Acute Pancreatitis with Hyperlipemia:

The Incidence of Lipid Abnormalities in Acute Pancreatitis

JOHN L. CAMERON, M.D., DAVID M. CAPUZZI, M.D.,
GEORGE D. ZUIDEMA, M.D., SIMEON MARGOLIS, M.D.

From the Departments of Surgery and Medicine, The Johns Hopkins Medical Institutions, Baltimore, Maryland

An association between acute pancreatitis and hyperlipemia is well recognized.[4] The etiology and frequency of this association, however, are unknown. Experimental work has indicated that hyperlipemia may result from pancreatitis;[25,27] in contrast, some clinical data strongly suggest that the lipid disorder provokes acute pancreatitis.[6,21] Estimates of plasma lipid abnormalities in acute pancreatitis have ranged from 4%[20] to 53%.[26] In part, the wide disparity in the reported frequency of lipid abnormalities may result from differences in terminology. Increased levels of circulating lipids (hyperlipidemia) produce lactescent or lipemic serum (hyperlipemia) only when serum triglyceride levels exceed 300–400 mg./100 ml. Although it has been estimated that only 3 to 12% of patients with acute pancreatitis have lipemic serum during the episode,[5,13] it has been our impression that lipid abnormalities are present in a much larger proportion of patients with acute pancreatitis. In order to examine this question, lipid metabolism was studied prospectively in a consecutive series of patients who presented to our hospital with acute pancreatitis.

Clinical Material

Criteria for inclusion in the study group were 1.) the clinical diagnosis of acute pancreatitis, and 2.) either an elevated serum amylase (exceeding 250 Caraway units) or lactescent serum. Forty-eight patients met these criteria and were studied in 57 consecutive hospital admissions (Table 1). The pancreatitis was associated with alcoholic intake on 45 admissions and biliary tract disease on nine admissions. After inclusion in the study the following parameters of serum lipid metabolism were measured: total lipids, triglycerides, cholesterol, phospholipids, free fatty acids (FFA), post heparin lipolytic activity (PHLA), PHLA inhibition, and lipoprotein electrophoresis. A 24-hour urine collection for amylase excretion was obtained during one of the first 3 days after admission in 33 patients.

An identical battery of lipid studies was performed on a "control" group of 34 patients with a variety of abdominal disorders (Tables 1 & 2). This was a prospective, nonconsecutive group of patients picked randomly after admission with acute abdominal pain. None was thought to have acute pancreatitis, and all had normal serum amylase values (40 to 160 Caraway units). Twenty-four-hour urinary amylase excretion was measured during one of the first 3 days after admission in nine patients.

Methods

Serum cholesterol, triglycerides, phospholipids, and total lipids were determined by Bio-Science Laboratories. FFA levels were assayed by the titration method of Dole and Meinertz.[10] Serum lipoprotein electrophoresis was carried out on paper as described by Lees and Hatch.[22] PHLA was determined by the method of Frederickson, Ono, and Davis.[12] Sera were tested for their inhibitory effect on the PHLA of normal subjects as follows. The PHLA activity of a normal subject was determined after adding 0.2 ml. of saline or 0.2 ml. of pre-heparin serum from test patients to the other components of the PHLA assay. Inhibition was calculated by comparing the effects

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TABLE 1. *Characteristics of Patients in Study and Control Groups*

Group	No.	Average Age	Race			Sex	
			B	W	Chinese	M	F
Study	48	43	37	10	1	35	13
Control	34	38	27	7		19	15

TABLE 2. *Diagnoses of Patients in Control Group*

Diagnosis	Number
Small Bowel Obstruction	12
Acute Cholecystitis	6
Acute Appendicitis	3
Gastroenteritis	3
Pyelonephritis	3
Acute Diverticulitis	3
Pelvic Inflammatory Disease	2
Large Bowel Obstruction	1
Abdominal Wall Abscess	1

of saline or patient's serum on the release of FFA. Inhibition was considered significant when it was greater than 20%.

