

Diabetes and Abdominal Surgery: The Mutual Risks

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The patient with diabetes represents to the surgeon a particular challenge in the management of acute abdominal problems. In addition to their ongoing and potential metabolic problems, diabetics have specific difficulty in their ability to handle infections and heal wounds. The present report reviews the general principles in the peri-operative management of diabetics and discusses the implications in the diabetic of several specific clinical problems. In view of the known accelerated atherosclerosis associated with diabetes, the risks of anesthesia and surgery must be assessed in the context of the coronary, cerebral, visceral, and peripheral vascular status. Infections in diabetics (potential or established) must be treated aggressively and promptly. Acidosis in the diabetic with abdominal pain must be considered both a metabolic problem and a possible secondary manifestation of an intra-abdominal process. In view of these challenges, the need for careful, anticipatory management of the diabetic patient facing major abdominal surgery is clear.

The management of surgical problems in diabetic patients represents a common but significant challenge. Approximately 3-4 percent of the general population is affected by diabetes, and about one-half of them will eventually require surgery. It has been estimated that in about one-quarter of the cases, the diabetes will be discovered for the first time in the peri-operative period [1]. Advances in the understanding and treatment of diabetes, as well as advances in anesthesia, peri-operative care, and surgical technique have dramatically improved the outcome of operations performed in diabetics. The reported operative mortality in diabetics has decreased from as high as 50 percent in the 1930s to a current low of under 2 percent [1,2,3,4]. While a better understanding of the metabolic problems in diabetics has resulted in improved peri-operative management, diabetes continues to represent a significant risk factor in patients with surgical problems (a factor that increases morbidity, mortality, and the cost of health care).

We will review the pathophysiologic derangements which make surgery on the diabetic a particular problem, emphasizing the peri-operative management of diabetics and the role of diabetes as a risk factor in patients with abdominal surgical problems.

While the underlying endocrine abnormalities in diabetes mellitus remain incompletely defined, the diabetic state is characterized by hyperglycemia and glycosuria secondary to a relative or absolute lack of insulin. The implications of these changes in diabetic surgical patients are predominantly metabolic, infectious, and vascular. Of the post-operative complication rate of 15-20 percent reported in diabetics, about one-third are wound infections, one-third are genito-urinary or

other infections, 10 percent are cardiovascular, and 5 percent are problems in wound healing [1,3].

First, the diabetic is prone to metabolic decompensation in response to the stress of either surgery, anesthesia, or the underlying condition which precipitated the need for operation. Marked hyperglycemia, nonketotic hyperosmolar coma, and diabetic ketoacidosis represent particular problems in the surgically stressed patient.

Second, diabetics undergoing surgery are considered to be at increased risk to develop a significant infection. While it has been widely assumed that diabetics are more prone to developing wound and other infections than are non-diabetics, the clinical data are conflicting [3,5,6] and the correlation is not clearly established. Perhaps the severity of infection in the diabetic, once established, contributes to the impression that these patients have a higher infection rate.

Many studies have examined the increased susceptibility of diabetics to infection. On a cellular level, it has been shown that migration of polymorphonuclear leukocytes is delayed and phagocytosis is impaired [7] with a significant decrease of both intracellular bacteriostatic activity of leukocytes [8] and a decrease in serum opsonic activity for both staphylococcus aureus and *E. coli* [9]. Lymphocyte activity, as measured by response to phytohemagglutinin, is also depressed in poorly controlled diabetics [10]. These abnormalities are significantly more likely in hyperglycemic, poorly controlled diabetics and less likely when the diabetes is well-controlled. The control of hyperglycemia and infection are interrelated in that infection leads to both hyperglycemia and acidosis which, in turn, lead to depression of the diabetic's cellular immune system and further progression of the infection [11].

The third consequence of diabetes which is relevant to surgical decision making is the accelerated development of diffuse, occlusive arteriosclerotic vascular disease. Thickening of arteriolar and capillary vessel walls associated with dilatation of venules is known as "small vessel disease" and is often associated with the retinopathy, ischemic extremity ulcers, renal arteriosclerosis, and heart disease seen in diabetics. In addition, diabetics have a significantly increased incidence of occlusive disease in larger vessels (as a consequence of accelerated atherogenesis), particularly in intermediate-sized muscular arteries such as the radial, tibial, and coronary. These vascular lesions account for a significant percentage of the post-operative morbidity and mortality in patients undergoing abdominal surgery.

