Fat Absorption in Diabetes Mellitus

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SUMMARY

In a group of fifty-eight diabetics (fifty with a family history of diabetes mellitus) in whom the disease was present for at least nine years and in whom a high prevalence of systemic complications was found, five had steatorrhea. In one of them, the steatorrhea appeared to be due at least in part, to an impairment of exocrine pancreatic function. In the others it seemed to be of intestinal origin, although the d-xylose test was normal and jejunal mucosa biopsy did not show significant abnormality. Fecal fat output was measured while the diabetes was rendered poorly controlled, and in two cases the steatorrhea was responsive to improved diabetic control. As far as fat absorption is concerned, no difference was noticed between patients with maturity-onset diabetes and those with diabetes which developed before thirty-five years of age. On the basis of the authors' experience and a review of the literature the existence of true diabetic steatorrhea and its diagnosis are discussed. DIABETES *16*:455-61, July, 1967.

Several reports recently have been published on defective fat absorption arising as a complication of diabetes mellitus,[1-9] and the existence of diabetic steatorrhea as a distinct clinical entity has been proposed.[10-11] But the pathogenesis of this form of intestinal malabsorption remains obscure. Its prevalence has not been established, and its diagnostic criteria have still to be defined. Though the therapeutic results often have been disappointing, improvement has been reported following improvement in control of diabetes[6,10,11] and administration of antibiotics[2,4,11] and corticosteroids.[3,11]

The above considerations led us to carry out a systematic investigation of intestinal fat absorption in a group of fifty-eight diabetics in whom the disease had been known to exist for at least nine years and in whom no history of pancreatitis or of other pancreatic disease could be detected. The great majority of the patients gave histories of familial diabetes mellitus and presented evidence of systematic complications, mainly vascular and neurological. The results of studies have been conflicting. In some, no abnormality in fat absorption was observed in patients with uncomplicated diabetes mellitus,[12,13] while in others an increased stool fat excretion was detected in about one third of the patients examined.[14]

In the present investigation, fat absorption was assayed with a chemical fat balance method and with the I-131-triolein test. In addition, absorption of I-131-triolein was compared with that of simultaneously ingested Br-82 or I-125-oleic acid. In patients with steatorrhea the d-xylose test and peroral jejunal mucosa biopsy were carried out, and the effects of administration of pancreatin and of control of the diabetes were examined.

MATERIALS AND METHODS

The studies were conducted in fifty-eight patients with a history of known diabetes mellitus for at least nine years who had been admitted to the Medical Clinic for control of the diabetes or treatment of some of its complications. None of them was believed to be affected by any other disorder affecting intestinal fat absorption. Particular care was taken to select patients with diarrhea and with evident systemic complications and to avoid those suspected of having diabetes as a result of pancreatic disease. The subjects were divided into two groups: the first was composed of twenty-six patients in whom the diagnosis of diabetes was made before thirty-five years of age (ten of them had labile diabetes); the second included thirty-two patients with diabetes diagnosed at thirty-five years of age or later (two had labile diabetes). Twenty-two patients of the first group and twenty-eight of the second group had a family history of diabetes. Details of the patients' major clinical data are reported in table 1.

Patients were classified as being in good, fair and poor control by clinical assessment. Those with good control had been cooperative in following treatment, had adhered carefully to their diets and had fasting blood sugar levels in the range of 80-120 mg. per 100 ml. (Folin-Wu) in more than 80 per cent of the

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TABLE 1

Data in all groups

Group		Age	Total lipids	Average fecal fat output* Fatty acids Free	Esterified	Saponified	Lipolytic ratio	Plasma ratio of oleic acid and triolein labeled with radioactive tracers† 2 hr.	4 hr.	6 hr.
		yr.	gm./day		gm./day					
Group 1										
Patients without steatorrhea—23 cases: 10 men, 13 women	Mean	36.3	3.71	1.08	0.64	2.04	5.80	1.02°	1.37	1.43
	SD		1.10	0.42	0.20	0.67	1.06	0.17	0.26	0.24
	range	23	2.25	0.65	0.30	1.12	4.05	0.86	0.96	1.16
		58	5.80	1.80	1.10	3.33	8.05	1.35	1.69	1.77
	Mean							0.96°°	0.98	1.04
	SD							0.10	0.11	0.13
	range							0.80	0.80	0.80
								1.15	1.29	1.25
Patients with steatorrhea: 3 cases										
Case No. 24 ♂		39	18.50	4.50	3.50	10.50	5.29	1.64	2.39	1.68
Case No. 25 ♀		40	14.90	5.10	2.05	7.55	7.27	0.74	1.04	1.01
Case No. 26 ♂		50	8.30	1.66	1.80	4.84	4.61	0.76	0.96	1.18
Group 2										
Patients without steatorrhea—30 cases: 13 men, 17 women	Mean	58.9	3.54	0.98	0.70	1.86	5.37	1.30•	1.56	1.62
	SD		0.97	0.35	0.23	0.50	0.99	0.25	0.17	0.17
	range	50	2.10	0.55	0.35	1.02	4.10	0.84	1.29	1.28
		72	5.80	1.75	1.20	3.12	7.66	1.60	1.80	1.90
	Mean							0.89••	1.04	1.11
	SD							0.12	0.11	0.09
	range							0.73	0.90	0.96
								1.18	1.20	1.22
Patients with steatorrhea: 2 cases										
Case No. 57 ♀		70	8.20	1.96	1.54	4.30	5.32	0.97	1.15	1.10
Case No. 58 ♂		54	12.90	4.00	3.10	5.80	4.16	0.88	0.96	1.04

