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Discussion

DR. JOHN M. HOWARD (Toledo, Ohio): I should like to pay tribute to a wonderful presentation.

It seems to me that one of the important things is the prevention of abscess. As we are improving at carrying patients through the acute episode, we are salvaging patients that would have died a number of years ago, and we are seeing patients with massive necrosis who survive without abscess.

I wonder perhaps if prophylactic antibiotics, during the time when we have many portals of entry, are playing a role in prevention.

(Slide) Here is a patient. This is a debrided necrotic tissue, necrosis without abscess, debridement of almost the entire pancreas.

(Slide) This is a necrotic pancreas debrided 5 months after the acute onset. We thought we were going in to drain a pseudocyst, but there was no pseudocyst. The tissue was dried, almost like a peat bog, with this necrotic pancreas.

In my experience, the things that have predisposed to infection are early operation or ERCP in the presence of necrotic pancreas. As we are not draining pancreases early, we are not seeing abscess very often.

(Slide) Finally, we have recently surveyed our experience over a period from 1962 with nonoperative management of acute pancreatitis excluding posttraumatic, postoperative drug-induced pancreatitis, and our mortality rate is 0.3%.

DR. GABRIEL A. KUNE (Melbourne, Australia): I very much enjoyed Andy Warshaw's paper, and although his view of the world is from Boston, this is kind of our view of how we visualize the world (Slide). Thus, there is a difference in our geography.

(Slide) But the interesting thing is that if I had to give that paper, it would have been in almost exactly the same way as he has done it, right down to the last decimal point.

We have also experienced a continual improvement in the survival of pancreatic abscesses, and this is our experience in the last 15 years, let's say not a quantum leap but a quantum pull with the advent of nutritional support and CT scanning. Thus, we, as have they, have experienced an incredible, improvement in the survival of these people.

At the same time, the number of complications and the number of reoperations that are needed are almost exactly the same as are his.

The other comment I would like to make is that percutaneous drainage of the original abscess has been recommended in several places, but we really would be against that because we have been as unsuccessful as his group has at this procedure, mainly because the original abscess is really an infected slough. This type of situation would be rather difficult to remove with a needle, and, therefore, we would advocate an open operation in exactly the same way that he performed it.

For postabscess recurrence, we also have had good luck with percutaneous drainage, but not with the original abscess. That is my comment, and my question is the following (and this has also been alluded to by Dr. Howard). In the last 5 years, we have started systematically to do necrosectomies and sequestrectomies in the severe cases. It is our impression that, although we do not have sufficient data and sufficient numbers to support it, we have decreased the number of subsequent abscesses with this type of operation. Our problem is that we cannot

diagnose, even with CT or other means, who are the people that have substantial necrosis. My questions to Dr. Warshaw are: Has his group done systematic necrosectomies? Has it decreased the subsequent incidence of abscesses? And also, can they pick up the sort of person who needs a sequestrectomy and a necrosectomy?

Thank you for the privilege of allowing a surgeon from "down under" to attend this august meeting.

DR. JOHN H. C. RANSON (New York, New York): I, too, would like to congratulate Dr. Warshaw on the excellent result he has achieved with this difficult problem of pancreatic abscesses.

I agree with him that the appropriate timing of surgical intervention, radical debridement of necrotic tissue, and adequate drainage together with meticulous supportive care are the key to the management of these patients.

He has stressed the value of computed tomography (CT) in early diagnosis and mentioned possible drainage of these collections before infection occurs. Other authors have also recommended that the demonstration of a fluid collection in acute pancreatitis is by itself an indication for drainage. We, however, recently reported that in 54% of patients with acute pancreatitis and a pancreatic collection on CT, the fluid resolved completely without specific treatment, and I would like to ask my first question, therefore, which is: What criteria other than CT findings of a fluid collection led to surgical intervention in these patients, and what proportions of the patients in the recent series were, in fact, infected at the time of initial surgery?

Secondly, it has been our experience that the morbidity and mortality rates of patients with pancreatic abscesses are clearly related to the severity of the underlying pancreatitis. In this regard, the morbidity rate is relatively low in patients whose initial pancreatitis is treated elsewhere, but who are well enough to be transferred for treatment of their abscess. Do you, therefore, have any information about the severity of the underlying pancreatitis in your two time periods, and what proportion of patients were transferred to you from other hospitals for management of a pancreatic abscess?

Finally, I note that pulmonary embolism was responsible for only one of your deaths. In our experience, such emboli have been so frequent in this group of patients that we routinely treat them with heparin. Do you recommend any specific measures to prevent this particular complication?

