Prevalence and Clinicopathological Characteristics of Islet Amyloid in Chinese Patients With Type 2 Diabetes

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Islet amyloid has been suggested to be an important link between insulin resistance and β-cell dysfunction in type 2 diabetes. To investigate the prevalence and clinicopathological characteristics of islet amyloid, we examined consecutive autopsies of 235 Chinese patients with type 2 diabetes and 533 nondiabetic subjects. Islet amyloid deposits were identified using Congo red staining and quantitated by image analysis. We found that 3.0% of the nondiabetic subjects versus 39.6% of the diabetic patients displayed islet amyloid (P < 0.001). In diabetic patients, the amyloid deposits occupied a mean islet area of 36.2%, which was positively associated with BMI, blood pressure, and glycemic control. Pancreatic fibrosis and fat infiltration were more frequently found in diabetic patients with islet amyloid than those without islet amyloid, whereas pancreatic arteriosclerosis was identified in all diabetic patients. These findings suggest that islet amyloid deposits reflect greater insulin resistance and islet failure in a subgroup of type 2 diabetic patients. Islet failure may also have been exacerbated by fat infiltration, fibrosis, and arteriosclerosis. Optimal blood pressure and metabolic control may reduce these pathological changes and help preserve islet cell mass.

Type 2 diabetes is characterized by insulin resistance and progressive islet β-cell dysfunction (1,2). However, the link between insulin resistance and β-cell failure is unclear (2,3). Islet amyloid has been considered by many as a pathological hallmark of type 2 diabetes (4,5). In diabetic monkeys, the extent of islet amyloid is proportional to the degree of loss of islet mass and insulin secretion (6). In type 2 diabetic patients, islet amyloid is associated with severity of β-cell dysfunction (7). Islet amyloid is most diffuse and abundant in type 2 diabetic patients on insulin therapy (8,9). All these findings point to an intimate association between islet amyloid and β-cell dysfunction in type 2 diabetes (5,10,11).

Islet amyloid is a relatively common feature of type 2 diabetes across different ethnic groups, including Caucasians (11–13), Pima Indians (14), Asian Indians (15), and Japanese (16), with prevalence ranging from 50 to 100%. This wide range in reported prevalence may reflect true ethnic differences or sampling heterogeneity. However, most of these autopsy studies included <100 diabetic cases and thus might not have been truly representative of actual prevalence.

Furthermore, the clinicopathological characteristics of islet amyloid in type 2 diabetes have seldom been documented. An increase in islet amyloid deposits has been reported in Japanese type 2 diabetic patients with long disease duration and obesity (17). However, other autopsy studies have shown that islet amyloid developed independent of duration of diabetes over 2–40 years (11,18). Hence, the relation between islet amyloid and clinical phenotypes of type 2 diabetes remains to be clarified.

Because not all type 2 diabetic patients harbor islet amyloid, other underlying pathologies may also contribute to β-cell failure in type 2 diabetes. Pancreatic fibrosis, chronic inflammation, and fat infiltration have been described in type 2 diabetic patients (11,18–20). However, the relation between these pathological changes and islet amyloid has not been defined. Therefore, we conducted a multicenter study of 708 consecutive autopsies to estimate the prevalence of islet amyloid in Chinese type 2 diabetic patients and characterize the clinicopathological features of islet amyloid in type 2 diabetes.

RESEARCH DESIGN AND METHODS

Case selection. Over a 6-year period (1994–1999), 5,298 consecutive autopsies of adult Chinese subjects were performed at two medical centers in Beijing and one medical center in Hong Kong. Candidate cases were first identified through review of the autopsy databases in all three medical centers. Autopsy cases meeting the following criteria were included: 1) performance of a full autopsy within 24 h of death; 2) availability of clinical data on blood pressure, fasting plasma glucose, HbA1c, and fasting serum lipids (total cholesterol, triglycerides, and LDL and HDL cholesterol); and 3) availability of sufficient pancreatic tissue, with at least one representative block resected from the tail/body region. Cases were excluded if 1) pancreatic tissue had undergone autolysis, 2) diabetes was secondary to a known cause of hyperglycemia such as Cushing’s syndrome, or 3) patients had type 1 diabetes, defined by presentation with ketoacidosis, requirement of insulin therapy from disease onset, or the presence of autoimmune markers of islet β-cell.