Group 1 includes patients with diabetes developed before thirty-five years of age and Group 2 includes patients with diabetes developed at thirty-five years of age or later.

*The total lipids represent the sum of free, esterified, and saponified fatty acids. The lipolytic ratio is given by the amount of total lipids divided by that of esterified fatty acids, and in normal people its average is 7; when it is under its lower limit of 4, a defective hydrolysis of glycerides is suggested.

†Eight patients of the first group (°) and eighteen of the second group (•) ingested Br-82-oleic acid and I-131-triolein, while fifteen patients of the first group (°°) and twelve of the second group (••) ingested I-125-oleic acid and I-131-triolein. When Br-82-oleic acid and I-131-triolein are used in normal subjects, the Br-82 to I-131 plasma ratio is 0.95 ± 0.21, 1.21 ± 0.23 and 1.38 ± 0.27, respectively 2, 4 and 6 hrs. after the radioactive meal; when I-125-oleic acid and I-131-triolein are used, the plasma ratio between I-125 and I-131 is almost constant at 2, 4 and 6 hrs., with an average value of 0.98 ± 0.11.

‡R = Retinopathy; PN = Peripheral neuropathy; N = Nephropathy; CAD = Coronary artery disease; PA = Peripheral arteriopathy; I = Impotence; D = Diarrhea; LD = Labile diabetes; OH = Orthostatic hypotension.

TABLE 1 (continued)

Data in all groups

Group		Age	Fecal excretion of I-131-triolein (per cent dose)	Duration of known diabetes	Diabetic control (number of cases)	Incidence and nature of complications (number of cases) and other findings‡
		yr.		(yr.)		
Group 1						
Patients without steatorrhea—23 cases: 10 men, 13 women	Mean	36.3	3.84	12.2	Good: 4	R: 12; PN: 10; N: 9; CAD: 4; PA: 3; I: 2; D: 2; LD: 9
	SD		2.08		Fair: 6	
	range	23	1.70	10	Poor: 13	
		58	9.10	20		
	Mean					
	SD					
	range					
Patients with steatorrhea: 3 cases						
Case No. 24 ♂		39	12.50	10	Poor	I, OH, D. D-xylose test: gm. 2.50. Normal jejunal mucosa. Delayed gastric emptying; rapid transit through the small bowel.
Case No. 25 ♀		40	15.30	13	Poor	PN, R, D. D-xylose test: gm. 2.10. Normal jejunal mucosa. Delayed gastric emptying.
Case No. 26 ♂		50	11.40	25	Poor	CAD, R, PA, LD, D. D-xylose test: gm. 1.85. Delayed gastric emptying; rapid transit through the small bowel.
Group 2						
Patients without steatorrhea—30 cases: 13 men, 17 women	Mean	58.9	4.11	13	Good: 6	CDA: 14, R: 12, PN: 9, PA: 9, N: 8, LD: 2.
	SD		1.94		Fair: 11	
	range	50	2.00	9	Poor: 13	
		72	10.14	21		
	Mean					
	SD					
	range					
Patients with steatorrhea: 2 cases						
Case No. 57 ♀		70	8.45	10	Poor	CAD, R, PN, OH, D. D-xylose test: gm. 1.90. Normal jejunal mucosa. Delayed gastric emptying.
Case No. 58 ♂		54	10.10	11	Poor	R, N, D. D-xylose test: gm. 2.15. Normal jejunal mucosa.

Group 1 includes patients with diabetes developed before thirty-five years of age and Group 2 includes patients with diabetes developed at thirty-five years of age or later.