Results

Study Group. Ten patients (21%) in this series were noted to have lactescent serum on admission (Table 3). Serum amylase values were normal in eight and elevated in the remaining two patients (312 and 695 Caraway units) with lipemic serum. Both patients with elevated amylase values and lactescent sera died. In those patients without lipemic serum, amylase values ranged from 260 to 2304 units. All ten patients with lactescent sera had markedly elevated serum triglycerides which ranged from 493 to 7520 mg./100 ml. An additional eight patients (nine admissions) who were not noted to have lactescent serum, had serum triglyceride levels that exceeded 175 mg./100 ml. Thus, a total of 18 patients (38%) had elevated triglyceride levels on admission (Fig. 1). Eleven patients had elevated levels of total serum lipids (normal 1000 mg./100 ml.). Although this included all 10 patients with lactescent sera, total serum lipids were normal in seven of the eight patients with high serum triglycerides but without lactescent sera. Only four patients, all with lipemic sera, had serum cholesterol levels greater than 350 mg./100 ml. (363 to 545 mg./100 ml.). Elevated serum phospholipids were present in nine patients. Seven of these patients had lactescent serum on admission, but the remaining two had normal serum triglycerides.

FFA levels were elevated in 30 of 53 admissions (Fig. 2). PHLA levels were depressed on 38 admissions and normal on 15 admissions. These values bore no relationship to lactescence or to serum triglyceride levels (Fig. 3). PHLA inhibition was measured on 50 admissions. Seventeen patients showed no inhibition of PHLA. However, on 33 admissions PHLA inhibition ranged from 22 to 65%. Although these levels bore no consistent relationship to serum triglyceride levels, inhibition was detected in eight of the nine hyperlipemic patients (Table 3).

Lipoprotein electrophoretic patterns were done on all patients with acute pancreatitis and lipemic serum. In nine of these patients a type V pattern was present. One patient had only a very prominent chylomicron band, consistent with type I hyperlipoproteinemia. Several months after this patient had recovered from acute pancreatitis, repeat lipoprotein electrophoresis showed a typical type V pattern. Electrophoretic patterns were done on seven of the eight patients who had hypertriglyceridemia, but not lipemic serum. Four had a type IV pattern, two had elevated chylomicrons (type I) and one had a normal pattern. Among the patients with acute pancreatitis and a normal serum triglyceride level, 24 had normal electrophoretic patterns, 12 had decreased β-bands, and one had a serum cholesterol of 300 mg./100 ml. and an electrophoretic pattern consistent with type II hyperlipoproteinemia.

Control Group. No patient in the control group had lactescent serum on admission, but serum triglycerides

TABLE 3. *Data on the Ten Patients with Acute Pancreatitis and Hyperlipemia*

Patient	Age, Race, Sex	Serum Triglyceride mg./100 ml. (150 mg./100 ml.)	Lipoprotein Pattern	PHLA μEq FFA/ min./ml. (.25–.55)	PHLA Inhibition % (0–20%)	Serum Amylase (Caraway Units) (40–160 u)	Urinary Amylase (Cawaway Units /24 hrs) (0–5000 u)
J.Y. 115 75 05	48 BM	1033	V	.18	32	77	6849
W.M. 14 20 93	50 WM	3515	V	.33	35	65	—
C.B. 88 15 70	22 BM	4643	V	.44	23	20	3680
W.F. 77 94 58	37 BM	1104	I → V	.17	0	62	4900
L.L. 139 17 26	41 BF	2265	V	.19	36	312	—
J.B. 98 58 17	38 BM	7520	V	.66	27	53	946
B.M. 133 70 70	40 WF	2553	V	.26	35	150	—
E.S. 59 62 33	49 WM	493	V	.22	64	53	—
R.W. 135 58 20	21 WF	800	V	.09	33	20	2574
D.C. 139 63 35	27 WF	2833	V	—	—	695	—

were elevated in three patients (Fig. 1). Serum phospholipids were increased only in these three patients. Serum total lipids and cholesterol were each elevated in single patients. FFA levels were elevated in 15 of 33 patients tested (Fig. 2). PHLA was normal in six patients and depressed in 27 patients. The three patients with elevated triglycerides all had depressed PHLA (Fig. 3). No inhibition of PHLA was detected in 28 patients, but inhibition ranging from 31 to 34% was found in three patients. Serum from three patients stimulated PHLA from 21 to 23%.