The patients’ hospital records, including autopsy reports, were reviewed, and relevant data including BMI, glycemic and lipid measurements, and cause of death were obtained. The lipids were measured routinely using enzymatic methods. Cut-off values used for BMI were ≥23.0 kg/m2 to indicate patient

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was overweight and \( \geq 25.0 \text{ kg/m}^2 \) to indicate obesity in Asian patients (21). All diabetic subjects had been treated with oral antidiabetic drugs or insulin, whereas nondiabetic subjects were identified using a fasting plasma glucose \(<7 \text{ mmol/l} \) and absence of treatment with antidiabetic medications. Using these criteria, we selected 235 type 2 diabetic patients and 533 age- and sex-matched nondiabetic subjects with biochemical parameters and sufficient pancreatic tissues for comparison. The type 2 diabetic patients were further classified as islet amyloid positive or negative based on Congo red staining with polarization.

**Tissue sampling and staining.** Pancreatic tissues were taken at postmortem examination, fixed in 10% buffered formalin, and embedded in paraffin blocks. Tissue sections (6 \( \mu \)m) were cut from these paraffin blocks and stained by hematoxylin-eosin (H-E), periodic acid Schiff (PAS), and Congo red.

Congo red is a reliable, practical, and sensitive technique for detecting amyloid deposits containing islet amyloid polypeptide (IAPP) or amylin (22,23). IAPP is the building block of islet amyloid deposits (5,10,24). Although Congo red is not a specific marker for IAPP, it is as sensitive a marker for islet amyloid as IAPP immunolabeling (22). In this study, we identified islet amyloid using a Congo red technique (25), the specificity of which was further confirmed in thyroid medullary carcinoma, Alzheimer’s disease, and insulinoma (26). Briefly, all 6-\( \mu \)m thick pancreatic sections were treated with 1% sodium chloride-hydroxide solution for 20 min and stained with alkaline Congo red solution for another 20 min. All the Congoophilic amyloid deposits exhibited characteristic green/yellow birefringence under the polarizing microscope.

**Morphometry of islet amyloid.** For each patient, representative tissue slides for the pancreatic tail, body, and head were examined. The severity of type 2 diabetes—associated islet amyloid was assessed by the distribution, frequency, and degree of the amyloid deposits. The distribution described the localization of islet amyloid deposits in different pancreatic regions (i.e., pancreatic head, body, and tail). The frequency of islet amyloid was defined as the ratio of the number of amyloid-affected islets to the total number of islets in 10 randomly selected objective fields (magnification \( \times 20 \)). The degree of islet amyloid was quantitated on representative Congo red–stained sections from pancreatic tail/body at an objective magnification of \( \times 20 \) (Automatic Nikon Integrated Biological Imaging Microscope with Digital CCD Camera; Nikon, Tokyo, Japan; MetaMorph 4.0 image acquisition program for Windows 1999; Universal Imaging, Downingtown, PA). For each case, 10 fields of the section were randomly selected. Morphometric data were expressed as the proportion of area of Congo red–positive amyloid to the total pancreatic islet area examined.

**Statistics.** Data are expressed as means \( \pm \) SD or percent. Student’s \( t \) test was used to compare means (Statistics Package for the Social Sciences 10.0.7 for Windows 2000; SPSS, Chicago, IL). The association between two sets of parametric data was evaluated using the Pearson correlation coefficient. Differences in frequencies were assessed using the \( \chi^2 \) test (GraphPad InStat version 3.00 for Windows; GraphPad Software, San Diego, CA). A two-tailed \( P < 0.05 \) was considered significant.

**RESULTS**

**Prevalence of islet amyloid.** The prevalence of islet amyloid was higher in type 2 diabetic than in nondiabetic subjects (39.6 vs. 3.0\%; \( P < 0.001 \)). Islet amyloid deposits were identified in 93 of the 235 type 2 diabetic cases and in 16 of the 533 nondiabetic cases. All the 16 nondiabetic subjects with islet amyloid were older than age 60 years.

**Clinical demographic data.** The clinical demographic data of the 235 type 2 diabetic patients are depicted in Table 1. The mean BMI was 23.5 \( \pm 4.9 \text{ kg/m}^2 \) and 25.5% of the diabetic subjects were obese (BMI \( \geq 25 \text{ kg/m}^2 \)) (21). Of the 235 diabetic patients, \(-80\%\) were elderly (age \( >65 \) years).

