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Prednisolone is associated with an increase in serum insulin but not serum fructosamine concentrations in dogs with atopic dermatitis

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ABSTRACT

The aim of this study was to assess the effects of a standard therapeutic protocol of prednisolone (Pred) on glucose homeostasis in atopic dogs and compare it with previously published data for ciclosporin A (CsA). The central aim of the study was to assess and compare the effects of standard therapeutic protocols of prednisolone (Pred) and ciclosporin A (CsA) on glucose homeostasis in dogs with atopic dermatitis (CAD).

Both treatments significantly reduced the physical signs of CAD, as determined by the canine atopic dermatitis extent and severity index version 3 (CADESI-03) and the Edinburgh Pruritus Scale (EPS). Post-treatment plasma glucose concentrations were not significantly different in the two groups, but serum insulin concentrations were significantly higher following Pred therapy ($P < 0.05$). Serum fructosamine concentrations were not significantly different pre- and post-treatment with Pred, although previous studies had shown that CsA treatment increased fructosamine concentrations ($P < 0.005$). The two treatment groups were recruited in a similar timeframe, were numerically matched and there were no differences in CADESI-03 and EPS scores between the CsA and Pred groups either before or after treatment. Thus, both CsA and Pred treatment were associated with mild disturbances in glucose metabolism, but only CsA therapy resulted in a significant increase in fructosamine concentrations. This information may be relevant to clinicians when considering therapeutic options for dogs with atopic dermatitis which already have impaired glucose homeostasis.

Introduction

Canine atopic dermatitis (CAD) is defined as a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features associated with IgE antibodies most commonly directed against environmental allergens (Halliwell, 2006). Outcomes of randomised controlled trials (RCTs) on the treatment of CAD, subjected to systematic review, have indicated that medications with good evidence of efficacy include topical glucocorticoids, oral glucocorticoids (GC) (prednisone, prednisolone (Pred) and methylprednisolone), and calcineurin inhibitors (ciclosporin (CsA) and topical tacrolimus) (Olivry et al., 2010). Allergen specific immunotherapy may also be helpful in reducing the recurrence of physical signs upon subsequent exposure to environmental allergens to which the patient is hypersensitive (Olivry et al., 2010).

Unfortunately, adverse effects of systemic GC, such as polyuria/polydipsia and polyphagia, are common, may be severe, and appear to be related to dosage, treatment duration and individual sensitivity (Olivry et al., 2010). Common adverse effects of CsA treatment include vomiting and diarrhoea although these are often transient and/or mild (Olivry et al., 2010).

One potential consequence of GC and CsA administration is that both drugs can negatively influence glucose metabolism. For example, it has been reported that GC treatment may be associated with an increase in the plasma concentration of glucose in man, dogs and cats (Munck, 1971; Issekutz et al., 1972; Owen et al., 1973; Campbell and Latimer, 1984; Jeffers et al., 1991; Lowe et al., 2009), which may be accompanied by increases in plasma insulin concentration that has been attributed to a compensatory response to either reduced peripheral insulin sensitivity or increased hepatic glucose production (Rizza et al., 1982). Pagano et al. (1983) reported that a standard anti-inflammatory dose of prednisone administered for 7 days induced insulin resistance characterised by depressed peripheral utilisation of glucose.

By contrast, CsA has been shown to inhibit insulin secretion during in vivo assessments of islet pancreatic β-cell function in

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