Spring 1983

Impotence And Diabetes: The Vascular Component

S. Diane Lund  
Carroll College

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IMPOTENCE AND DIABETES:
The Vascular Component

Submitted in Partial Fulfillment of the Requirements for Graduation with Honors to the Department of Biology at Carroll College, Helena, Montana

S. Diane Lund
March 22, 1983
This thesis for honors recognition has been approved for the Department of Biology.

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ABSTRACT

Alloxan induced diabetes produced angiopathic changes in the corpora cavernosa. These changes induced thickening of the tunica albuginea, deterioration of the arteries supplying the cavernous bodies & thickening of endothelial lining of intact arteries in the cavernosa. The vascular component and the limited breeding experiment conducted here suggests a possible correlation between vascular changes and impotency in male diabetic rats.
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INTRODUCTION

Impotence is a widespread problem among diabetic males. Upwards of 40% of diabetic men suffer from impotence (9). The prevalence of impotence with increasing age in diabetic men of 20-64 years is still approximately 18-71%, which is considerably greater than 0.1-18.4% for the corresponding normal male population (2). As many as 6 out of every 10 males with diabetes mellitus may be impotent (17). Recent studies have found impotence in males with severe microangiopathy, as demonstrated by proliferative retinopathy and symptomatic autonomic neuropathy.

Past studies on diabetic men have found a vascular and/or neural component to their impotence. This paper is concerned with the vascular component. Histological studies were made of the pancreas and the penis in a diabetically induced male rat. Effects of alloxan induced diabetes on the corpora cavernosa and associated arteries were observed. Possible correlations to the human problem of diabetic impotence and plans for further research will be presented.
LITERATURE REVIEW

Diabetes is a complex disease, showing many different kinds and degrees of pathology. Susceptibility to diabetes has a large genetic component. Physical exercise and the quality of the diet have an effect on its incidence.

The primary symptom of acute diabetes mellitus is hyperglycemia, often accompanied by glucosuria and polyuria. Correspondingly, there is considerable hunger, thirst, and weight loss. Changes include degeneration of the walls of blood vessels, especially of the fine capillaries and their basement membranes. Many different organs are affected by these vascular changes.

Alloxan destroys the pancreatic islet tissue in animals (monkeys, rabbits and rats). The basic deficiency in glucose metabolism is a matter of impaired transport of blood glucose across the membranes of skeletal muscles and other tissues (7). This transport impairment is the major defect in glucose metabolism of diabetic animals, but there is also an enhanced rate of gluconeogenesis. Another impairment in diabetes is the almost complete cessation of the conversion of glucose to fatty acids via acetyl-CoA. The acceleration of gluconeogenesis
from amino acids and the inhibition of fatty acid synthesis from glucose shows that metabolism in the diabetic organism is tuned to maintain as high a concentration of glucose in the blood as possible, even if the blood level may already greatly exceed the renal threshold for glucose. The constant loss of glucose in the urine in diabetics (at the expense of ingested amino acids and body proteins), accounts for the constant hunger and loss of body weight (7).

In diabetic animals relatively little glucose is oxidized as fuel, except by the brain. The rest of the tissues burn a large amount of fat, especially in the liver, where the amount of acetyl-CoA formed from fatty acids exceeds the capacity of the tricarboxylic acid cycle to oxidize it. The excess acetyl-CoA is converted into ketone bodies, large amounts of which may appear in the blood (ketonemia) and urine (ketonuria), a condition known as ketosis (7).

Insulin is the main regulator of the impairments. Administration of insulin reverses the effects of diabetes. It increases the rate of removal of glucose from the blood to normal blood glucose concentration. It increases the amount of gluco-kinase, phospho-fructokinase, and pyruvate-kinase in the liver. It depresses the biosynthesis of enzymes specifically required in gluconeogenesis (i.e., depresses the formation of glucose from amino acids). It restores the normal rate of conver-
sion of glucose into fatty acids, and inhibits the oxidative degradation of fatty acids. As a result, the blood sugar level becomes normal, glucosuria ceases, and the ketone bodies recede to their normal levels in the blood and urine (16).

Deficiency of insulin in diabetes can theoretically be due to a number of different causes, such as a reduced amount of otherwise normal islet tissue, defective synthesis of proinsulin, defective conversion of proinsulin to insulin, deficient release of insulin into the blood in response to increased blood glucose, production of a genetically defective insulin, or an abnormally high rate of destruction of insulin (7).