The mean age of the 533 nondiabetic patients was 71.9 \( \pm 10.7 \) years (range 26–95 years). The male-to-female ratio was not significantly different between the diabetic and nondiabetic subjects (1.9 vs. 1.6; \( P = 0.3301 \)). Hypertension was noted in \(-60\%\) of the type 2 diabetic and nondiabetic subjects. The three leading causes of death in the type 2 diabetic and nondiabetic cases were cardioencephalovenous diseases (42.1 and 43.6%, respectively), cancers (22.1 and 20.2%), and respiratory diseases (9.8 and 11.6%). No significant differences in the major causes of death were found between patients with and those without islet amyloid (\( P > 0.05 \)).

**Clinical characteristics of islet amyloid in type 2 diabetes.** The clinical characteristics of islet amyloid in type 2 diabetes are summarized in Table 1. Patients with islet amyloid had greater mean BMI than those without islet amyloid (\( P = 0.019 \)). The prevalence of islet amyloid was 26.5, 45.2, and 58.3% in diabetic patients with BMI <23, 23–24.9 kg/m\(^2\), and \( \geq 25 \text{ kg/m}^2 \), respectively (\( P < 0.001 \)).

The type 2 diabetic patients with islet amyloid also had higher mean HbA\(_{1c}\) levels than those without islet amyloid (\( P < 0.001 \)). However, the mean fasting plasma glucose levels were not significantly different between the two groups (\( P = 0.669 \)). Compared with the type 2 diabetic patients without islet amyloid, the diabetic patients with islet amyloid had higher mean blood pressure and prevalence of hypertension, had a shorter duration of disease, and formed a larger proportion of male subjects (all \( P < 0.05 \) (Table 1). No differences were found in the mean age or lipid levels between the two groups (all \( P > 0.05 \)).

<table>
<thead>
<tr>
<th>( n )</th>
<th>Total patients with type 2 diabetes</th>
<th>Patients with islet amyloid</th>
<th>Patients without islet amyloid</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI (kg/m(^2))</td>
<td>23.5 ( \pm 4.9 )</td>
<td>24.2 ( \pm 4.9 )</td>
<td>23.1 ( \pm 4.9 )</td>
<td>0.019</td>
</tr>
<tr>
<td>Fasting plasma glucose (mmol/l)</td>
<td>12.8 ( \pm 4.8 )</td>
<td>12.6 ( \pm 4.1 )</td>
<td>12.9 ( \pm 5.2 )</td>
<td>0.669</td>
</tr>
<tr>
<td>HbA(_{1c}) (%)</td>
<td>9.6 ( \pm 2.2 )</td>
<td>11.7 ( \pm 2.3 )</td>
<td>8.6 ( \pm 2.2 )</td>
<td>(&lt;0.001 )</td>
</tr>
<tr>
<td>Known duration of diabetes (months)</td>
<td>128 ( \pm 41 )</td>
<td>118 ( \pm 46 )</td>
<td>135 ( \pm 38 )</td>
<td>0.004</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>57.9</td>
<td>66.7</td>
<td>52.1</td>
<td>0.038</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>146 ( \pm 36 )</td>
<td>149 ( \pm 29 )</td>
<td>144 ( \pm 39 )</td>
<td>0.042</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>78 ( \pm 17 )</td>
<td>80 ( \pm 15 )</td>
<td>77 ( \pm 19 )</td>
<td>0.039</td>
</tr>
<tr>
<td>Total cholesterol (mmol/l)</td>
<td>4.7 ( \pm 1.1 )</td>
<td>4.8 ( \pm 0.9 )</td>
<td>4.6 ( \pm 1.2 )</td>
<td>0.070</td>
</tr>
<tr>
<td>Triglyceride (mmol/l)</td>
<td>1.9 ( \pm 1.4 )</td>
<td>1.9 ( \pm 0.9 )</td>
<td>2.4 ( \pm 1.7 )</td>
<td>0.660</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/l)</td>
<td>2.5 ( \pm 0.4 )</td>
<td>2.6 ( \pm 0.5 )</td>
<td>2.5 ( \pm 0.3 )</td>
<td>0.133</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/l)</td>
<td>1.0 ( \pm 0.7 )</td>
<td>1.2 ( \pm 0.6 )</td>
<td>0.9 ( \pm 0.8 )</td>
<td>0.595</td>
</tr>
</tbody>
</table>

Data are \( n \) or means \( \pm \) SD.
Histopathological characteristics of islet amyloid in type 2 diabetes. The pancreatic histopathological features associated with islet amyloid in type 2 diabetes are shown in Table 2. Islet amyloid was often accompanied by pancreatic fibrosis, fat infiltration, and arteriosclerosis (Fig. 1).

