

SALIVARY IgA IN PATIENTS WITH PSORIASIS AND DERMATITIS HERPETIFORMIS

C. H. Oon, P. G. Goodwin, P. R. N. Kind, P. P. Seah and L. Fry

*From the Departments of Dermatology and Chemical Pathology,
St. Mary's Hospital, London, England*

Abstract. By means of a radial immunodiffusion technique, salivary IgA levels have been estimated in parotid gland saliva in 12 patients with dermatitis herpetiformis, 12 patients with psoriasis and in 12 healthy controls. Significantly elevated values were found in patients with psoriasis (mean $6.85 \pm \text{S.E. } 1.20 \text{ mg/100 ml}$) compared with the normal controls (mean $3.48 \pm \text{S.E. } 0.69 \text{ mg/100 ml}$) ($P < 0.05$). In dermatitis herpetiformis the values were not elevated (mean $3.84 \pm \text{S.E. } 0.50 \text{ mg/100 ml}$).

Abnormalities of serum immunoglobulin have been reported in dermatitis herpetiformis (D.H.) (6, 7). These authors found low serum IgM levels similar to those described in coeliac disease (C.D.) (8). By contrast, serum IgA levels have been reported to be elevated in D.H. (6) and similarly in C.D. by some authors, though others (3) found depressed levels. Despite the elevated serum IgA levels in C.D. and D.H., Beale et al. (1) have recently suggested that there is an impaired IgA response in patients with C.D., possibly related to an abnormality of secretory IgA.

We have, in this study, collected parotid saliva (a good source of IgA) to determine if any quantitative IgA abnormality could be detected in D.H. In addition, because of the reports (8, 16) of elevated serum IgA levels in psoriasis, we have studied salivary IgA in a group of patients with psoriasis.

METHOD

Twelve patients with psoriasis (age range 18-71 years) and 12 with D.H. (age range 25-70) were investigated. Twelve healthy individuals, mainly medical and nursing staff with no history of skin disease or oral infection (aged between 19-50 years) served as controls. Specimens of saliva (stimulated with lemon juice) were collected using Curby cups (4) into plastic containers and stored immediately at -20°C until processed.

Salivary IgA was measured by a modified single radial immunodiffusion technique (10) on commercially available immunoplates (Hyland Low Level Plates from Baxter Laboratories, Thetford, Norfolk). The immunoglobulin was measured by using low strength antisera to IgA in agarose plates. The wells were filled to the brim and allowed to empty completely before being refilled again in the same way. This procedure was performed three times altogether in all samples and suitably diluted standards, thus internally correcting any error resulting from refilling of the wells. The rings of the precipitate were then measured in all samples and standards and plotted on semilog graph paper. From the standard graph it was thus possible to read off the values for the patients' saliva. Salivary albumen levels were measured using a similar immunodiffusion technique.

RESULTS

The levels of salivary IgA in stimulated parotid secretion are shown in Fig. 1.

The mean salivary IgA was $3.48 \pm \text{S.E. } 0.69 \text{ mg/100 ml}$ in the control group and $3.84 \pm \text{S.E. } 0.50 \text{ mg/100 ml}$ in the D.H. group. This is not significantly different. In the patients with psoriasis the mean value was $6.85 \pm \text{S.E. } 1.20 \text{ mg/100 ml}$. This is a significant increase ($P < 0.05$) compared with the control group.

The albumen levels were in the normal range ($< 3 \text{ mg/100 ml}$) in all patients except one with psoriasis in whom it was 12 mg/100 ml . However, the salivary IgA level in this patient was not elevated (3.0 mg/100 ml).