*The total lipids represent the sum of free, esterified, and saponified fatty acids. The lipolytic ratio is given by the amount of total lipids divided by that of esterified fatty acids, and in normal people its average is 7; when it is under its lower limit of 4, a defective hydrolysis of glycerides is suggested.

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‡R = Retinopathy; PN = Peripheral neuropathy; N = Nephropathy; CAD = Coronary artery disease; PA = Peripheral arteriopathy; I = Impotence; D = Diarrhea; LD = Labile diabetes; OH = Orthostatic hypotension.

tests carried out, with moderate glycosuria. Those with poor control were uncooperative in following treatment and diet and had an abnormal fasting blood sugar in more than 50 per cent of the examinations made, with heavy glycosuria or frequent episodes of ketosis. Those with fair control were intermediate. These distinctions, although very approximate, were considered sufficient for the purpose of the present investigation.

In each patient the fecal fat output was measured after diabetic control deliberately had been slackened for at least twenty days (the great majority of patients, however, had shown poor control lasting much longer). The fasting blood sugar was in excess of 200 mg. per 100 ml., and glycosuria was greater than 25 gm. per day. A certain amount of insulin was given when necessary to avoid significant ketosis. After the usual breakfast the patients received a meal consisting of 15 gm. of both oleic acid and triolein containing about 20 μC. of oleic acid labeled with Br-82 or with I-125 and about 20 μC. of I-131-triolein. In plasma samples obtained two, four and six hours later, the radioactivity of each isotope was measured with a well-type scintillation counter (Tracerlab) using gamma-spectrometry. The values were expressed as percentage of the administered dose, and the ratios of Br-82:I-131 or I-125: I-131 were calculated, as previously described.[15] In the feces collected for four days following the radioactive meal, the amount of I-131-triolein was determined and expressed as a percentage of the ingested dose.[15]

The fats were labeled with iodine by the method of Lubran and Pearson[16] and with bromine by the method of Lubran and Corsini.[17,18] The labeled fats were purified before use by passage through a column of ion-exchange resin, Deacidite J (Permutit Co., Ltd., London, England) as previously described,[17,18] so that their content of free tracer was not greater than 2 per cent, and their purity was ascertained by paper chromatography as described by Anghileri.[19]

In the patients found to have steatorrhea, the d-xylose test and peroral biopsy of the jejunal mucosa, as well as x-ray examination with barium meal, were carried out. In addition, the fecal fat output was measured during treatment with pancreatic extract (Cotazym, Organon, 12 capsules per day) and then again after good diabetic control had been achieved and maintained for about three months (table 2).

Quantitative studies of fat balance were carried out with the patients ingesting a 65 to 75 gm. fat diet. The fecal fat was measured with use of the Monasterio and Gigli[20] wet method and the results expressed as a daily average calculated from a pooled collection of four to seven days (usually six days).

The d-xylose test was carried out with a dose of 5 gm. according to the technic of Santini et al.[21] D-xylose in urine collected for five hours following the ingestion of the sugar was measured by the Roe and Rice[22] method. No food was given for twelve hours before the test.

The jejunal mucosa biopsy was performed with a Crosby capsule. The specimens were treated according to standard procedure, and the sections were stained with hematoxylin and eosin.

RESULTS

1. *Patients without steatorrhea.* The fecal fat output was normal (daily excretion of fat < 6 gm.) in twenty-three of twenty-six patients of the first group and in thirty of thirty-two patients of the second group (table 1). In these subjects the lipolytic ratio was in the normal range (> 4) and no significant difference was observed between the absorptions of oleic acid and triolein. The fecal excretion of I-131-triolein was normal (< 7 per cent of the ingested dose) in forty-eight

TABLE 2

Influence of diabetic control and pancreatin treatment on fecal fat output*

	Poor diabetic control		Poor diabetic control Pancreatin treatment			Good diabetic control			Poor diabetic control
	Weeks		Weeks			Months			Months
Cases	1	4	6	9	14	7	10	13-16	19
No. 24	18.10	20.12	10.10	9.20	12.45	4.50†	3.10	5.40	14.40
No. 25	14.90	12.80	12.40	16.90	18.80	15.80	16.20	13.40	
No. 26	8.30	11.80	7.94	11.50	—	9.10	14.45	10.35	
No. 57	8.20	10.50	12.55	13.40	—	9.40	13.50	10.24	
No. 58	12.90	16.40	16.86	14.90	—	5.50	4.30	3.40	

*The values of the fecal fat output are expressed in gm. per day; the data represent a daily average over a period of four to seven days' (usually six days) collection. Control of diabetes was obtained with insulin.