Type 2 diabetic patients displayed a full spectrum of arteriosclerotic changes (Fig. 2). Changes in arterioles and small arteries, included hyaline arteriolosclerosis with subendothelial exudation, intimal foam cell accumulation, and calcification (Fig. 2). Atherosclerotic lesions in pancreatic large arteries showed prominent intimal lipid accumulation in the form of foam macrophages and cholesterol crystals (Fig. 2). Some arteries were obliterated by atheroembolism (Fig. 2). The prevalence of arteriosclerotic changes was slightly, albeit not significantly, higher in pancreases with than without islet amyloid.

Both fat infiltration and fibrosis were more common in diabetic pancreases with than without islet amyloid (all $P < 0.01$) (Table 2). Pancreatic fat infiltration was detected in 50.6% of the diabetic patients. Adipocytes were predominantly distributed in the exocrine acini and occasionally found in islets (Fig. 3). Fibrotic tissue was distributed irregularly throughout the diabetic pancreases, but extensive fibrosis often occurred in areas surrounding islets with amyloid, ducts, and arteries with hyaline changes (Fig. 4). Chronic pancreatitis was found in 7.7% of the diabetic patients, in whom diffuse pancreatic fibrosis was usually accompanied by acinar atrophy, chronic inflammatory infiltrates, and islet amyloid (Fig. 5).

Severity of islet amyloid in type 2 diabetes. The severity of type 2 diabetes-associated islet amyloid was assessed by the distribution, frequency, and degree of the amyloid deposits. The distribution of islet amyloid deposits was not uniform; 88.6% (62/70) of the diabetic patients showed islet amyloid in more than one anatomical region of the pancreas. Among the diabetic subjects with islet amyloid detected in only one anatomical region of the

TABLE 2
Association of islet amyloid with pancreatic histopathology in type 2 diabetic patients

<table>
<thead>
<tr>
<th></th>
<th>Total diabetic pancreases</th>
<th>Pancreases with islet amyloid</th>
<th>Pancreases without islet amyloid</th>
<th>$P$</th>
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<tr>
<td>$n$</td>
<td>235</td>
<td>93</td>
<td>142</td>
<td>—</td>
</tr>
<tr>
<td>Arteriosclerosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyaline arteriolosclerosis</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>—</td>
</tr>
<tr>
<td>Pancreatic atherosclerosis</td>
<td>34.0</td>
<td>40.9</td>
<td>29.6</td>
<td>0.077</td>
</tr>
<tr>
<td>Arterial calcification</td>
<td>31.9</td>
<td>31.2</td>
<td>32.3</td>
<td>0.959</td>
</tr>
<tr>
<td>Atheroembolism</td>
<td>13.2</td>
<td>12.9</td>
<td>13.4</td>
<td>0.927</td>
</tr>
<tr>
<td>Pancreatic fat infiltration</td>
<td>50.6</td>
<td>62.4</td>
<td>43.0</td>
<td>0.005</td>
</tr>
<tr>
<td>Pancreatic fibrosis</td>
<td>57.9</td>
<td>76.3</td>
<td>45.8</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>Chronic pancreatitis</td>
<td>7.7</td>
<td>7.0</td>
<td>8.6</td>
<td>0.803</td>
</tr>
</tbody>
</table>

Data are $n$ or %. $P$ values were determined using the $\chi^2$ test.
pancreas, four had islet amyloid in the pancreatic body and another four had islet amyloid in the tail region. None of the patients showed islet amyloid limited to the pancreatic head.

Islet amyloid was present in 40.4% (67/166) of the diabetic patients for whom representative tissue sections were available from all three regions of the pancreas (head, body, and tail). The prevalence was not significantly different from that of the diabetic cases for whom one or two representative tissue sections were available (40.4 vs. 37.7%; P = 0.813). Among the 93 diabetic cases with islet amyloid, 67 had representative sections from all three pancreatic divisions. In these 67 cases, islet amyloid was identified in the tail in 97.0% (65/67), in the body in 92.5% (62/67), and in the head in 58.2% (39/67). The highest probability of detecting islet amyloid was in the pancreatic tail and the lowest was in the pancreatic head (P < 0.001).