Alloxan provides a quick and convenient method for producing experimental diabetes. Three phases are generally observed after the administration of alloxan: transitory periods of hyperglycemia and hypoglycemia, followed by permanent hyperglycemia and other diabetic symptoms. Alloxan is thought to act directly and specifically on the beta-cells, causing them to undergo degeneration and resorption, the alpha-cells and the acinar tissue remain relatively unaffected. Among mammals, alloxan diabetes has been studied most extensively in rats, mice, hamsters, and guinea pigs (16).

There are certain conspicuous differences between the diabetes produced by alloxan and that produced by removal of the pancreas. In alloxan diabetes, the hyper-
glycemia and glycosuria are generally more severe than in total pancreatectomized animals. Alloxanized animals survive longer without insulin than do pancreatectomized animals, and ketonemia and ketonuria may be mild and transitory. It may be that some of these differences result from the fact that the alloxanized animal is not deprived of glucagon from the alpha-cells, whereas the totally pancreatectomized animal lacks both insulin and glucagon (16).

The source of endogenous fuels and the pattern of their distribution and consumption in severe diabetics is shown in Figure 1. The figures are for a 24 hour period under basal conditions, assuming total energy output of 2400 kcal. Note the heavy drain on muscle proteins for gluconeogenesis and the utilization of body triacylglycerols as an energy source for all organs except the brain and blood cells, which require glucose. A large part of the glucose made from muscle is lost in the urine (7).

Why is it that diabetics not only have a defect in the tissue utilization of glucose but also appear to be metabolically poised to produce maximal amounts of glucose from amino acids and to prevent glucose from being utilized to form fat? The hypothesis that the enhanced gluconeogenesis and the diminished fat synthesis are biological compensations for the lack of insulin. The concentration of glucose in the blood is increased
FIG. 1. The source of endogenous fuels for the severe diabetic
so that the deficiency in glucose transport can be overcome, to permit the utilization of glucose by peripheral tissues in the absence of insulin (7).

Today most of the morbidity and mortality from diabetes mellitus results from a particular type of small blood vessel disease commonly referred to as diabetic microangiopathy. Diabetic microangiopathy is usually thought of as the well known triad of retinopathy, neuropathy, and nephropathy. Ischemic heart disease and peripheral vascular disease were also quite common (Table 1). Note the fairly high percentage of peripheral vascular disease in both groups (11).

Thickening of the basement membrane is considered to be the basic pathologic change of diabetic microangiopathy. Siperstein et al in 1968 used a morphometric method to measure the basement membrane thickness of quadricep muscle capillaries. They showed that greater than 90% of adults (over 19 years of age) with diabetes had thickened capillary basement membranes as compared to a group of normal glycemic controls who had a negative family history for diabetes. In addition, they showed that in 30 genetic prediabetic adults (offspring of 2 overt diabetics) whose glucose tolerance was normal, 53% had thickened capillary basement membranes as compared to the control group. In these studies there was no correlation between severity of duration of the diabetes and the thickness of the basement membrane (Table 2)(11).
TABLE 1. Vascular complications in diabetes after 40 yr (Raskin, 11)

<table>
<thead>
<tr>
<th></th>
<th>BOSTON</th>
<th>LONDON</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td>73(32M,41F)</td>
<td>92(32M,60F)</td>
</tr>
<tr>
<td>Therapy</td>
<td>insulin</td>
<td>insulin</td>
</tr>
<tr>
<td>Complications (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Retinopathy</td>
<td>75.3</td>
<td>60.8</td>
</tr>
<tr>
<td>Nonproliferative type</td>
<td>45.0</td>
<td>43.4</td>
</tr>
<tr>
<td>Proliferative type</td>
<td>17.8</td>
<td>10.8</td>
</tr>
<tr>
<td>Proliferative type (blind)</td>
<td>12.3</td>
<td>6.5</td>
</tr>
<tr>
<td>Nephropathy</td>
<td>41.0</td>
<td>8.6</td>
</tr>
<tr>
<td>Proteinuria</td>
<td>28.7</td>
<td>6.5</td>
</tr>
<tr>
<td>Proteinuria &amp; renal failure</td>
<td>8.2</td>
<td>2.1</td>
</tr>
<tr>
<td>Neuropathy</td>
<td>48.0</td>
<td>16.3</td>
</tr>
<tr>
<td>Ischemic Heart Disease</td>
<td>20.5</td>
<td>45.6</td>
</tr>
<tr>
<td>Peripheral Vascular Disease</td>
<td>40.0</td>
<td>44.5</td>
</tr>
</tbody>
</table>
TABLE 2. Quadricep capillary basement membrane width in normal, diabetic and prediabetic subjects (Raskin, 11)