DISCUSSION

These results show that in patients with psoriasis there is a significantly elevated level of IgA in parotid secretion. In patients with D.H. this level was found to be similar to controls. Stimulated

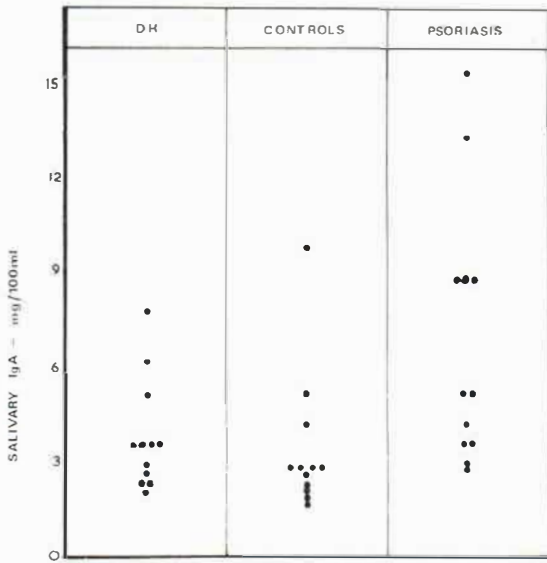


Fig. 1. Salivary IgA levels in dermatitis herpetiformis, psoriasis, and normal controls.

parotid salivary IgA was studied for various reasons. In secretions of the gastrointestinal tract, IgA is the major class of immunoglobulin present. The parotid gland has one of the highest concentrations of IgA plasma cells in the body and also produces higher levels of IgA than any other oral secretion (2). In addition, stimulated saliva provided a better frequency distribution (11). Our mean value of 3.84 mg/100 ml in patients with gluten-sensitive enteropathy (D.H.) is much lower than the 3–16 mg/100 ml range found by Douglas et al. (5) in untreated coeliac disease and <1–18 mg/100 ml in treated patients. However, as their controls had similarly raised levels this may reflect on their method of collection, as unstimulated specimens are known to have values of IgA as high as 25 mg/100 ml.

Our results in D.H. show normal salivary IgA levels and do not agree with those of McClelland et al. (9) who found elevated levels. Their mean salivary IgA level for 16 patients with D.H. was 10.9 ± 5.5 mg/100 ml compared with 7.0 ± 2.3 mg/100 ml in 16 controls, the difference being significant ($P < 0.05$). These authors also found elevated levels of IgM and IgA in the small intestinal secretions in D.H. and suggested that this finding, together with the possibility of raised IgA levels in saliva, might point to a widespread abnormality in the secretory immunoglobulin system in D.H.

patients. Further work is obviously necessary to further elucidate this point.

At present there is no evidence that psoriasis is an immunological disorder, whereas there is now considerable evidence that D.H. is (e.g. IgA deposits on the reticulin fibres in the skin (12, 15), and serum anti-reticulin and anti-nuclear antibodies (13)). Although elevated serum IgA levels have been reported in psoriasis (6, 16), no explanation for this finding has been put forward. The elevated salivary IgA reported here may be due to simple diffusion of 7 S IgA globulin (the predominant form in the serum.) However, Fraser et al. (6) found that the serum levels of IgA were raised to similar levels in both D.H. and psoriasis. It would therefore seem unlikely that the raised parotid IgA in psoriasis is due to simple diffusion. At the present time the site of production of the elevated serum IgA levels in D.H. and psoriasis is unknown and the modified radial immunodiffusion technique does not differentiate between 7 S and secretory 11 S IgA. Thus, we are unable to ascertain whether the elevated IgA in the parotid secretion is 7 S and due to simple diffusion, or 11 S as a result of increased production by the salivary gland. However, to exclude possible leakage of 7 S IgA into saliva due to unsuspected inflammation, we have measured the salivary albumen which is known to be increased in inflammation (14). This was only above normal in one patient with psoriasis and thus cannot explain our findings in the group of patients with psoriasis. It is obvious that techniques to distinguish between 7 S and 11 S globulin should be employed to define the type of IgA and thus demonstrate where the abnormality lies in psoriasis, as compared with normal persons.

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L. Fry, M.D.
Department of Dermatology
St. Mary's Hospital
Pread Street
London, W2 1NY
England