The frequency and degree of islet amyloid were variable in the 93 type 2 diabetic pancreases positive for amyloid. Most showed diffuse islet amyloid with extensive fibrosis and acinar atrophy (Fig. 4). The frequency of islet amyloid ranged from 2.6 to 91.8%, with a mean percentage of 38.8 ± 31.0%. In only 25.8% (24/93) of the diabetic pancreases, 50% or more islets were positive for amyloid (Fig. 4). The degree of islet amyloid also varied from 1.4 to 97.2%. The mean area fraction of islet amyloid was 36.2 ± 17.0%, indicating approximately a 33% reduction in mean islet cell area in the diabetic patients with islet amyloid. The degree and frequency of islet amyloid were positively correlated to BMI, HbA1c, and systolic and diastolic blood pressure in the 93 type 2 diabetic patients (Table 3). No significant correlation was found between the islet amyloid load and the known duration of type 2 diabetes, age of patient, or fasting plasma glucose and serum lipid levels (Table 3).
We have reported the prevalence and clinicopathological characteristics of islet amyloid in Chinese patients with type 2 diabetes. In this multicenter autopsy study, islet amyloid occurred in \(rac{4}{10} \) of the Chinese patients with type 2 diabetes who were on antidiabetic treatments, and the amyloid deposits occupied a mean islet area of 36.2% in affected patients. The type 2 diabetes–associated islet amyloid was positively associated with BMI, blood pressure, glycemic control, pancreatic fibrosis, and fat infiltration. Our findings suggest that islet amyloid deposits may reflect greater insulin resistance and β-cell failure in a

**FIG. 3.** Fat infiltration in pancreate of type 2 diabetic patients. Adipocytes (a) are shown in exocrine acini (A; H-E, magnification \( \times 100 \)), islet (B; PAS, magnification \( \times 400 \)), adjacent to islet amyloid deposits in red color (C; Congo red, magnification \( \times 400 \), ordinary light), or green/yellow birefringence (D; Congo red, magnification \( \times 400 \), polarizing light).

**FIG. 4.** Diffuse fibrosis (white birefringence; arrows) and islet amyloid deposits (green/yellow birefringence; asterisks) in pancreate of type 2 diabetic patients. Congo red magnification \( \times 200 \), polarizing light microscope.
subgroup of type 2 diabetic patients (27,28). In agreement with other reports, this study also confirmed the phenotypic heterogeneity of type 2 diabetes and the association of islet amyloid deposits with loss of islet cells (8,11,29).

A limitation of our study is that not all selected autopsy cases had representative sections from all three regions (head, body, and tail) of the pancreas. This approach may have predisposed us to underestimate islet amyloid deposits (8). However, most (88.6%) of the diabetic patients had islet amyloid in more than one anatomical region of the pancreas. Only ~3% of pancreases contained amyloid in <1% of the islets (8,30). The prevalence of islet amyloid in the diabetic patients for whom representative sections were available from all three divisions of the pancreas was not significantly different from that of the diabetic cases for whom one or two representative tissue sections were available (40.4 vs. 37.7%; P = 0.813). To minimize the bias of underestimation, we included autopsy subjects who had at least one representative tissue block resected from the β-cell-rich region (tail/body) of the pancreas (22,29).

Islet amyloid deposits are present in only a portion of patients with type 2 diabetes. Because of differences in sample size and diagnostic criteria, the prevalence of islet amyloid reported in type 2 diabetic patients varies substantially (4,5,10). For example, the widely cited study by Westermark and Wilander (31) showed that islet amyloid was found in 100% of their sample of only 12 Caucasian patients with adult onset of diabetes. However, the prevalence of islet amyloid in the nondiabetic subjects, defined as such by the absence of clinical signs of diabetes and one or several negative tests for glucose in the urine in the same study, was 60% (9/15) (31). In an early study, Bell (12) reviewed over 1,000 autopsy cases and found islet amyloid in 44% of diabetic subjects over age 40 years. Using more objective diagnostic criteria, we found islet amyloid in 39.6% of type 2 diabetic patients and 3.0% of nondiabetic subjects. These data indicate that islet amyloid occurs only in a subgroup of type 2 diabetic patients.