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Average Basement Membrane Width (\text{Å})</th>
<th>Prevalence of Basement Membrane Thickening (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (50)</td>
<td>1080 ± 27</td>
<td>8</td>
</tr>
<tr>
<td>Diabetic (51)</td>
<td>2403 ± 119</td>
<td>98</td>
</tr>
<tr>
<td>Prediabetic (30)</td>
<td>1373 ± 44</td>
<td>53</td>
</tr>
</tbody>
</table>

FIG. 2. Percentage of impotent diabetic subjects in different studies (modified from Koncz et al., Medical Times, Aug. 1970, p. 160)
Kefalides and Westberg in 1974 studied the changes in the diabetic basement membrane. They found slight increases in the amounts of hydroxylysine, hydroxyproline, and the glucoslygalactose disaccharide units and found decreases in cystine. Since it is the disulfide bonds that form the structural cross-linkages in basement membranes, decreases in cystine could explain the observed changes in permeability of diabetic basement membranes. Those changes include making the diabetic basement membrane more permeable to large macromolecules (11).

Increased vascular permeability in diabetes produces extravasation of lipoproteins into the walls of the vessels. This increased permeability plays a role in the development of atherosclerotic vascular changes. Since diabetes is a systemic disease, few organs are spared the atherosclerotic changes. Small arteries are affected first. This applies to the penile arteries as well (3).

Impotence usually manifests itself as a later complication, after many years of diabetes. Sometimes, however, impotence is found at the onset of diabetes and may even be the presenting symptom. Figure 2 illustrates the percentage of impotent diabetic subjects in different studies (5).

The term "impotence" properly refers only to erectile difficulties. It is defined as a pattern of persistent
or recurrent inability to develop or maintain an erection of sufficient rigidity for successful coitus. Impotence develops gradually with a cumulative effect. Erections take longer to develop, with full-strength erections a rarity. This decreased tumescence leads to incomplete, partial erections, barely sufficient for intromission. The condition may stabilize, but eventually, a progression towards total impotence with inability to develop erections under any circumstance occurs (9).

Impotence in male diabetics is usually assumed to be neuropathic. It is known that in diabetics, the main pathologic changes are microangiopathy, atherosclerosis, and nephropathy, as well as neuropathy. Since penile erection is a neurologically mediated hemodynamic event, it logically follows that both vascular and neurologic destruction can lead to impotence. Dispute exists over the relative weight to be given to either component in the production of diabetic impotence.

Melman et al, in 1980, examined histologically, samples of erectile tissue taken from the corpora cavernosa of 16 impotent diabetics. They reported that myelinated and unmyelinated nerve fibers were intact and unaltered in either insulin-dependent or diet-controlled patients (10). Elleberg and Weber stated in 1966, "It would appear that retrograde ejaculation does not occur frequently in diabetics and is seen in 1-2% of cases. Ejaculation is under control of the autonomic nervous
system, yet almost 98% of impotent diabetics have normal ejaculations despite the assumption that the autonomic nervous system is affected (3).

Regulation of blood flow to the cavernous bodies is one of the basic functions of the penile artery and its branches and is of critical importance in erection. The morphologic mediators of this function are pads first described by Ebner in 1900. Their importance for the release of arterial flow was stressed by Corti in 1952. The action of these pads is as follows: at rest the longitudinal muscle fibers are contracted, and the pad bulges into the lumen, creating a decreased rate of blood flow. Erection involves a relaxation of both circular and longitudinal fibers in the media and of the longitudinal fibers of the pads. These changes allow an increase in blood flow into the cavernous bodies and an elongation of the arteries to accommodate for the erect state. Any change or pathology of this muscle system in the pads therefore results in a partial block of blood flow in the involved artery. This is further complicated by fibrotic intimal proliferation, fibrotic changes in the media, and calcification. The resulting arterial bed of the penis is then only capable of supplying blood to maintain the organ, but not to erect it (13).

Ruzbarsky and Michal in 1977 used microscopy to study the arterial tree of the penis postmortem in a
sample of 30 men, 15 of whom had had diabetes for an average of 30 years. In all men over 38 years of age, he demonstrated fibrosis of the Ebner pads and generalized intimal fibrosis, medial fibrosis, calcification and luminal narrowing to the point of obliteration from thrombi. These changes were considerably accelerated in the diabetic group (13).

