Online ISSN: 2581-3935
Print ISSN: 2589-7837
International Journal of Medical Science and Diagnosis Research (IJMSDR)
Available Online at www.ijmsdr.com
Volume 2, Issue 6; November-December: 2018; Page No. 179-183

CLOSTRIDIAL MYONECROSIS OF ANTERIOR ABDOMINAL WALL

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Abstract:

This is a rare case report of anterior abdominal wall clostridial myonecrosis developing spontaneously in a young patient without any pre-existing disease or history of any trauma/ surgery.

Extensive debridement of necrotic muscle tissue was done and wound was closed by approximating the overlying rectus sheath and skin flaps (uninvolved). Patient was subsequently managed with broad spectrum antibiotics and fluid resuscitation, but succumbed to overwhelming sepsis.

A high index of suspicion & an aggressive management including thorough debridement, intensive monitoring & resuscitation is essential for the diagnosis & treatment of this life threatening condition.

Keywords: Spontaneous**,** myonecrosis, clostridial infection, gas gangrene, anterior abdominal wall necrosis

INTRODUCTION

Clostridial infections involving the abdominal wall are rare conditions. Classically, they are complications of trauma and more often associated with military casualties than civilian surgical practice. When clostridial infection invades abdominal muscles and causes a gas-forming saprophytic digestion of devitalized tissue, gas gangrene occurs with development of an aggressive, lethal infection unless prompt treatment is instituted. Early diagnosis of this disease is difficult because of the low index of suspicion; secondly the classic sign of crepitus due to gas infiltrating the subcutaneous tissues occurs late. Moreover there are no characteristic radiological signs to detect the disease. The disease runs a fulminant course characterized by overwhelming sepsis and death within a few hours or days. Successful treatment depends on early diagnosis & radical debridement. A smear of the wound showing bacteria resembling Clostridium perfringens is sufficient justification for beginning treatment.

CASE STUDY

A 39 year old male patient came with pain in the abdomen (predominantly in the right iliac fossa), fever & oliguria since 2 days. Patient was non-diabetic, non-hypertensive, had no other co-morbidities. There was no history of any trauma or surgery. Patient also denied any history of intravenous drug abuse. On examination, patient was found to be tachycardiac with pulse of 110/min. Abdominal examination revealed diffuse tenderness most prominent in the right iliac fossa & generalised guarding. Patient was catheterised & found to have a low output with concentrated urine. Vigorous hydration was initiated.

DIAGNOSTIC ASSESMENT:

Ultrasound of abdomen & pelvis was suggestive of mild free fluid with internal echos in the abdomen & mesenteric fat stranding in the right iliac fossa. Appendix could not be visualised. Bowels appeared grossly normal & there was no evidence of abdominal lymphadenopathy.

CT cuts were taken to supplement the USG findings. No gross intra-abdominal pathology was found. No gross abnormality was detected on erect X-ray abdomen.

Total leukocyte counts- 19270
Hb- 15.6
Platelet count- 630000.

MANAGEMENT:

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In spite of aggressive fluid resuscitation patient remained oliguric. In view of clinical findings of rigid abdomen decision to do an urgent exploratory laparotomy was taken.

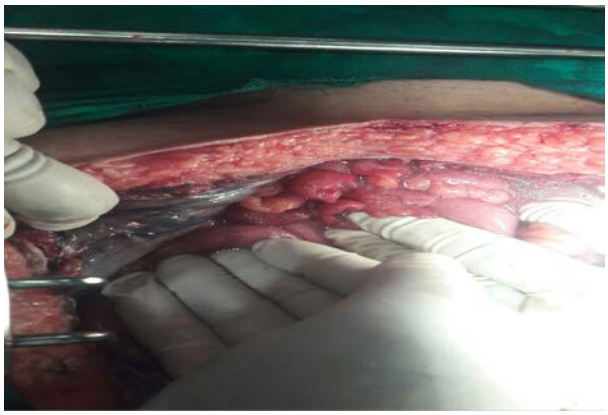

Figure 1: Necrotic peritoneum

Abdomen was opened by a midline vertical incision extending from 4cm below xiphoid to 7 cm below umbilicus. Incision was deepened through the subcutaneous plane, rectus sheath was separated & abdomen was opened in the midline. Fluid was found in the subcutaneous planes. 200cc of foul smelling brownish fluid was drained from the abdominal cavity which was immediately sent for gram staining & culture sensitivity. Whole peritoneum was found to be necrotic and underlying omentum showed few necrotic patches. Bowel loops were explored from the duedeno-jejunum junction up to the sigmoid colon & were found to be normal with no evidence of perforation or pus flakes. Liver, gall bladder, spleen & urinary bladder appeared grossly normal.