Lipoprotein electrophoretic patterns were done on 33 patients in the control group. The pattern was normal in seventeen patients. In another 13 patients there was decreased staining of the β-band. This is consistent with the relatively low levels of serum cholesterol in many of these patients. One, for example, had a serum cholesterol of 84 mg./100 ml. In one patient the electrophoretic pattern and serum cholesterol value was consistent with type II hyperlipoproteinemia. One patient had a distinct pre β-band but normal levels of serum triglycerides. Serum triglycerides and the electrophoretic pattern were consistent with type V hyperlipoproteinemia in one patient.

Discussion

In an attempt to document the incidence of lipid abnormalities in acute pancreatitis, a consecutive series of patients was studied who met our established criteria for this diagnosis. Since a relatively high serum amylase level (greater than 250 Caraway units) was one requisite for inclusion into the study group, some patients with acute pancreatitis and minimal serum amylase elevations (160–250 Caraway units) may have been excluded. A relatively high serum amylase was chosen to exclude patients with gastritis, cholecystitis and other abdominal diseases that can present with abdominal pain and minimal abnormalities of serum amylase. Since most patients with lactescent serum accompanying acute pancreatitis have normal serum amylase values,[4] the presence of lactescent serum was accepted in place of an elevated serum amylase for inclusion into the study. This criteria can be justified on the following grounds. Over the past 6 years the authors have carefully evaluated 31 patients with abdominal pain and lactescent serum. Eight had elevated serum amylase values on admission. Of the remaining 23, five patients had the diagnosis of pancreatitis confirmed at operation. Three additional patients had amylase elevations in fluid obtained by peritoneal lavage. Five others developed serum amylase elevations as their lactescent serum cleared. At the time of discharge from the hospital, the diagnosis in all 31 patients was acute pancreatitis. From this experience, we consider lactescent serum accompanying abdominal pain at least as accurate as an elevated amylase

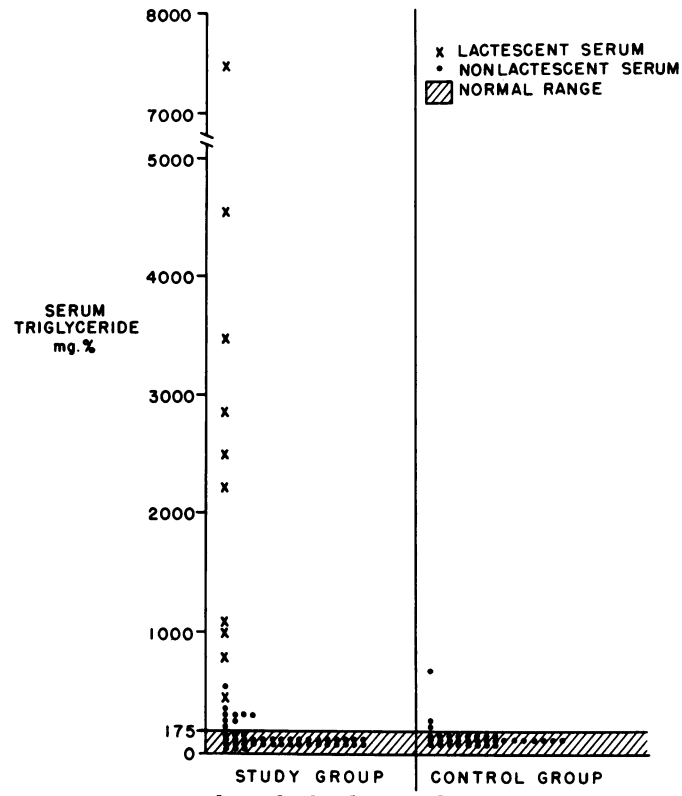

FIG. 1. Serum triglyceride levels on admission for study and control patients.