A prerequisite for normal erection is an adequate blood supply to the genital region as seen by the occurrence of impotence in the Leriche syndrome (a vascular disease). Fournier and Huget in an unconfirmed angio-pathic study of diabetics, have claimed an absence of blood supply to the corpora cavernosa of the penis (8). Gaskill in 1971 demonstrated penile vascular obstruction in 7 of 8 diabetic men with known peripheral disease (2). Conti in 1952 stated that erection is produced by 3 steps: a shunt of blood to the cavernous spaces, a decrease in blood flow in the external tissue of the penis, and the trapping of blood flow in the erectile tissue (1). Ruzbarsky and Michal in 1977 showed that arterioschlerotic changes in the arterial vascular bed of the penis lead to decreased flow of blood into the lacunae of the cavernous bodies at the time of erection. The extent of pathology was related to age and the presence of diabetes mellitus (Figure 3) (13).
FIG. 3. The relationship of age and impotence in normal and diabetic men
MATERIALS AND METHODS

Five male and five female rats were obtained from Carroll College. The rats were from 1 to 3 months old. They received standard Purina Rat Laboratory Chow and water.

Three male rats were injected intraperitoneally with a 10% alloxan solution. The solution was made using 0.5g alloxan and 5 ml of saline with pH adjusted to 5 using HCl. The dose of alloxan was 0.2 ml of the 10% solution per 100g body weight. The total dose was administered as five smaller intraperitoneal injections of 0.04 ml/100g body weight given one each day for five days.

In order to demonstrate alloxan diabetes, a blood sample was taken by cardiac puncture using light nembutal anesthesia. The rats were fasted 24 hr before the blood sample was taken. Two to three ml of blood was taken each time the test was performed. The blood was placed into 5 ml tubes containing dried heparin. The tubes were centrifuged at 746g for 20 min to obtain the blood plasma. Plasma glucose was determined colorimetrically with Sigma test reagents (glucose oxidase and peroxidase). The blank contained 0.5 ml distilled water and
the standard contained 0.5 ml of a 20-fold dilution of a Sigma Glucose Standard Solution (100 mg/dl). Along with the plasma samples, the blank and the standard were combined with 5 ml of the combined enzyme color reagent. All tubes were incubated in a water bath at 37° C for 30 ± 5 min. The tubes were removed and absorbance was read at 450 nm. The test volumes were calculated as follows:

\[
\text{Serum glucose (mg/100ml)} = \frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times 100
\]

Once the rats were shown to be hyperglycemic by an increase from normal glucose levels (150 mg/100 ml), blood was drawn weekly to monitor the blood glucose level. Weight was also monitored. The three diabetic rats maintained a high level of blood glucose, 270-290 mg/100 ml, for about one month, then a sharp increase was noted. At this time to keep the rats alive, an isophane insulin suspension was administered. The dosage was 4 Units of insulin per day intramuscularly.

Two diabetic rats were also used for a mating experiment and were mated with two female rats. The two pairs were left in separate cages for 2 wk. These same females were mated later with the two normal control rats. Litter production in these females was used as an index of male potency.

The three diabetic and two control rats were sacrificed by carbon dioxide asphyxiation. Preparation of
slides for observation involves a series of steps. The extracted tissues (penis and pancreas) were immediately placed in 10% formalin. Fixation, dehydration and paraffin infiltration were all accomplished by the use of the Autotechnicon. The tissues were then embedded in paraffin embedding blocks. Sections of tissue six micrometers in thickness were cut and adhered to standard glass microscopic slides using unflavored gelatin. The slides were placed in a warming oven to remove excess paraffin. Two staining procedures were employed using duplicate sets of slides. The duplicate sets were prepared by making 2 slides from the same ribbon of tissue and using consecutive sections for each slide. Hematoxylin and eosin (H-E) was used as a primary stain to select the more representative slides for specific stainings. The slides that exhibited the least number of beta-cells by H-E staining indicated their duplicates to be stained with Gomori's Aldehyde Fuschin (GAF). This stain displays the alpha-cells and the acinar tissue in a magenta red and the beta-cells in a violet blue.

In the same manner, control slides were prepared from tissue taken from non-diabetic male rats. Here the duplicate slides subjected to GAF staining were those showing the highest number of beta-cells by the H-E method.
OBSERVATIONS AND RESULTS

Alloxan diabetes was accomplished in 3 male rats. Blood glucose levels and weight were monitored for the diabetic (Fig. 4) and control (Fig. 5) rats.

