PERCUTANEOUS DRAINAGE OF INTRAABDOMINAL ABSCESSES

Thesis

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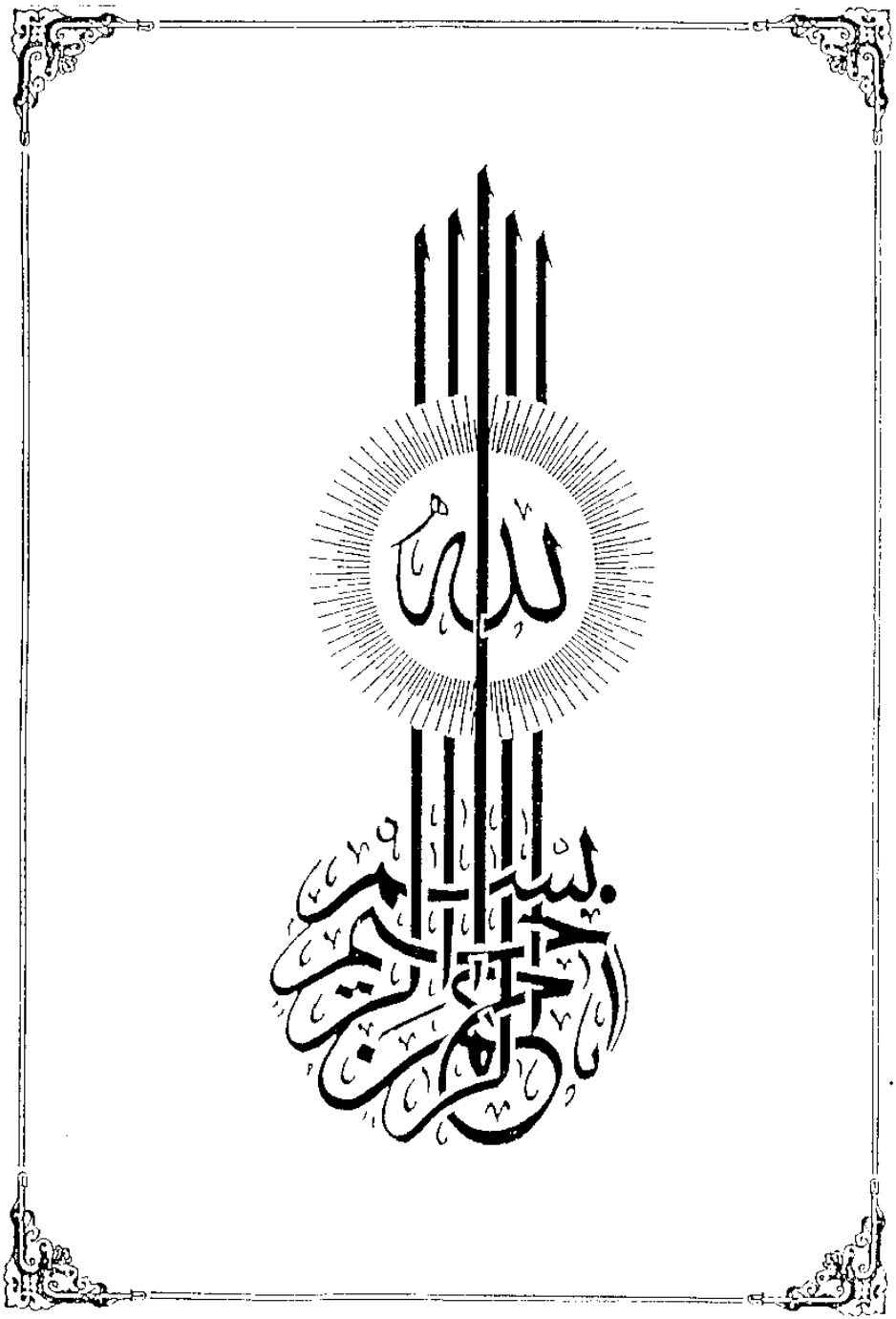

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Infectious complications arising within the abdomen have traditionally been diagnosed and treated by the general surgeon alone, but concurrent advances in interventional radiology have demonstrated the efficiency of percutaneous drainage in the complete eradication of abdominal abscesses **(Hallasz and Sonnenberg, 1983)**. The reported success rates of percutaneous drainage are high (75% to 90%) and mortality rates low (less than 10%) **(Pruett and Simmons, 1988)**. These results stand in marked contrast to published surgical series on abdominal abscesses in which operation and drainage has been characterized by significant recurrence. morbidity, and mortality **(Mandel et al, 1983)**.