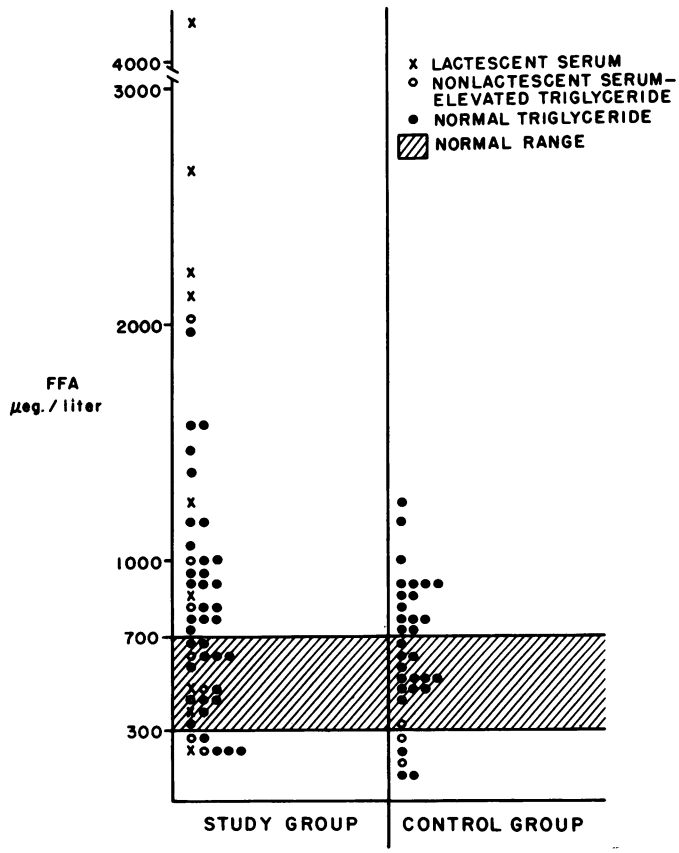

FIG. 2. Serum free fatty acid levels on admission for study and control patients.

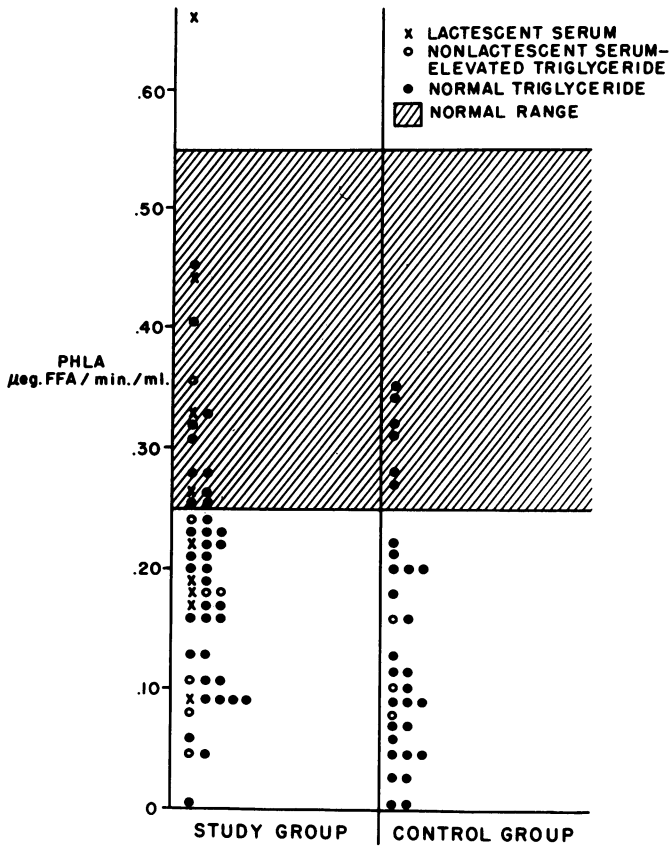

FIG. 3. Post heparin lipolytic activity on admission for study and control patients.

in confirming the clinical diagnosis of acute pancreatitis.

The reason for the normal levels of serum amylase in the presence of lactescent serum remains speculative. One explanation is a circulating inhibitor of amylase, possibly in the lipid fraction. Attempts to remove such an inhibitor in this laboratory by dilution or by dialysis have been unsuccessful. Early data suggested that increased levels of urinary amylase, despite normal serum levels, might provide diagnostic aid.[4] As seen in Figure 4, however, in patients with lactescent serum urinary amylase values also tended to be low and overlapped the urinary amylase values of the control group. It is important to emphasize that serum amylase levels were elevated in all of the eight patients with acute pancreatitis whose mild elevation of serum triglycerides was insufficient to cause lactescence. In addition, elevated levels of serum amylase despite lactescent serum may portend an unfavorable prognosis since both patients who presented in this manner underwent a fulminant course and died of acute pancreatitis.

