

Islet amyloid, increased A-cells, reduced B-cells and exocrine fibrosis: quantitative changes in the pancreas in type 2 diabetes.

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Morphometric analysis of the endocrine and exocrine pancreas was done on immunoperoxidase stained post-mortem tissue from 15 Type 2 diabetic and 10 age-matched control subjects. Thirteen of the 15 Type 2 diabetic patients had islet amyloid deposits (mean, 6.5% islet area) in the corpus (body, tail and anterior part of the head) but not in the caput (the "pancreatic polypeptide rich" part of the head) whereas none was seen in control subjects. In the corpus in diabetic subjects, the pancreatic area density of B-cells was decreased by 24% ($p = 0.005$) and A-cells increased by 58% (p less than 0.001) compared with control subjects. The mean A/B-cell ratio increased in the corpus from 0.27 in control subjects to 0.57 in Type 2 diabetic patients. Positive immunoreactivity for the amyloid constituent peptide, Diabetes Associated Peptide, was demonstrated in islet amyloid of diabetic subjects and in B-cells of control and diabetic subjects. The increase in A-cells may contribute to the hyperglucagonaemia and hyperglycaemia of Type 2 diabetes. The impaired insulin secretion in Type 2 diabetes may be due to a decrease in B-cells and to disruption of the islet structure by amyloid. Exocrine fat was similar in the control and diabetic subjects with both groups having more in the corpus than the caput. Diabetic subjects had increased exocrine fibrosis in the corpus region (p less than 0.001), but not in the caput. Exocrine fibrosis may be secondary to disordered islet cell function.