Symposium

Management of the Acute Complications of Diverticular Disease:

Peritonitis and Septicemia

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DR. CONDON

Thank you, Dr. Gathright. There is some peritonitis in every patient who has symptomatic diverticular disease, but that is not really the sort of problem I need to address. What I really need to review with you is fortunately uncommon, but extremely serious, perhaps the most serious complication of diverticular disease, and that is spreading peritonitis. Material that leaks out of the colon, or bursts out of the colon, spreads throughout the peritoneal cavity, principally by two mechanisms. Simple gravity drainage drains material both into the pelvis and up into the subphrenic spaces. Dr. Robert Monk, who practices surgery in Waukesha, Wisconsin, demonstrated ten years ago that the usual concepts of spatial distribution of the abdominal cavity are erroneous and that, in fact, you've got two valleys between a peak. The peak is the area of the lumbosacral joint, and one valley we all recognize as the true pelvis; the other is the subphrenic spaces, which also run downhill. The distance from the lumbosacral joint to the subphrenic spaces isn't as long as you might think it is. The other mechanism that spreads things around in the belly is respiration. Every time the diaphragm moves, suction is created and fluid is literally pulled from the lower abdomen into the upper abdomen.

How does the peritoneum respond to the insult of fecal material? Well, the first response is a transudative one; it is characterized by edema and vascular congestion. The transudation of a low-protein fluid content into the abdominal cavity in peritonitis is accompanied by diapedesis of large numbers of polymorphonuclear leukocytes; their purpose obviously is to phagocytize bacteria and other foreign bodies. The problem during this engorged, vascular and transudative phase is that the peritoneum acts as a two-way street, so that bacteria and, more importantly, bacterial toxins, leave the peritoneal cavity, enter the lymphatics and the blood stream, and lead to symptoms of sepsis.

A patient undergoing systemic septicemia has a real problem; you are all familiar with much of this, but let me briefly touch some high spots. First, there is a decrease in blood volume, partly due to loss of fluid to within the peritoneal cavity, but also partly due to venous pooling as a consequence of the effects of coliform endotoxemia. In addition, there is difficulty with the heart. Cardiac output decreases at this stage as a primary response to the endotoxemia. The poor circulation leads to a shift from aerobic to anaerobic metabolism

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in muscle and other peripheral tissues and, as a result, anaerobic end-products of carbohydrate metabolism accumulate and the patient begins to develop lactic acidosis.

Another thing that happens in endotoxemia is that blood vessels between the cortex and the medulla of the kidney—those special vessels so important in controlling cortical and glomerular blood flow, which are particularly sensitive to endotoxin—undergo a kind of squeeze, leading to cortical ischemia and a consequent reduction of urinary output. This then leads to acidosis of a more general sort, and the accumulating and progressing circular problem now is that acidosis brings about secondary dysfunction in cardiac contractility and a further decrease in cardiac output, and that compounds the problem.

Finally, there is the important aspect of renal failure. I brought along one slide, not because it is so exciting, but because it helps to show how all of these things interact. (Slide, please.) We have talked about inflammation. Distention is the reaction of ileus. It leads to elevation of the diaphragm and restriction of diaphragmatic motion secondary to pain, and that leads to atelectasis and intrapulmonary shunting and to peripheral hypoxia. The metabolic demands of sepsis and infection increase tissue demand for oxygen; but instead of getting it (as we have just reviewed), there is poor cardiorespiratory function and an increasing acidosis. Muscular fatigue, inefficient respiratory mechanics, inefficient intra-lung pulmonary function, progressing hypoxia, and combined metabolic and respiratory acidosis culminate in death unless the process is reversed.

Respiratory failure plays an important role in the deaths of patients suffering peritonitis and sepsis. The treatment of this situation is obvious from a review of the abnormal physiology. Patients need volume replenishment; they need their circulation restored; they need sepsis brought under control with antibiotics; they need short-term—perhaps long-term, depending on the outcome—support of renal function, initially, by restricting potassium intake, and, subsequently, by providing appropriate dialysis and by looking for all of the complications of renal failure. In terms of respiratory failure, patients need oxygen support and may require respirator support if the disease becomes advanced.

How does a patient get into this mess? Well, there are essentially three different mechanisms that lead to spreading peritonitis in patients who have diverticular disease. The first is spontaneous rupture of a diverticulum; that is a relatively rare event. Peritonitis following rupture begins as a stercoraceous inflammation. There is an immediate local peritoneal response and, most of the time, the perforation is contained. As Dr. Griffen mentioned, free perforation may occur early in any attack, but if it occurs later in the disease, it represents failure of normal peritoneal responses, which ordinarily pretty well wall off the slowly progressing typical diverticulitis.