In addition to the 10 patients with lactescent serum and markedly elevated serum triglycerides, eight others had moderate triglyceride elevations. The upper limit of normal for fasting serum triglycerides ranges from 135 to 150 mg./100 ml. A value of 175 mg./100 ml. was chosen as the upper limit of normal to eliminate borderline elevations. All but one patient with elevated triglycerides had serum levels in excess of 200 mg./100 ml. Because of abdominal pain, nausea and vomiting, all patients were fasting for at least 12 hours prior to blood sampling. The 19 admissions with triglyceride elevations represent 33% of admissions and 38% of all patients in the study group. Because lactescence is detected consistently only when serum triglycerides exceed 300 to 400 mg./100 ml., lesser elevations are overlooked unless triglyceride determinations are obtained. Greenberger *et al.*[13] found only three patients (12%) with serum triglycerides greater than 200 mg./100 ml. in a consecutive series of 25 patients with acute pancreatitis. Their series had a lower percentage of patients with pancreatitis related to alcohol ingestion. Pancreatitis was secondary to biliary tract disease in 40% of their patients; this could partly explain the different incidence of lipid abnormalities in the two series. However, triglyceride elevations were noted in our study group in two of the nine patients with acute pancreatitis secondary to biliary tract disease. Possibly accounting at least in part for our high frequency of hyperlipemia in association with pancreatitis, is that two of the ten hyperlipemic patients had been followed here in the past with a type V lipoprotein pattern and who might otherwise have sought hospitaliza-

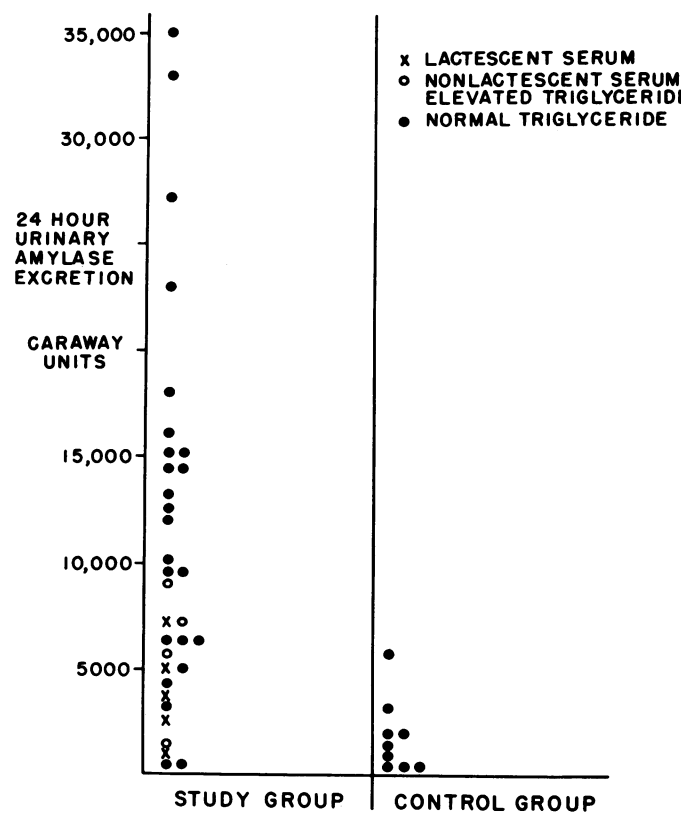

FIG. 4. Urinary amylase excretion for study and control patients measured during one of the first 3 days following admission.

tion elsewhere for their acute attack of pancreatitis. Three patients in the control group had increased serum triglyceride levels. None was noted to have lactescent serum although one patient had a serum triglyceride level in excess of 700 mg./100 ml. Clinically, this patient was thought to have gastroenteritis. In retrospect, particularly in view of the marked triglyceride elevation and his type V electrophoretic pattern, acute pancreatitis was probably the correct diagnosis. This leaves two moderate triglyceride elevations among 33 control patients (6%).