Simple, nonfungal, nonfistulous abdominal abscesses were cured with pecutaneous drainage in 96,5 . Percutaneous drainage failure was encountered in infected organised hematomas or thic phlegmons. fungal infection, abscesses with enteric communication, and infected necrotic

tumors. Percutaneous drainage is a valuable diagnostic and therapeutic tool that is curative in simple abdominal abscesses. Its therapeutic role in complex abdominal infections seems to be limited **(Haage, 1990)**.

The aim of this study is to assess the efficiency of percutaneous draiange of intraabdominal abscesses guided by CT scan and ultrasonography and to assess its effect in decreasing both morbidity and mortality in cases of intraabdominal abscesses.

REVIEW OF LITERATRE

NATURAL HISTORY OF ABDOMINAL ABSCESS

Infectious complications of a recent laparotomy are probably the most common cause of reoperation. The initial operative procedure may have been for acute peritonitis, with the magnitude of bacterial contamination being so great that an abscess forms and a subsequent reintervention for drainage is necessary. In patients who have sustained penetrating abdominal trauma, the combination of bacterial contamination and adjuvant factors (hemoglobin, dead tissue, foreign bodies, schock, transfusion) may result in abscess. Not uncommonly, complications of elective abdominal procedures such as anastomotic leak necessitate a drainage procedure for infection **(Fry and Clevenger, 1991)** .

Infections within the peritoneal cavity , regardless of its cause, has a peritonitis phase. Bacteria are released into the peritoneal cavity, and these contaminants are rapidly disseminated because of the multiple influences of gravity and pressure gradients that are normally present. When the human body is upright, peritoneal fluid seeks a dependent position within the pelvis. However, when the body is supine, the dependent positions of the peritoneal cavity change, with the subphrenic spaces and pericolic gutters also becoming dependent. In the supine position, pressure gradients within the peritoneal cavity result in natural movements of fluid from one dependent area to another **(Bohnen et al, 1983)** .

The piston-like motion of the diaphragm with inspiration and expiration means that a relative negative pressure is created beneath the diaphragm with each expired breath. This natural movement of peritoneal fluid in the supine position, and intraabdominal pressure gradients will spreaqd bacteria that are released into the coelomic space **(Richardson et al, 1983)** .

The dissimination of bacteria by the normal movement of peritoneal fluid actually can serve a nonspecific funstion of peritoneal host defense. All studies of bacterial infection, regardless of location, have focused on the importance of bacterial density at the tissue level before clinical infection develops. If the numbers of bacteria are counted in the urine of patients in whom the house staff has diagnosed pyelonephritis, bacterial counts greater than 100,000 bacteria per milliliter are found in 95% of specimens, pyuria in about half of these. It has been known for may years that bacteria can usually be found in stained specimens of urine if infection is present. About 100,000 bacteria per milliliter must be present in urine for the stained specimens to be consistently read as positive. If the patients have fever, flank pain, dysuria, and pyuria, more than 100,000 bacteria per milliliter of urine are almost always found **(Kass, 1957).**

The clear implication is that bacterial concentrations which do not exceed 10,000 organisms per milliliter of tracheal secretions are seldom if ever associated with respiratory infection. Those patients in whom more than 100,000 organisms per milliliter of tracheal secretions were

identified has respiratory infection regularly **(Polk, 1975).**

Although quantitative considerations of bacterial density in the peritoneal cavity have not been defined, it certainly is likely that there is a threshold of bacterial density that is necessary for infection. The dissemination process reduces bacterial density within a given locale in the peritoneal cavity. It also increases the interface between the microbial invaders and the patient's host defense mechanisms **(Fray and Clevenger, 1991).**

Bacteria are removed by two natural mechanisms within the peritoneal cavity. **First**, the organisms may be ingested and then lysed by phagocytic cells. Macrophages and neutrophils serve this function. **Second**, bacteria may be cleared via lymphatic Fenestrations present on the diaphragmatic surface of the peritoneal cavity. The direct connection of these diaphragmatic fenestrations of the lymphatic system means that the entire peritoneal cavity can be viewed as a giant lymphocele. Peritoneal fluid is thus lymph fluid that is normally cleared via the lymphatic Fenestrations **(Tsilibary and Wissig, 1977).**

Studies by **Hau and Simmons, 1978** have suggested that obstruction of the lymphatic fenestrations by fibrin and peritoneal debris actually interferes with peritoneal bacterial clearance. They also concluded that heparin has a therapeutic effect in experimental canine peritonitis by preventing the apposition of fibrin and, thus, rendering the bacteria more susceptible to cellular and noncellular clearing mechanisms.