DIABETES MANAGEMENT

Surgical management in the diabetic must be directed toward anticipating and aggressively treating both hyperglycemia and acidosis, while avoiding hypoglycemia. If the diabetes is unrecognized or inappropriately treated, severe acidosis may develop in the post-operative period. With the diabetic's abnormal glucose utilization, endogenous fat and protein are metabolized to provide calories to meet energy needs. Acidosis results from the accumulation of ketone bodies with a reduction in both plasma bicarbonate and blood pH. As the post-operative energy demands increase, the catabolism of fat and protein as well as the acidosis increase. Concomitantly, there is an increase in the blood sugar (with the baseline glucose intolerance often compounded by an increased peri-operative output of epinephrine); this hyperglycemia (if not corrected) will lead to an osmotic diuresis, severe extracellular fluid deficit, and potentially life-threatening acidosis. It requires aggressive treatment with insulin, bicarbonate, and intravenous fluid (usually hypotonic salt solutions).

The pre-operative evaluation of the diabetic patient should particularly focus on an assessment of: (a) the severity of the diabetes and the adequacy of its control (oral hypoglycemics or insulin, a history of ketoacidosis or hyperosmolar coma), and (b) the significance of associated cardiac or vascular problems.

While a variety of treatment regimens have been recommended and are effective, the goal in the peri-operative management of the diabetic patient is to maintain a relative steady state of glucose metabolism with the blood sugar between 100–250 mg%. Mild to moderate hyperglycemia and glycosuria is better tolerated (and safer) than hypoglycemia. For the insulin-dependent diabetic undergoing elective surgery, the smoothest control with the minimal catabolism results from continued treatment with intermediate acting forms of insulin (NPH or lente insulin, usually at one-half the normal dose, given in the morning before surgery) with supplementation by regular, subcutaneous insulin (dose best determined by four-to-six-hour monitoring of the blood sugar). A glucose-containing infusion as well as glucose monitoring should obviously be continued intra-operatively as well [1].

In the diabetic patient who requires emergency surgery, extended pre-operative preparation is obviously not possible. In these instances, it is important to correct any existing acidosis and to control the hyperglycemia with insulin as well as possible before surgery. The need for adequate fluid resuscitation and correction of electrolyte abnormalities is obvious and fundamental. Surgery should be performed expeditiously, but the risks of operating on a patient who is hemodynamically and metabolically unstable usually make a delay of a few hours justifiable.

Post-operatively, these patients will often have a significant amelioration of their diabetes, and care must be taken not to allow the patient to become hypoglycemic from overly vigorous insulin therapy.

COMPLICATIONS

The majority of surgical complications in diabetics fall into two categories: infectious and cardiovascular. Accelerated atherosclerosis accounts for the high incidence of myocardial infarction and stroke in these patients. Pratt reported that two-thirds of the diabetic surgical mortality in his series was secondary to cardiovascular complications [12]. Infectious complications are prominently mentioned in most series [3,13,14,15] despite advances in sterility, technique, and antibiotic therapy. Other significant complications (e.g., pulmonary embolus, GI hemorrhage, pneumonia) occur with about the same frequency as in non-diabetics.

DIABETES AND THE GI TRACT

There are numerous gastrointestinal manifestations of diabetes mellitus. Though they are of varying surgical importance, they will be mentioned briefly.

Esophageal dysfunction, though rarely a source of complaint in the diabetic, has been detected radiographically and manometrically. The abnormalities observed include decreased peristalsis, delayed esophageal emptying, and low UES pressure.

Poor gastric emptying and associated motor dysfunction occurs in as many as 30 percent of patients with diabetes [16]. "Gastroparesis diabeticorum" can cause delayed emptying, a sense of fullness, and, often, a marked difficulty in controlling the diabetes itself, as food is not delivered to the small bowel at a reliable rate. Surgical drainage procedures (e.g., gastroenterostomy or pyloroplasty) have been attempted for some time with equivocal results [17].

Recent evidence suggests that metoclopramide will improve both gastric emptying

and the symptoms of gastric stasis in some of these diabetic patients [18]. Patients who fail metoclopramide therapy continue to be a major surgical challenge.

Diabetics also have an increased incidence of parietal cell antibodies, mucosal atrophy, and pernicious anemia [19].

Constipation in the diabetic is felt to be secondary to an autonomic neuropathy although it rarely reaches surgical attention. Diarrhea has been reported in up to 7 percent of diabetic patients and may also be attributable to a neuropathy and secondary bacterial overgrowth; the evidence for this is, however, equivocal.