Total serum lipid determinations were usually not helpful in detecting moderate lipid abnormalities. In fact, determination of total serum lipids was no more valuable than gross inspection for lactescence. An increase in total serum lipids was present in only one of the eight patients with elevated serum triglycerides but non-lactescent serum. In most instances cholesterol and phospholipid elevations occurred in patients with lactescent serum. However, two patients with normal triglycerides and total lipids had slightly increased serum phospholipids. The significance of this is unknown.

FFA elevations were present in 57% of the patients with acute pancreatitis. This was not significantly different from the incidence of FFA elevations in the control group (45%). FFA elevations can follow a variety of physiological stimuli, including starvation, catecholamine release, glucagon stimulation, and insulin deficiency.[11] Many or all of these stimuli may have been present in both the study and control groups. Elevated FFA levels have been associated by others with the pathogenesis of cardiac arrhythmias[24] and the respiratory insufficiency accompanying fat emboli.[1] There is no direct evidence from the present study, however, to incriminate elevated serum FFA levels specifically in pancreatic disease.

Absent or decreased PHLA is a consistent finding in patients with type I hyperlipoproteinemia[11] and has been described in some patients with acute pancreatitis and hyperlipemia.[2,13] Fat induced hyperlipemia and recurrent pancreatitis, however, have also been reported in a patient with normal PHLA levels.[7] In the present study 72% of the patients with acute pancreatitis and 82% of the patients in the control groups had low PHLA levels. However, PHLA bore no relationship to the serum triglyceride level. The patient with the highest PHLA in the study group (.66μEq FFA/min/ml) also had the highest serum triglyceride level (7520 mg./100 ml.). The explanation for the low levels of PHLA in many patients from both groups is unclear. It should be pointed out that the values utilized to define the normal range for PHLA were obtained from a large published series[12] rather than from a control group studied in our own laboratory. Part of the PHLA is hepatic in origin[28] and patients with liver disease commonly have low PHLA in their serum.[9,23] This would not explain the frequency of reduced PHLA in our control group patients who generally did not have a strong history of alcohol excess. From this study it appears that nonspecific depression of PHLA may accompany many acute abdominal illnesses. The results further indicate that reduced PHLA by itself cannot explain the hyperlipemia associated with acute pancreatitis.

Kessler *et al.*[19] reported the presence of an inhibitor of PHLA in the plasma of a patient with hyperlipemia and pancreatitis. In the present study the PHLA of normal subjects was inhibited much more frequently (66%) by preheparin plasma from patients with acute pancreatitis than by preheparin plasma from members of the control group (10%). However, there is little to suggest that the hyperlipemia is secondary to inhibition of PHLA since there was no correlation between PHLA inhibition and the degree of triglyceride elevation.

Lipoprotein electrophoresis showed that chylomicrons were present in the serum of all patients with acute pancreatitis and hyperlipemia. Each of these patients had a type V pattern except for one who had a type I pattern on admission with pancreatitis, but had a type V pattern several months later. These results suggest that patients with the type V pattern are especially prone to develop acute pancreatitis or that prolonged chylomicronemia may trigger pancreatitis. Alternatively, the presence of chylomicrons may only reflect the severity of the triglyceridemia with the resultant saturation of clearing mechanisms for endogenous fats. Studies in progress, on the persistence of the hyperlipemia in these patients, may provide better understanding of the relationship between the elevated serum lipoproteins and pancreatitis.

It is difficult to interpret the lipoprotein abnormalities in the patients with pancreatitis and increased triglycerides, but without lactescent sera. It is possible that hyperlipemia triggered pancreatitis in these patients as well, but that they sought medical attention later in the course of their illness, at a time when the lipid values had returned nearly to normal. Thus, the four patients with a type IV pattern might have shown chylomicronemia if tested a few days earlier.

Most of the normolipemic patients from both groups had normal lipoprotein electrophoretic patterns. A surprisingly large number of patients had poorly staining β-bands associated with low serum cholesterol levels. These findings suggest that acute intra-abdominal illness may frequently lead to a substantial reduction in serum cholesterol and β-lipoprotein levels.