O'Leary et al, 1979 have suggested that obstruction of the lymphatic fenestrations by fibrin and peritoneal debris actually interferes with peritoneal bacterial clearance. Bacteria within the peritoneal cavity are naturally moved toward the diaphragmatic surface, but if the egress is occluded, the subphrenic space becomes the natural depot for contaminated peritoneal fluid and subsequent abscess formation.

The quantity of bacteria released within the peritoneal cavity from a biliary enteric perforation dictates the probability of a fatal outcome or the development of an intraabdominal abscess. A perforated peptic ulcer in a younger patient that is managed with prompt surgical care commonly will have a sterile peritoneal culture at the time of operation. This condition is essentially a chemical peritonitis. a perforated ulcer in a patient with a gastric outlet obstruction will have a larger number of bacteria. A perforated appendix will have bacterial counts of 106 to 107 per gram of contaminating intestinal contents, and a perforated colon cancer will have bacterial counts approaching or exceeding 10 10 organisms per gram of colon contents. Although peritonitis and intraabdominal abscess is commonly perceived to be a single illness, it is in reality a constellation of illnesses that have various degrees of physiologic consequences depending on the total number of viable bacteria within the inflammatory response and the efficiency of the host response to that bacterial assault **(Fry and Clevenger, 1991).**

The physiologic consequences of the bacterial numbers are amplified by the local environment within the peritoneal cavity. Adjuvant factors commonly encountered in the peritonitis

patient may make a given inoclulum of bacteria considerably more virulent than would be customarily be the case. Hemoglobin and haematoma are potent adjuvants for bacterial growth. The adjuvant effect may be attributable to the important value of ferric iron as a bacterial growth factor. The kinetics of enhancement suggest that fully saturated transferrin either no longer capable of, or interfere with, non specific host defense **(Polk and Miles, 1971)**

A hematoma within the peritoneal cavity after an operative procedure may be the basis for abscess formation. A contaminated clot within the pelvis or subphrenic space becomes the substrate for bacterial proliferation. Relatively small numbers of bacteria after seemingly uncomplicated elective operative procedures may result in abscess because of the retained intraabdominal hematoma. The adjuvant for bacterial growth may relate to a leukotoxin eleborated by the bacterial metabolism of hemoglobin **(Pruett et al, 1984).**

Both dead tissue and foreign bodies may be adjuvants for bacterial proliferation. Both materials probably represent havens for bacterial contaminants that are not accessible to phagocytic cells. Dead tissue may be retained after a traumatic injury, may arise from excessive "bites" of omentum and other tissues that are strangulated by suture ligatures, or may be present from excessive use of the electrocautery intraoperatively. These variables are commonly identified as being of significance in the pathogenesis of wound infection, and they may be of significance in the pathogenesis of intraabdominal abscess and ultimately be

responsible for drainage procedures that are needed **(Dellinger et al, 1984)**.

Esrig and associate, 1977 suggested that hypovolemia and hemorrhagic shock enhance the frequency and severity of infection. Shock and hypovolemia around the period of bacterial contamination because of tissue underperfusion likely interfere with the efficiency of soft tissue bacterial clearance, allowing lethal infection to develop. Another possibility, they added, is that lethal cellular products are released despite successful phagocytosis and intracellular killing. An explanation not related to the immune mechanisms is that the peritoneum; like the lung, becomes edematous or otherwise altered as an accompaniment to adequate resuscitation and, thus, dilutes host defenses, thus allowing development of local infection.

Acquireed immunodeficiency states are frequently seen in surgical patients suffering trauma, malnitrition, sepsis, and thermal injuries. These immunodeficient states can predispose to potentialy lethal infectious complications. Such patients may also require blood transfusions to replace losses due to hemorrhage, operative procedures, or inadequate bone marrow production. Experimental work on the effects of transfusions in transplant immunology has indicated that such transfusions may also suppress immune function. Animals that received transfusions did exhibit impaired cell-mediated immunity and macrophage migration. Such an effect, although desirable in preventing allograft rejection in the transplant patient, might further predispose the patient with trauma to infectious complications **(Waymack et al, 1986)**.