The second mechanism leading to spreading peritonitis is related to the unfortunate consequences of extrusion of barium from an inflamed diverticulum during the course of a barium-enema examination. The barium enema is an important diagnostic tool; even when patients have relatively acute symptoms, it is indicated in at least some. All of us and our radiologic colleagues are aware of the possibility of this complication, and we take great pains to make sure that there is no excess pressure and so on; nonetheless, perforation of a diverticulum with extrusion of barium does occur in a small proportion of patients. It is very frustrating to watch; the can is quickly lowered, the rectum evacuated, but the barium keeps oozing out into the belly and spreading around. You

know there is going to be a lot of trouble very shortly.

The third mechanism is perhaps the most common in this uncommon complication: that is, re-perforation, or secondary perforation, of a previously walled-off abscess. The rupture initially is localized. A big abscess, often the size of a grapefruit, grows and finally ruptures, spreading pus through the belly. This is the commonest way in which spreading peritonitis occurs in diverticular disease.

Each of these three different mechanisms produces a somewhat different clinical picture and somewhat different pathophysiologic response. The spontaneous free rupture of a diverticulum spills both feces, that is, vegetable matter, and fecal bacteria into the peritoneum.

As I mentioned to you this morning, the fecal microflora consist of a complex mixture of aerobic and anaerobic organisms but, in an acute situation like this, there are two organisms that are of prime concern: *Escherichia coli* and *Bacteroides fragilis*. One is an aerobe; the other an anaerobe. Neither is sensitive to penicillin. *E. coli*, in general, is sensitive to aminoglycoside antibiotics. *Bacteroides* is sensitive to a more limited group consisting of clindamycin, chloramphenicol, tetracycline, and erythromycin. In the context of an acute, spreading peritonitis, systemic antibiotic treatment is warranted. I would pick either kanamycin or gentamicin for the aminoglycoside and clindamycin in high systemic doses to handle the *Bacteroides* component.

You can recognize this particular syndrome clinically because the patient generally has rather mild symptoms at onset, followed by acute and relatively rapid progression of symptoms. But it is not the kind of clinical picture where the right hand of God comes down to strike the patient, as is so typical of a perforated ulcer.

The second mechanism, involving rupture of a "tic" and leakage of barium, in addition to spilling fecal material, also spills barium. You all recognize that barium-fecal peritonitis is a particularly lethal condition, but the reasons for that lethality are not completely understood, so let me remind you of some information. Barium without feces, instilled into the peritoneal cavity of an experimental animal, produces an exudative, sterile, chemical peritonitis. There is a significant shift of extracellular fluid volume to within the peritoneal cavity and, in addition, a severe desmoplastic reaction to the barium.

These reactions occur in the total absence of any infecting organism, so that barium, of and by itself, produces a severe form of chemical peritonitis. Add to this the fecal contamination and the fact that bacteria get entrapped in the fibrinous and desmoplastic reaction and you can see that you have a particularly difficult form of peritonitis to treat. It's hard to get the barium out; it is almost impossible to get the bugs out by irrigation, wiping or anything that you can do during operation. You and your patient have to depend on the slow walling-off of all these small nidi of barium and bacteria to accomplish resolution of the process. Clinically, this syndrome is easy to recognize; it occurs in the radiology department in the context just outlined. The patient experiences sudden onset of pain during the course of the barium enema.

Finally, let us consider perforation of a previously walled-off abscess. This is a situation in which the right hand of God may come down and go "whack" on the abdomen. There is sudden onset of pain which very rapidly, within a minute or three, spreads diffusely across the lower abdomen. There is very early onset of toxemia and prostration. In this situation you've got not only viable bacteria but also a mixture of bacterial toxins, all of which,

when absorbed, will systemically affect the patient. The fluid nature of this purulent material leads to its rapid spread to the farthest corner of the peritoneum.

So, this is a situation that produces the worst early picture, but it's perhaps the most amenable to treatment. At least, it is certainly more amenable to effective treatment than the situation in which the feces are accompanied by barium.

Next, some remarks about treatment. Earlier, I was sitting in the corner listening to Drs. Williams and Griffen give my talk about treatment; so there really isn't an awful lot left for me to say. There are some differences in our points of view, so I will very quickly remind you about my views of the treatment of perforation and peritonitis in the course of diverticular disease.