†This patient continued the pancreatin treatment during this time.

patients and increased in the other five. In these last subjects a second chemical fat balance was carried out, which confirmed the results of the chemical determination (the results of the first tests only are reported in table 1).

In the twelve patients with labile diabetes mellitus there were eleven with normal fecal fat output; two patients out of fifty-three without steatorrhea had moderate diarrhea of unexplained origin.

2. *Patients with steatorrhea.* The findings in the five patients with steatorrhea are summarized in tables 1 and 2. The fecal fat loss was moderate (between 6 and 12 gm. per day) in cases 26 and 57 and marked (greater than 12 gm. per day) in cases 24, 25 and 58. The I-131-triolein test was abnormal and the lipolytic ratio normal in every patient. The absorption of oleic acid was parallel to that of triolein in all these cases except Case 24 in whom the absorption of oleic acid was significantly faster than that of triolein. In no patient did the d-xylose test give abnormal results, nor did peroral jejunal biopsy show any significant abnormality in four cases. In Case 26 the tube failed to pass through the pylorus. X-ray examination with barium meal revealed a delay in gastric emptying in cases 24, 25, 26 and 57, and a rapid transit through the small bowel in cases 24 and 26. In no patient was any other abnormality seen within the small bowel.

In Case 24 the fecal fat output appeared to be reduced during the administration of pancreatin and was brought back to normal with good control of diabetes. In the other four cases no effect was obtained with administration of pancreatin, and good control of the diabetes appeared to be effective in Case 58 in whom the fecal fat output became normal (table 2).

Diarrhea began five years (Case 24), eight years (Case 25), ten years (Case 26), seven years (Case 57), and thirteen years (Case 58) after the diagnosis of diabetes, the control of which had been very poor in each case. When steatorrhea was relieved by treatment, diarrhea also almost completely disappeared.

Case 26 had labile diabetes, but the appearance of intestinal symptoms did not seem to modify the clinical pattern of the diabetes.

DISCUSSION

Steatorrhea was found in five out of the fifty-eight diabetic patients. The genesis appeared to be due to impairment of the exocrine pancreas only in Case 24 (who had a family history of diabetes mellitus), as can be judged more completely from the fast absorption of oleic acid as compared with that of triolein and from the reduction of the fecal fat output induced by pancreatin treatment. The normal lipolytic ratio does not invalidate this assertion since in pancreatic steatorrhea other lipolytic enzymes (of bacterial or intestinal origin) can efficiently hydrolyze undigested fat. In this patient steatorrhea was reduced by administration of pancreatin, completely eliminated by good control of diabetes, and reappeared again some months later when the patient experienced hyperglycemia and heavy glycosuria (table 2). This suggests strongly that the intestinal malabsorption was not due solely to impairment of exocrine pancreatic function.

In the other four patients, steatorrhea did not appear to result from pancreatic insufficiency since absorption of oleic acid was parallel with that of triolein, and treatment with pancreatin did not significantly modify fecal fat output. In these patients, other known causes of intestinal malabsorption were absent (including infection by protozoa or helminths). Also, the d-xylose test and the jejunal mucosa pattern were normal.

Patients reported suffering from steatorrhea, possibly related to diabetes mellitus, generally have had long-standing and poorly controlled diabetes, and persistent diarrhea (not always, however, associated with steatorrhea[2,7,11]) without evident impairment of the exocrine pancreas. Such patients usually have exhibited signs of neuropathy and abnormalities of gastric and intestinal motility, together with a high prevalence of systemic complications. Conflicting findings have been reported on the pattern of the jejunal mucosa. Changes similar to sprue have been observed in some patients,[9] while in others normal findings or evidence of mild inflammation have been seen.[6,7,11]

In general, the present results (table 1) agree with those of previous studies. The normality of the d-xylose test and of the jejunal mucosa pattern observed in our cases does not support the view, however, that diabetic steatorrhea is associated with a sprue-like lesion of the small bowel. We are inclined to agree with certain others[10,23] that in diabetic patients with histological findings of nontropical sprue, steatorrhea is very likely due to the occasional association of a gluten-induced enteropathy, for malabsorption was usually relieved by a gluten-free diet or by treatment with corticosteroids.

The observation that diabetic steatorrhea appears principally in male patients[10] and in juvenile diabetes[10] was not confirmed by the present results. Also, we could not confirm that labile diabetes is often a conse-

quence of intestinal malabsorption. In only one of the twelve patients with labile diabetes was there steatorrhea. But in this connection the number of the patients investigated was too small to allow conclusion.