DR. JOSEPH M. CIVETTA (Miami, Florida): I would like to compliment the authors for achieving such an outstanding survival rate in patients with pancreatic abscess. They emphasized the use of computerized tomography as a means to confirm a specific diagnosis. Indeed, it was specific in 74% and helpful in 95% of their cases.

We examined the use of abdominal CT scans from a different perspective: to search for a source of sepsis in postoperative patients in the ICU. In 77% of the scans, the information was of no value or was actually deleterious. In fact, CT scans provided little information that changed the clinical management or outcome. Thus, we agree that abdominal CT is useful to confirm a specific diagnosis as demonstrated in this paper, but feel it should not be used as a screening tool to try to discover an unsuspected septic source.

I wonder if the authors have any information concerning the per cent and utility of CT scans on other patients with persistent pancreatitis but who did not have abscesses. Also, I would like to ask them to characterize the concepts of "earlier diagnosis" and "earlier intervention" for our future use. I certainly agree with the principles but have often been perplexed in attempts to identify the proper moment to initiate diagnostic and therapeutic steps in these very complex patients.

DR. HERBERT B. GREENLEE (Maywood, Illinois): We also looked at the incidence of pancreatic abscess following admission for pancreatitis during a recent 10-year period. Twenty pancreatic abscesses out of 1092 admissions for pancreatitis occurred. This is an incidence of 1.8%, which is similar to yours.

The etiology of pancreatitis resulting in pancreatic abscess differed somewhat from yours in that almost one-half of the pancreatic abscesses occurred in the setting of postoperative pancreatitis. This finding supports my bias that postoperative pancreatitis is a particularly lethal problem.

We also divided our review and analysis into two 5-year sequential periods from 1970 to 1975 and 1975 to 1980. There was a significant decrease in mortality during the second 5-year period. We also attempted to analyze the reasons for the improved mortality rates during the more recent period. We concluded that more effective drainage with suction tubes plus wider and more radical debridement of the pancreatic abscess was accomplished during the latter period. In addition, the availability of hyperalimentation permitted us to provide better nutritional support.

I have several questions for Dr. Warshaw. First, do you believe that there is a disproportionate incidence of pancreatic abscess subsequent to pancreatitis that develops in the postoperative setting? Secondly, do you agree that the availability of hyperalimentation and improved nutrition has been an important adjunct in reducing mortality rates in these patients who usually require many weeks of hospitalization with little or no oral intake? Finally, I heartily agree wth your general recommendation to drain pancreatic abscess as early as possible. However, I find it extremely difficult to distinguish between recurrent or persistent pancreatitis, phlegmon, and early abscess. Early and aggressive surgical intervention may, in some instances, not have been necessary, as the pancreatic phlegmon would have resolved on continued conservative management. Do you have any special clinical criteria or diagnostic tests that you particularly rely on to guide you as to the timing of surgery?

DR. JOHN H. SIEGEL (Baltimore, Maryland): I would like to congratulate Dr. Warshaw on an excellent group of patients.

Our experience has been that the critical factor determining whether a patient will survive from pancreatic abscess is whether he develops a septic process that he cannot control, and that the best criteria for establishing whether the patient, the host, is able to have an adequate host defense has been related to the cardiovascular relationships. If he develops an uncontrolled hyperdynamic state with a drop in vascular resistance and especially if oxygen consumption begins to fall, that is a good indication that the patient is failing to control his septic process. In our experience, this has been more characteristic than the CT scan, which often may not show a clear abscess until quite late in the process.

The group in Milan, I know, has used this criterion for intervention, as have we, and I think that they have published a series that demonstrated that aggressive pancreatic debridement in the face of rising hyperdynamic response results in a better outcome than in those patients in whom drainage only occurred at that point. I think that has been our experience as well.

I would also like to comment on the nutritional aspects. It seems to us that this is one of the major components in any septic process, that is, the use of aggressive nutritional support. There is some evidence that one can reverse or alter the acute phase protein response that may have something to do with host defenses. This is an area where investigation needs to be carried out aggressively, but it does seem to have a major role in our experience as well.

DR. JONATHAN E. RHOADS (Philadelphia, Pennsylvania): Until about 3 years ago, I think I had never seen a pancreatic abscess complicating carcinoma of the pancreas. At about that time, the interventional radiologist became quite enthusiastic about needle biopsy, passing the needles through whatever was in the way to get a few cells, and since then we have had two abscesses that presented as abscesses in patients who turned out eventually to have carcinoma. The diagnosis of carcinoma was not made with the needle in either case. It emerged much later.

May I ask whether you have encountered this phenomenon?

DR. DONALD C. MCILRATH (Rochester, Minnesota): I rise to compliment Dr. Warshaw for his excellent results, especially those in the second 5-year period of study.