PANCREAS

It has been suggested that there is a higher incidence of pancreatitis in diabetics than in non-diabetics. A large autopsy series reported an incidence of pancreatitis of 11.2 percent in diabetics as opposed to 5.3 percent in non-diabetics. These findings were attributed to occlusive vascular lesions noted in the arteries supplying the pancreas of the diabetics [20]. Whether this interesting, apparent difference is clinically relevant remains unproven. The incidence of pancreatic carcinoma has been reported to be twice as high in the diabetic as in the non-diabetic [21]. The reason for this is unknown. The surgical approach to both these diseases is similar to that in the non-diabetic.

PEPTIC ULCER DISEASE

Gastric ulcer appears to occur with the same frequency in diabetic and non-diabetic populations. Duodenal ulcer has, however, been reported to have a lower incidence in diabetics. This negative correlation is not well understood but may be due to the neuropathy which creates an "autovagotomy" resulting in decreased acid secretion [22]. Complications of duodenal ulcer, are, however, more frequent and severe in diabetics [2].

BILIARY TRACT

The question of a positive association between cholelithiasis and diabetes remains controversial and unconfirmed. It has been reported that the autopsy incidence of gall stones is about 30 percent in diabetics as compared to 20 percent in non-diabetics [23]. Any significant increase in the incidence of gall stones in diabetics may, however, be related more to obesity and hypercholesterolemia than to the diabetes itself. While the presence of diabetes has not been clearly shown to increase the likelihood of cholecystitis in a patient with gall stones, it has been repeatedly suggested that acute cholecystitis has a more fulminant course and graver prognosis in diabetics. Mortality rates of between 11 and 22 percent have been reported in diabetic patients with acute cholecystitis as compared to a mortality of less than 1 percent in non-diabetics. Emergency cholecystectomy in this setting has resulted in an operative mortality in diabetics as high as 20 percent with a 50 percent post-operative complication rate [14,15,23].

In addition, it has been noted that the infectious and necrotizing components of acute cholecystitis are more rapidly progressive in diabetics, with some series reporting an incidence of necrosis of the gall bladder wall as high as 70 percent in patients undergoing cholecystectomy [14]. Perforation and gangrene can be seen within 24 hours of the onset of symptoms. The explanation for this is unclear, as there is no evidence of vascular insufficiency or neuropathy in the gall bladder bed of these patients. Also, there is no glucose in human bile, so the argument cannot be made (as

in the urinary tract) that bacterial overgrowth progresses more rapidly in diabetic bile. While these data are alarming and have significantly affected surgical decision making in the diabetic patient with biliary tract disease, more recent studies have raised important questions about these past conclusions [24]. Whether this increased morbidity and mortality is related to the diabetes alone or whether it results from the increased incidence of obesity, vascular disease, and renal disease seen in diabetics remains unresolved. It seems probable that there is a sub-group of patients with complications of their diabetes in whom both acute cholecystitis and biliary tract surgery represent a significantly increased risk while the results in uncomplicated diabetics may well approach those in normals. In view of the data currently available most authors agree that cholelithiasis in the diabetic (even if asymptomatic) should be treated by elective cholecystectomy. An emergency cholecystectomy is indicated if an episode of acute cholecystitis does not rapidly resolve with medical management.

APPENDICITIS

There is little in the literature regarding the association of diabetes with other abdominal surgical conditions. One series of 60 cases of diabetics undergoing surgery for acute appendicitis reported a "strikingly large number of patients with atypical appendicitis." These patients often presented with atypical signs and symptoms, such as absence of pain or leukocytosis, which delayed diagnosis and operation [25].