The data from this study suggest that lipid abnormalities are common in acute pancreatitis. It is possible that the two conditions coexist, but are not related etiologically. Since both elevated serum triglycerides[18,23] and pancreatitis can result from prolonged alcohol ingestion,

the two may occur together just coincidentally. Experimentally both hyperlipemia and the histologic changes of chronic pancreatitis can be produced by chronic alcohol administration in dogs.[8] However, alcohol had not been ingested in at least two of the ten hyperlipemic patients, and in at least two of the eight patients with elevated triglycerides but without lactescent serum. Therefore, the association between lipid abnormalities and pancreatitis cannot in every instance be just a coincidental one related to alcohol ingestion. Experimental studies have documented triglyceride elevations, and in some instances hyperlipemia, following the induction of acute pancreatitis in animals.[25,27] In addition, in most patients with hyperlipemia and acute pancreatitis, the lactescent serum clears as the pancreatitis subsides. Also, most patients with pancreatitis and hyperlipemia have no past history or family history of lipid disorders. These points favor the thesis that pancreatitis is the primary disease and that the hyperlipemia is secondary. On the other hand, patients with familial type I or type V hyperlipoproteinemias have recurrent bouts of abdominal pain that probably represent episodes of pancreatitis.[11] All patients with lipemic serum and acute pancreatitis in our series had either a type I or type V electrophoretic pattern. In the type I and V familial diseases, the episodes of pancreatitis decrease or stop if triglyceride levels are significantly lowered.[17,21] Eight of the ten hyperlipemic patients in the present study were restudied on a metabolic ward after a period of at least 6 weeks had elapsed following hospitalization for acute pancreatitis. Each patient was stressed with alcohol, carbohydrate, and lipid loads. Lipid transport was abnormal in all eight patients. During the tests one patient developed abdominal pain radiating through to her back, probably representing mild pancreatitis, when her triglyceride level rose to 3000 mg./100 ml. Presumably lipid metabolism should have been normal in these patients six weeks following their episode of pancreatitis, if their initial hyperlipemia had been secondary to pancreatic disease. These data will be reported subsequently in detail.[3]

Massive elevations of serum triglycerides could cause or predispose to acute pancreatitis by several mechanisms. Havel[16] proposed that pancreatic lipase, presumably present in high concentration in pancreatic capillaries and lymphatics, may produce rapid hydrolysis of the triglyceride component of circulating chylomicrons. This would lead to locally high concentrations of FFA that could cause microthrombi in pancreatic capillaries with resultant tissue ischemia, or could exert direct toxic effects on acinar cells. Alternatively, the attack on chylomicrons could clump these large particles and thus produce capillary occlusion. Animal studies have demonstrated that diets high in lipids predispose to pancreatitis.[14,15] These studies showed that the increased susceptibility was not related to changes in the quantity or quality of pancreatic secretions, and it was suggested that the integrity of acinar cell membranes was influenced by the diet. Possibly acinar cells are more susceptible to injury when exposed to high levels of serum lipids. In such a situation elevated serum triglycerides or FFA would not actually initiate pancreatic inflammation, but rather would create an environment that enhanced the toxic effects of some other agent. Since ethanol ingestion can raise serum triglycerides, it is possible that alcoholic intake and pancreatitis are related through this mechanism.

It appears clear from this study that lipid abnormalities are frequent in acute pancreatitis and that this association is more than coincidental. There is some experimental evidence in animals to suggest that lipid abnormalities can be secondary to inflammatory pancreatic disease. However, it seems likely from our data and from other work that in some instances in humans lipids play an intermediary role in the pathogenesis of acute pancreatitis.

Summary

In order to document the incidence of lipid abnormalities, lipid metabolism was studied in 48 consecutive patients with acute pancreatitis. Results were compared to 34 patients with abdominal pain, but without acute pancreatitis, used as controls. Ten patients (21%) with acute pancreatitis had lactescent serum; their triglyceride levels ranged from 493 to 7520 mg./100 ml. An additional eight patients (17%) with acute pancreatitis had elevated triglyceride levels ranging from 186 to 551 mg./100 ml., but did not have lactescent serum. None of the controls had lactescent serum but 9% had triglyceride elevations. Decreased post heparin lipolytic activity (PHLA), and PHLA inhibition were detected in both groups of patients, but neither bore a consistent relationship to triglyceride levels. Lipoprotein electrophoresis demonstrated that all patients with lactescent serum and acute pancreatitis had either a type I or type V pattern. Possible mechanisms by which lipid abnormalities and acute pancreatitis might be related are discussed.

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