The first thing important to recognize that most of the time the disease process localizes. Patients who are localizing a perforation don't necessarily need an operation. The old, conventional wisdom about bed rest and nasogastric suction, and the provision of appropriate antibiotics, still holds in cases of patients who have localizing, not spreading, symptoms. Sometimes patients are taken too quickly to the operating room, just as, on the other side of that coin, some patients are not taken quickly enough.

The objective of operative treatment is either to remove or to control the source of peritoneal contamination, by whatever means that can be accomplished and that seem best for a particular patient in his clinical context. There are two rules: one, you shouldn't open new tissue planes in the presence of peritonitis, and two, you should never do a colonic anastomosis in the presence of pus.

A couple of other comments about operative treatment—before you take the patient to the operating room, it is essential to restore blood volume, and to ensure that the patient is out of shock, and that effective tissue levels of antibiotics are aboard. I'm really not sure whether we should be using steroids in such patients, but if they get very sick and septic, I use them. In some ways that practice is another surgical amulet, but I'm clinically convinced that giving a couple of grams of prednisolone intravenously probably doesn't do any harm on a one-time basis; it might or might not do some good.

If you are going to explore, do so through a midline incision: 1) it allows access to all corners of the abdomen so you can thoroughly irrigate, 2) it opens up a minimum number of new tissue planes in the presence of infection, and 3) it leaves you with a wound intrinsically stronger and quicker to heal than a paramedian incision. A transverse wound would probably be stronger yet, were it not to get infected, but a transverse wound opens up an enormous number of new tissue planes and I think is best avoided. The first thing to do when you open the abdomen, unless feces are gushing out of a great hole in the colon, is irrigate out the material already leaked. Next, look at the disease and decide what to do in terms of controlling the source of contamination. At a minimum, this requires a colostomy. I agree with my colleagues on this panel that the colostomy should be placed as close to the point of leakage as possible.

Secure suture of a perforation, in my experience, has never really been possible. If they are walled off, I don't tear the tissues open to try to find where the leak used to be. If the perforation is still open and leaking at the time of operation, the edges are retracted, enormously edematous, won't hold sutures. I've never been able to suture one of these things, and I don't try anymore. I can't move the appendices epiploicae, either; they are so stiff and edematous. When I try to turn them over, they just snap back to where they were.

So, I don't think that closure of a perforation, in most advanced cases, is possible.

What needs to be done, if you can, is to get the perforated segment of bowel out of the abdomen. I prefer to resect, if I can and if the patient will tolerate it; but if not, then I will do a colostomy. Then when I have gotten through with that, I irrigate again. I think there is a great advantage to a lot of irrigation. I get the nurses to hang up two Kelly flasks and find us some long pieces of hose; then we pour lots and lots of saline solution through and after we have irrigated about 20 liters, using the hose just like you would use a garden hose to clean off the walk, I let my assistant surgeons go through the same act. By the time 35 to 40 liters of solution have irrigated through the abdomen, I think we have probably removed most of the material that we are going to be able to remove mechanically. The final irrigation is made with a dilute mixture of kanamycin-bacitracin, which is left behind within the abdomen.

In Britain, surgeons sometimes use a substance called Noxytiolin; perhaps Mr. Alexander-Williams can talk a bit about whether he uses it. Noxytiolin is a peculiar compound that slowly breaks down to form formalin within the peritoneal cavity. Those who have used it indicate that it probably is more effective than a dilute antibiotic solution in handling a truly purulent peritonitis.

The issue of drains has come up. You all know it is impossible to drain the general peritoneal cavity. When you have an established abscess it certainly should be drained. If there were special circumstances that had led me to leave a perforation or a weakened or dangerous area of colon behind, I certainly would drain it. But, I don't drain the general peritoneal cavity. If you are going to get an abscess, you are going to get an abscess. Pus will either be cleaned up by the body's defense mechanisms with the aid of antibiotics or it won't; the presence of drains probably doesn't influence the resolution of infection, except to increase the incidence of abscess formation.

In managing cases of purulent peritonitis, the fascia should be closed with nonabsorbable, monofilament suture. I use a running suture of 0 Prolene and do not place retention sutures. I don't think that retention sutures prevent dehiscence. When a patient develops dehiscence after purulent peritonitis, it is because there is a terrible infection in the wound. All the retention sutures in the world aren't going to prevent that. I don't close the skin for 48 hours, and then I pull it together loosely with Steri-strips.

DR. GATHRIGHT

Thank you, Dr. Condon. I think that is sufficient material to last us for the next few minutes.