to the action of these exotoxins. Such a classification may also provide a practical basis for the administration of specific antimicrobial chemotherapy, convalescent gamma globulin, and future development of an effective antidote. Greater awareness and better management of toxin mediated haemolytic uraemic syndrome should ultimately improve its prognosis.

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Blizzard et al. found parathyroid specific antibodies in the sera of 46 of 74 (38%) of patients with idiopathic hypoparathyroidism, 25 of 93 (26%) of patients with idiopathic Addison’s disease, and in 6 of 245 (2.5%) of controls, using an indirect immunofluorescence technique. These figures have remained unconfirmed.

Irvine and Scarth studied sera from nine patients with idiopathic hypoparathyroidism and described an antibody to parathyroid oxyphil cells in one of these patients. Doniach and Bottazzo subsequently expressed the opinion that the oxyphil cell reactivity could be attributed to human specific mitochondrial antibodies. They further reported screening “normal, hyperplastic and adenomatous parathyroid glands with several hundred polycendric sera and hypoparathyroid cases.” They identified only three sera that reacted specifically with parathyroid chief cells; one of which was obtained from a patient with idiopathic hypoparathyroidism.

We wished to obtain parathyroid autoantibody positive sera for the purpose of antigen characterisation. In view of the high incidence of parathyroid autoantibodies found in idiopathic Addison’s disease, in Blizzard’s series, and the known rarity of adrenal autoantibodies in the normal population (<0.1%) we determined to obtain adrenal autoantibody positive sera for parathyroid autoantibody assessment.

Methods

Twenty six adrenal autoantibody positive sera were obtained from six British immunology centres. Normal control sera were obtained from healthy volunteers. We also obtained sera from two patients with idiopathic hypoparathyroidism. All sera were stored at –20°C until assay.

A standard indirect immunofluorescence technique was used to determine the presence of autoantibodies. Parathyroid autoantibodies were assessed on unfixed 5 µm cryostat sections of normal human parathyroid gland obtained at necropsy. All sera were applied to these sections, both undiluted and at a 1/5 (v/v) dilution. Five sera were additionally assessed on cryostat sections of a surgically resected parathyroid adenoma. Adrenal autoantibodies were confirmed and titrated on cryostat sections of normal human adrenal gland obtained at necropsy. Fluorescein isothiocyanate (FITC) conjugated sheep antihuman immunoglobulins G, A, and M (heavy and light chain) were applied as second antibody, and fluorescence was assessed using a fluorescence microscope. Negative and positive controls were included in each batch of sections tested. Negative controls comprised replacement of test serum by buffer, replacement of test serum by normal human control serum, and replacement of both the test serum and FITC conjugate by buffer. All unknown autoantibody positive serum is normally titrated simultaneously with each batch of sections as a positive control. In the absence of a known parathyroid autoantibody positive serum an antinuclear antibody positive serum was substituted for this control on parathyroid sections. This was titrated and gave a satisfactory assessment of the performance of the conjugate.

Results

None of the 26 adrenal autoantibody positive sera and neither of the sera from the two patients with idiopathic hypoparathyroidism showed specific reaction with normal human parathyroid tissue. Similarly, of the five adrenal autoantibody positive sera which had been additionally assessed on a human parathyroid adenoma, failed to show any evidence of the presence of parathyroid autoantibodies. The Table shows the results of titration of the adrenal autoantibody positive samples by indirect immunofluorescence.

Discussion

If 26% of patients with idiopathic Addison’s disease had parathyroid autoantibodies we would have expected to detect several patients with coexisting parathyroid and adrenal autoantibodies in our series. We found none, however, which suggests that such antibodies are rare. It may be argued that the differences between our findings and those of Blizzard could be based on patient selection. It is likely, however, that many of our patients had adrenal disease. This opinion is supported by two factors. First, adrenal antibodies are rare in the general population (<0.1%) and when present are often associated with the disease. Second, many adrenal antibody positive patients who are

<table>
<thead>
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<th>Titré of adrenal autoantibodies</th>
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<tr>
<td>1/160</td>
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</tr>
<tr>
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Do parathyroid and adrenal autoantibodies coexist?

The aetiology of idiopathic hypoparathyroidism remains unknown, although an autoimmune pathogenesis seems probable in some cases. Controversy exists over the prevalence of parathyroid autoantibodies in idiopathic hypoparathyroidism and in association with other autoimmune diseases.

References

clinically normal subsequently develop the disease. In a study by Betterle et al., four of nine adrenal autoantibody positive, non-Addisonian patients developed the disease within one to 31 months, and a fifth had reduced adrenocortical reserve.

Our results, therefore, suggest that the incidence of parathyroid autoantibodies in autoimmune adrenal disease is less than that originally observed. 1

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References

Effect of BPL on haemoglobin electrophoresis

It is the practice of this department to add the compound B-propiolactone (BPL) to whole blood or plasma from patients who

are HTLV-III positive. Previous workers, 1 2 have described the effect of BPL on several biochemical and haematological measurements.

During the laboratory investigation of a patient positive for HTLV-III with a sickle haemoglobin, a sample treated with BPL (Sigma Chemicals; final concentration 0.25%) gave a changed haemoglobin electrophoretic pattern, using cellulose acetate in Tris-edetic acid-borate (TEB) at pH 8.9 (Figure). This was also observed with treated normal samples and samples treated with another structural variant (Hb-C). Detection of abnormal haemoglobins was thus rendered impossible.

Further investigation showed that the Itano solubility test 3 for sickle haemoglobin and the sickle test (using sodium metabisulphite) 4 gave inconsistent results that were difficult to interpret. This could lead to false negative findings in patients with the sickle gene. Samples from such patients requiring investigation of a possible haemoglobinopathy should not be treated with BPL.

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References

The effect of BPL on haemoglobin electrophoresis (1) sickle cell trait control; (2) sickle cell trait sample treated with BPL; (3) sickle cell trait sample untreated; (4) normal sample treated with BPL; (5) normal sample untreated.