MESENTERIC VASCULAR DISEASE

In view of the accelerated atherosclerotic vascular disease seen in diabetics, mesenteric ischemia and its complications should always be considered by the physician treating an older diabetic patient with abdominal complaints, especially if the patient has co-existing coronary, cerebral, or peripheral vascular disease. Acute mesenteric ischemia is a diagnostic and therapeutic challenge in diabetics as well as non-diabetics. Its etiology may be arterial or venous, occlusive or non-occlusive. The arterial occlusion may be caused by an in situ thrombosis of an already diseased and stenotic vessel or an embolus dislodged from some proximal site in the arterial tree; non-occlusive ischemia is associated with low-flow states and results in diffuse, splanchnic vasoconstriction (most often in a setting marked by congestive heart failure, hypovolemia, or vasoconstrictive agents like digitalis). Patients with acute mesenteric ischemia rarely develop significant abdominal findings (peritoneal signs) prior to the development of full-thickness intestinal necrosis and gangrene. The obvious clinical challenge is to make the diagnosis prior to this stage of irreversible infarction and institute therapy that will stop the progression of the ischemia. In a patient whose symptoms develop in a clinical setting compatible with mesenteric ischemia and who is having severe abdominal pain that is out of proportion to the physical findings (and not readily explainable on a non-vascular basis) angiographic confirmation of the diagnosis of mesenteric ischemia should be sought. If non-occlusive ischemia is confirmed angiographically by the finding of diffuse, splanchnic vasoconstriction, superior mesenteric artery perfusion with vasodilating agents (e.g., papaverine) should be instituted. If an arterial occlusion is documented, the need for surgical intervention should be guided by the clinical evidence of ongoing ischemia. For patients who come to surgery, all infarcted bowel must be resected; revascularization should be undertaken to preserve the marginally perfused but reversibly ischemic intestine (embolectomy if the etiology is embolic; bypass if there

has been thrombosis of a stenotic arterial segment). The challenge to the surgeon lies in early intervention (before extensive ischemia mandates massive resection) and in accurately assessing the extent of viability (realizing that the serosal surface of the bowel is the last of the bowel layers to become ischemic). It is obvious that the cost of a late or missed diagnosis is high: the outlook is grim, indeed, for an elderly, diabetic patient with diffuse atherosclerosis in whom the only therapeutic alternative is an emergency massive intestinal resection for extensive and irreversible ischemia.

DIABETIC ACIDOSIS AND ABDOMINAL PAIN

A confusing aspect of surgical diagnosis in the diabetic with abdominal complaints is the presence of acidosis. While acidosis can be precipitated by an acute surgical intra-abdominal condition, diabetic ketoacidosis alone often presents with abdominal complaints and indeed can mimic an acute abdomen (with pain, nausea, and vomiting associated with signs of peritoneal irritation). These patients may also have a significant leukocytosis. Attempts to rule out significant intra-abdominal pathology can be extremely difficult. In these cases, several approaches may be helpful. A careful history obtained from the patient or his family may indicate an intra-abdominal condition that existed before the onset of symptoms (such as gall stones, diverticulosis, or peptic ulcer disease). The sequence of symptoms may be helpful. Diabetic acidosis usually presents with nausea and vomiting, followed by pain (often upper abdominal), and sometimes drowsiness, and dyspnea. This is in contrast to the characteristic presentation of many intra-abdominal conditions, particularly appendicitis, in which the pain usually precedes the nausea and vomiting. The evaluation of the patient should proceed at the same time the diabetes and acidosis are being appropriately treated. Abdominal findings secondary to acidosis alone should improve with correction of the acidosis. If they do not improve and signs of peritonitis persist, surgery is usually indicated. Allowing several hours to treat the underlying acidosis is not only of diagnostic value but, as mentioned previously, is mandatory in the event that the patient subsequently requires surgery.

In a reported series of 211 episodes of severe diabetic metabolic decompensation, 46 patients (22 percent) had associated severe abdominal pain and tenderness. In seventeen of these (37 percent) an intra-abdominal source for the pain and metabolic decompensation was found. In these cases the location of the pain correlated fairly well with its source except in pyelonephritis, which had a variable presentation. The two cases of appendicitis in the series presented with right lower quadrant pain. The episodes of abdominal pain for which an abdominal source was never found all occurred in insulin-dependent diabetics less than forty years of age. In these patients only three had a plasma bicarbonate of greater than 10 mEq/liter. The authors conclude that "abdominal pain occurring in patients more than forty years old, irrespective of the plasma bicarbonate level, and in patients of any age with a plasma bicarbonate level in excess of 10 mEq/liter, almost invariably indicates a specific underlying cause" [26]. These guidelines, in addition to frequent re-evaluation of the patient during treatment of acidosis, should help define those patients requiring early exploration for an acute intra-abdominal problem [27].

SUMMARY

Diabetes has significant implications both as a risk factor in patients undergoing surgery and as a factor affecting the pathogenesis of a variety of intra-abdominal conditions. The management of diabetes and its complications is a common part of any surgical practice. Aside from the life-threatening metabolic derangements which

can occur in diabetics with surgical problems, surgical decision making is particularly affected by an awareness of the diabetics' propensity toward infection and accelerated atherogenesis. Because of these, the incidence of surgical complications in diabetics is significantly higher than that in non-diabetics. Careful, anticipatory management of the diabetic patient in the peri-operative period is essential to minimizing these complications.

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