We suggest that steatorrhea may be reasonably accepted as a complication of diabetes mellitus. This view is supported by the analogy of the clinical pattern of the patients described and by the response of the steatorrhea (at least in cases 24 and 58 in the present investigation) to good diabetic control. The link between steatorrhea and diabetes remains unknown, however. The assumption that it is related to autonomic neuropathy of the gastrointestinal tract is not supported by pathological findings.[24] Evidence of a neuropathic origin arises from clinical and laboratory observations, such as its high prevalence in patients with other neurological complications and the frequent finding of abnormal motility of the stomach and of the bowel. It has been suggested also that bacterial contamination of the upper part of the gastrointestinal tract may play a part in diabetic steatorrhea.[2,4,11] So far diabetic microangiopathy has not been seen in the small intestine of patients with such a syndrome.[6,24-27] Very likely many factors are responsible for diabetic steatorrhea, and they probably differ in severity and reversibility. Therefore, at the moment it does not seem possible to agree with Wruble and Kalser[10] that responsiveness to good diabetic control is an essential feature of true diabetic steatorrhea.

It is apparent that the diagnosis of diabetic steatorrhea is one of exclusion. The elements suggesting its presence are as follows: (1) poorly controlled and long-standing diabetes; (2) normal exocrine pancreatic function; (3) normal d-xylose test; (4) absence of significant lesions in the jejunal mucosa; (5) abnormality of gastric emptying and of transit through the small bowel; (6) systemic complications, mainly in the nervous system. The diagnosis is strongly supported if intestinal malabsorption is relieved by good diabetic control.

In the present investigation, Cases 25, 26, 57 and 58 had typical patterns of diabetic steatorrhea, and in Case 58 intestinal malabsorption disappeared after good control of diabetes was obtained. In Case 24, steatorrhea very probably was not due solely to pancreatic insufficiency but in part was connected with underlying metabolic disease, as suggested by its sensitivity to diabetic control.

Our data do not provide any grounds for estimating the prevalence of diabetic steatorrhea, since patients were selected according to the above-mentioned criteria. It is our impression that this condition is more common than appreciated. Our results are in agreement with those obtained by others in a small series of patients, but differ considerably from those of Vacca et al.[14] These workers found steatorrhea in about one third of fifty-five patients with uncomplicated diabetes mellitus. This discrepancy has no ready explanation but possibly may be related to the different proportion of patients with a family history of diabetes mellitus, fifty out of fifty-eight in our group and ten out of fifty-five in theirs.[14] These authors may have included some patients with silent chronic pancreatitis, as may be inferred from the concomitant finding of a high prevalence of impairment of the exocrine pancreatic function.

ACKNOWLEDGMENT

This investigation was supported by Association Contract 026-63-4-BIAC between Euratom, the University of Brussels, and the University of Pisa.

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The Nutritional Value of a Canned Food Diet

The nutritional characteristics of canned food may be evaluated by study of the effect of food processing, canning, and shelf life on the micronutrient composition of food prior to, during, and following these procedures. Thus, N. B. Guerrant et al. (J. Nutrition 32: 435, 1946) studied retention of ascorbic acid, carotene, thiamine, riboflavin, and niacin in various canned foods. They found that lima and snap beans lost significant vitamin content as a result of the process in use at that time. M. Ives, A. E. Pallard, C. A. Elvehjem, and F. M. Strong (J. Nutrition 31:347, 1946) also investigated the pyridoxine, biotin, and folic acid content of 101 samples of canned foods. The influence of temperature, time, and storage on the vitamin content of tomato juice, lima beans, and whole kernel yellow corn was studied by Guerrant, M. G. Varick, and R. A. Dutcher (Indust. Eng. Chem. 37:1240, 1945). These investigators found that storage temperature exerted a limited effect on carotene and riboflavin retention, but considerable losses of thiamine, ascorbic acid, and pantothenic acid were observed.

F. C. Lamb, A. Pressley, and T. Zuck (Food Res. 12: 273, 1947) reported on the adverse effect of blanching on vitamin C retention of peas, green beans, and spinach. However, excepting thiamine, the canning process preserved the vitamin content of the foods they studied. Further documentation on the vitamin status of foods subjected to the blanching process was made by the study of J. R. Wagner, Strong, and Elvehjem (Indust. Eng. Chem. 39:985, 1947). Study of the nutritive value of canned foods was given further impetus when methodologies became available for determination of the amino acid content of foods. J. B. Neilands et al. (J. Nutrition 39:187, 1949) found that the canning process did not materially affect the essential amino acid content of fish and meat; this was confirmed by M. S. Dunn et al. (J. Nutrition 39:177, 1949).

From *Nutrition Reviews,* Vol. 24, No. 2
February 1966, p. 46-48