with the margins surrounded by a halo of bright erythema. The extensive lesion on the dorsal tail was discharging a hemorrhagic exudate and had a hemorrhagic pustulation arising on its proximal border. Moreover, there was no lymphadenopathy, an invariably feature in humans (Ruocco et al., 2009). A Koebner phenomenon, present in 20% of human cases, and cribriform scarring were lacking diagnostic criteria. In published dog cases, these

examination revealed large crateriform ulcers, that penetrate to the deep dermis and often extend into the panniculus, with massive infiltrations of neutrophils beneath and adjacent to the ulcers (Figure 4). Superficial dermal neutrophilic inflammation was surrounded by mononuclear cells (Figures 5 and 6).

Pyoderma gangrenosum was diagnosed and antibiotics were stopped. Treatment with 1 mg/kg oral prednisolone (Prednisolone, Kela Laboratories, Sint-Niklaas, Belgium) twice a day was initiated. One week later, the owner reported a marked improvement. The cutaneous pain and nasal stridor had resolved. Treatment was continued. On re-examination, after five weeks of twice-daily prednisolone 1 mg/kg, the skin lesions were completely resolved, except for the alopecia and scarring. The prednisolone dosage was then reduced to 1.2 mg/kg given once every second day for three weeks, and finally given once every third day for another three weeks. The dog was followed up for eight months and no relapse occurred.

## DISCUSSION

The diagnosis of PG rests primarily upon clinical features and exclusion of other causes of ulceration, as there is no specific laboratory test and as the histopathology is indicative, but not diagnostic (Gross et al., 2005; Ruocco et al., 2009). In both humans and dogs, one of the hallmark signs of PG is evidence of extreme pain disproportionate to the gross appearance of the lesions. The salient clinical feature is an ulcer



**Figure 6. Photomicrograph. Closer view of the same section as in figure 5. Hematoxylin and eosin stain 400x.**

criteria have not always been present (Bardagi et al., 2007). The lesion distribution in the present case was trunk, tail and limbs, which fits with the description in published canine PG cases.

The involvement of mucous membranes has not been reported in canine PG, but has occasionally been observed in human PG (Ruocco et al., 2009). The dog in the present case had a late history of nasal disease characterized by nasal stridor, sneezing and 'bubble blowing'. Its nasal signs readily responded to prednisolone treatment. Therefore, it was reasonable to suspect nasal mucosa involvement in the dog's condition. However, true evidence by microscopic demonstration of neutrophilic mucosal inflammation, was lacking. Infectious causes of cutaneous ulceration were ruled out by the lack of micro-organisms on cytology and histopathology and by the lack of response to antibiotics of different classes, e. i. cephalexin, doxycycline, verapamil. Histopathological findings of large crateriform ulcers were non-specific, but were highly supportive for PG in conjunction with clinical features and the lack of response to antibiotics (Gross et al., 2005; Ruocco et al., 2009).

Underlying conditions could not be identified. Interestingly, prior to the onset of the skin lesions, the dog of this report had been treated twice with meloxicam for back pain. Triggering of PG by these antecedents or by the administered meloxicam cannot be completely discarded.

There is no specific and uniformly effective therapy for PG. Systemic corticosteroids have generally been the most predictable effective medication in human PG when delivered in adequate doses. The overall prognosis of human PG is good in those patients who readily respond to treatment (Ruocco et al., 2009). In this case, monotherapy with prednisolone was elected because the dog could readily be controlled with pred-

nisolone 1 mg/kg given twice daily. Within a week, the cutaneous pain had markedly decreased (in human the literature described as the first sign of remission) (Bardagi et al., 2007), and nasal stridor had ceased. At five weeks of treatment, all the skin lesions had completely resolved. Prednisolone dosage and the frequency of administration were reduced and stopped after six weeks. There was permanent scarring and no recurrences.

In summary, PG in dogs is a rare and markedly painful, ulcerative skin condition, which may have nasal mucosa involvement. Glucocorticoids alone can be an effective treatment option in some cases.

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