I would like to ask him three questions regarding the debridement and increased drainage that he feels is responsible for the improvement in results: (1) do you approach the lesser peritoneal sac only through the transverse mesocolon, (2) do you bring the Penrose drains through one opening in the abdominal wall, and (3) was there any difference in the incidence of pancreatic fistula between the two periods of study? The incidence seems extraordinarily high, and I wonder whether or not this could be related to the increased debridement.

DR. ANDREW L. WARSHAW (Closing discussion): I am grateful to the Association for this opportunity to present two more papers. There is no doubt in my mind that the necrotic tissue that develops in a subgroup of patients with pancreatitis is the absolute key as to whether or not they are going to develop pancreatic abscess. The problem is, as has been stated by a number of questioners, how do you know when it is present.

There does appear to be a disproportionate incidence among patients whose pancreatitis follows operations on the pancreas or nearby structures. It is certainly not the only cause but postoperative pancreatitis does seem to be a worse kind.

In general, the severity of the preexisting pancreatitis, as Dr. Ranson and a number of others have shown, clearly appears to be an important determinant as to whether an abscess later develops.

I do not have at hand the information as to how many of the patients in this series were transferred to us, rather than having their pancreatitis set off at our institution. It may be correct that, if there is time to transfer, a group of patients with lesser severity may be selected. However, some rather severe pancreatitis is transferred to us as well.

Dr. Siegel, your comments about the hyperdynamic state are of particular interest, but I think one must distinguish between different causes of hyperdynamic state. Your studies and those of others who point out the cardiovascular characteristics of sepsis are pertinent to the patient with the abscess, but an almost identical hyperdynamic state has been observed by Beger's group from West Germany in patients with uninfected, sterile pancreatic necrosis. Thus, the features of the hyperdynamic state could not discriminate between necrotic tissue which is infected and that which is not.

The question of the natural history of necrotic tissue *versus* fluid is an interesting one. The CT scanners are very interested in sticking needles into almost anything these days, and we certainly can document now the appearance of this peripancreatic fluid in many such patients. In some of these patients, the peripancreatic fluid collections (acute pseudocyst might be an appropriate label) does get reabsorbed and is not necessarily a sufficient reason to operate on these patients. In the future, as we begin to gain experience in this area, it may be rational to needle this kind of fluid to see whether it is infected or not. If it is infected, then I suppose that we had better get to it and drain it. If it is sterile, perhaps we can wait to see if it will be reabsorbed.

The problem of determining how much necrotic tissue is there, whether it is a small amount that could be spontaneously cleared or whether it needs to be debrided, is one that we do not claim to have solved. CT has been of some help to us in this regard, but CT shows us relatively late and unsubtle changes. Many radiologists call edema the irregular lucent areas that I depicted. We do not know how accurate you can be in determining whether a given lucent area is dead tissue or edema. Contrast enhanced CT scans may be of help here, as the Scandinavians believe. We do use the CT scan, not in every patient, but in those patients who have persisting signs. The patient who gets better in a day or two does not generally get a CT scan in our institution. We begin to worry about those who stay toxic for several days to a week, and those are the ones who get CT scans.

Dr. Civetta, you are quite correct. If I gave you the figures on all CT scans done in pancreatitis, we would show a much lower yield because there would be many more negative scans. The figures shown today are obviously derived from those who were found to have an abscess. Nonetheless, if we focus our surveillance on severe and persistent disease, I think we can justify the studies. Once we start, we do them approximately once a week to see what are the developments and to try to determine when to intervene.

We have a continuing interest in the measurement of serum ribonuclease as an index of pancreatic cell necrosis. I did not have time to mention it during the regular portion of this talk, but 11 of 14 patients in whom it was measured in this group of patients had an elevated serum ribonuclease. We are continuing to look at that as an adjunctive measurement, in some cases more sensitive than CT but perhaps in some cases too sensitive, to help select patients for operation.

We do believe that getting at the sterile necrosis before it becomes infected is of value. In my own group of patients, in the last 3 years I have debrided 20 such patients with sterile necrosis and had 19 survivors. That is the same as the 5% survival in the abscess group, but we are getting to the abscess patients early, too. We do not know the proper time to operate. We are doing it earlier and earlier. The criteria are still under development.

Clearly nutrition is of importance. I did not make more of it simply because, as far as I can tell, the nutritional support of our patients in the two different time periods was not better in the second than in the first, so that I could not use it to explain the differences.

We have not recognized pulmonary emboli as an important problem in our experience and do not use heparin routinely.

Finally, Dr. Rhoads, we have in this group of patients one patient out of the 45 whose preexisting lesion leading to the abscess was a pancreatic cancer.