Treatment with insulin was not undertaken until approximately 5½ wk into their diabetic condition. A sharp rise in their blood glucose level and a decrease in weight (Fig. 4) was observed and interpreted as a possible fatal condition. To counteract this, insulin was administered and blood glucose levels were monitored.

Histological Observations:

The two mated diabetic rats were sacrificed and 40 slides of their pancreatic tissue were stained with H-E. Thirty of those slides showed extensive beta-cell destruction, 10 slides had damaged tissue or too thick sections. Another set of 30 slides of diabetic pancreatic tissues were stained with GAF to help quantify the destruction. Eight slides did not stain adequately; of the 25 remaining slides, a total of 50 islets, chosen randomly, were observed. Fifteen islets had no beta-cells, 10 had fewer than 5 beta-cells and 5 had no more than 15.
FIG. 4 Diabetic rat weight and blood glucose level charts
FIG. 5. Control rat weight and blood glucose level charts
Two non-diabetic control animals were sacrificed and 40 slides of their pancreatic tissue were stained with H-E. Thirty-five of these slides were good representatives of a control pancreas, five had damaged tissue. Another set of 35 slides of pancreatic tissue were stained with GAF to quantify the presence of beta-cells. Ten slides did not stain well. Of the 25 remaining slides a total of 50 islets, chosen randomly, were observed. Forty islets had 15 or more beta-cells per islet, only 10 had fewer than 15 beta-cells per islet and no islets were observed to have no beta-cells.

Fifty slides of the diabetic and control rat penile tissue were stained with H-E. Minor changes were observed when these two were compared. A thickening of the tunica albuginea surrounding the corpora cavernosa was noted. Deterioration of the small arteries in the cavernous bodies was observed as a separation of the muscular wall surrounding the artery and small holes in the arterial wall itself. A slight thickening was noticed in the endothelial lining of the intact arteries, but it was not enough to quantify.

Breeding Experiment:

The two diabetic male rats were mated with two female rats. The two pairs were left in separate cages for 2 wk. The diabetic males were observed to attempt to mate, but there was no way to tell if copulation
was actually achieved. However, after leaving them with the females for 2 wk and waiting 4 more wk (gestation period for the females), the females never produced litters. These same females were then mated with the controls for 2 wk. Again the males were observed to attempt to mate, but with increased vigor and there was no way to tell if copulation was achieved by simply watching them. Both females here produced litters of 3 and 4 respectively. It should be noted that although Carroll was having problems with their breeding stock at this time, the problem seemed to be one of litter size. Nevertheless, the limited breeding experiment conducted here may be a preliminary indication of male impotency. A more extensive study of male impotence in rats should be performed on a larger population of diabetic and non-diabetic animals.
DISCUSSION

Alloxan provides a quick and convenient method for producing experimental diabetes. It is thought to act directly and specifically on the beta-cells, causing them to undergo degeneration and resorption; the alpha-cells and the acinar tissue remain relatively unaffected.

The minor changes noted in the histological observations of the penis could have contributed to a decrease in potency. Thickening of the tunica albuginea could be a fibrosis, resulting in less expansion of the cavernous bodies than normal. Hyalinization and calcification of this tissue could have occurred (13).

The thickening of the endothelial lining could be caused by the increased permeability of the diabetic rat basement membrane (11). Increased vascular permeability also produces extravasation of lipoproteins into the walls of the vessels. This increased permeability plays a role in the development of atherosclerotic changes (3). Increases in the amounts of hydroxylsine, hydroxyproline, and the glucosyl-galactose disaccharide units may cause this thickening (11).

Destruction of the arteries that supply the corpora
cavernosa can lead to a decreased flow into the lacunae of the cavernous bodies at the time of erection (13).

Human penile erection depends on a successful combination of neurologic, psychogenic and hormonal factors. Mediation of these factors depends upon an adequate blood flow to the cavernous bodies from the penile circulation. An intact venous outflow system is important for maintenance and detumescence once erection is achieved. To fill the cavernous bodies properly, an intact pudendal and corporal circulation must exist.

Cohen et al. (1980) results indicate a significant relationship between impotence and the severity of sclerotic changes in the corporal bed vasculature. They concluded that diabetes mellitus appears to accelerate the aging process in the corporal vasculature (1).

Taking all possible contributions into account, the obstruction of arteries is a primary factor in erectile dysfunction of diabetics. Possible solutions are surgical revascularization for severe cases and strict diet control for the applicable rest.

Important subsequent research should study the effect of diabetes on the triad of causes to see their possible interaction in causing impotence and to see their effect upon each other, possibly assigning a specific contribution to each component.
LITERATURE CITED