In this study, type 2 diabetic patients with islet amyloid displayed greater insulin resistance (presumed because of higher BMI and blood pressure) and insulin deficiency (higher HbA1c). The islet amyloid load has been reported to be proportional to the severity of type 2 diabetes (6,8). In our study, islet amyloid deposits occupied a mean islet area of 36.2%, and the frequency and degree of islet amyloid was correlated with HbA1c in 93 patients with type 2 diabetes. In type 2 diabetic patients, there was an inverse relation between the degree of islet amyloid and intracellular IAPP immunoreactivity (22). Several in vitro and in vivo (transgenic mouse) studies have revealed that small amyloidogenic human IAPP aggregates induce β-cell death and, subsequently, insulin deficiency (4,29,32–34).

All these data imply that the formation of extracellular islet amyloid deposits and the loss of β-cells may be associated through a common shared precursor, amyloidogenic human IAPP aggregates (35). These aggregates

![FIG. 5. Chronic pancreatitis characterized by broad-band fibrotic tissues, extensive acinar atrophy, inflammatory infiltrates, and islet amyloid deposits (asterisk). H-E, magnification x100.](image)

<table>
<thead>
<tr>
<th>Known duration of diabetes</th>
<th>Frequency</th>
<th>Degree</th>
<th>r</th>
<th>P</th>
<th>r</th>
<th>P</th>
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may cause β-cell death by disrupting plasma membranes or/and causing oxidative stress (33,36) while they grow as extracellular islet amyloid deposits (35).

On the other hand, islet amyloid might be a result of obesity-associated insulin resistance (5). We found the area of islet amyloid deposits correlated significantly with BMI and blood pressure. Aging-related weight gain in normal rats and genetic obesity in Wistar fatty rats are associated with elevated IAPP secretion (37). Although hemizygous transgenic mice expressing human IAPP in pancreatic β-cells have no diabetic phenotype, type 2 diabetic phenotypes such as islet amyloid deposition and decreased β-cell mass occurred when the hemizygous mice were crossed with an obese, insulin-resistant strain such as agouti viable yellow (38). In support of these experimental studies, patients with obesity and hypertension have high basal and stimulated levels of plasma IAPP (39,40). Taken together, it is plausible that insulin resistance may cause intracellular IAPP amyloidogenesis and, subsequently, β-cell death and extracellular amyloid deposits (29,33). In contrast to our findings, a recent autopsy study in 124 Caucasians revealed that islet amyloid load (presence, frequency, and extent) was not increased in type 2 diabetic patients with BMI ≥27 kg/m² compared with lean patients with BMI <25 kg/m², nor was it increased in obese subjects with impaired glucose tolerance compared with either obese or lean nondiabetic subjects (29). This discrepancy may reflect ethnic or phenotypic differences between the study populations.

Apart from amyloid deposits, β-cell failure in type 2 diabetes may also result from other pancreatic histopathologies, such as pancreatic fat infiltration and fibrosis (19,20,31). In the current study, >50% of the pancreases exhibited extensive fibrosis, fat infiltration, and severe arteriosclerosis in either exocrine acini or islets. Moreover, the frequency of fat infiltration and fibrosis in diabetic pancreases was higher in those with islet amyloid. Pancreatic arteriosclerosis and atherosclerosis were as common in diabetic patients with or without islet amyloid (41). These observations suggest that pancreatic remodeling induced by arteriosclerosis, islet amyloidosis, fat infiltration, fibrosis, and chronic inflammatory changes might cause islet and β-cell failure in type 2 diabetes (19,41).

In summary, the prevalence of islet amyloid was ~40% in autopsies of Chinese patients with type 2 diabetes. On average, islet amyloid deposits occupied 36% of islet area in these patients. The presence of islet amyloid was associated with greater BMI, higher blood pressure, shorter duration of disease, and worse glycemic control in diabetic patients. Islet amyloid was also frequently accompanied by pancreatic arteriosclerosis, fibrosis, and fat infiltration. Optimal control of blood pressure and metabolic indexes may prevent some of these pathological changes, reduce loss of islet cells, and preserve β-cell function in type 2 diabetes.

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Underwater Nepturens