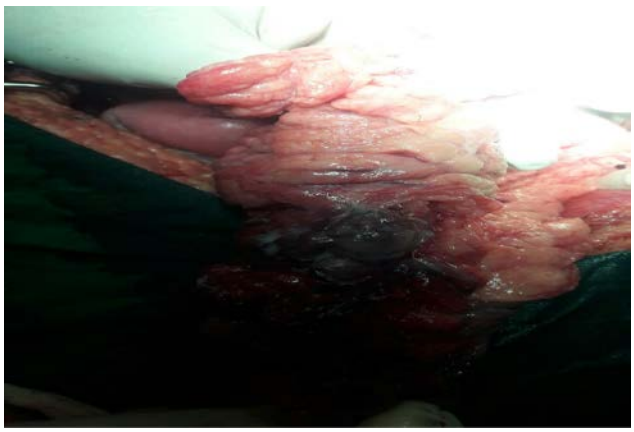

Figure 2: Necrotic patches on omentum

Foul smelling fluid was found to be oozing from the anterior abdominal wall near the right para-colic gutter. On blunt dissection the anterior abdominal wall muscles were found to be completely necrotic and sloughed off up to the level of deep rings bilaterally. All necrotic tissue was debrided radically and sample was sent for gram staining & culture sensitivity. Thorough peritoneal lavage was done with 9000ml of warm saline until clear fluid was seen. As scrotum also appeared edematous,bilateral vertical release incisions were taken over the scrotal wall and fluid was drained following which wash was given with normal saline. Drains were placed in right para-colic gutter & pelvis. Rectus sheath was closed with loop prolene intermittent sutures, subcutaneous tissue was closed with vicryl 2-0 intermittent sutures & skin was stapled. Since the patient could not be extubated, he was put on mechanical ventilation.

Gram staining of the smear revealed few pus cells, gram positive bacilli suggestive of clostridial species, gram positive cocci & gram negative capsulated bacilli. Patient was given clindamycin, metronidazole & pipracillin – tazobactum. Fluids were given as per central venous pressure recording and electrolytes imbalance was corrected. Patient's clinical course was followed with daily blood counts and blood biochemistry. WBC counts started decreasing however patient persistently remained oliguric with persistent rise of serum creatinine, despiteaggressive fluid resuscitation and finally succumbed to overwhelming septic shock on second day after operation.

DISCUSSION:

Gas gangrene is fulminant myonecrosis caused by gram positive bacilli of species Clostridium. Gas gangrene causing Clostridial species include Clostridium perfringes, Clostridium tertium, Clostridium septicum, Clostridium multifermentans. [1]& in IV drug users -C sordellil, & C nayvi [2]

Gas gangrene is produced by exotoxins rather than by invasion of organisms. There is no

direct correlation between in vitro toxin production and pathogenicity.[3] The alpha toxin, a lecithinase, produces hemolysis followed by anemia, hemoglobinuria, oliguria and jaundice. The theta toxin has less but similar enzymatic activity. The epsilon toxin, a hyaluronidase, causes dissolution of the intracellular matrix and, thereby, permits wider dissemination of the infectious process. The activity of several proteolytic and saccharolytic enzymes produces the tissue crepitus characteristic of gas gangrene. Gas gangrene is more commonly seen in the settings of traumatic wounds and ulcers as in warfare in presence of severe devitalised tissues, they are rarely suspected after surgical procedures. Among the earlier reports on clostridial myonecrosis of anterior abdominal wall- Welch [4] described three cases in his Shattuck lecture in 1900. One infection developed after an appendectomy, one after repair of a Littre's hernia, and one after nephrectomy. Gas gangrene has been reported following appendectomy, herniorrhaphy, enterostomy, gastrectomy, cholecystectomy, bowel resection[5, 6] and incision and drainage of para-rectal abscess. Other initiating causes reported are - rectal laceration after passage of a hard stool, scrotal abscess, and decubitus ulcer in paraplegic patients.

Table 1: Diagnostic Symptoms and Signs

Apprehension (early sign)	Profuse watery discharge	Fever	"Sickly sweet" odor
Tachycardia(early sign)	Wound pain	Crepitus in tissues	Jaundice
Extreme restlessness	"Bronze erysipelas"	Hypotension(usual measure for the treatment of shock, fever and jaundice)	

The best single diagnostic measure is a smear of the wound exudate. If clostridial myonecrosis is present the smear will show gram-positive rods with few leukocytes. Clostridial cellulitis will show gram-positive rods with rich bacterial flora and masses of white cells. Anaerobic streptococcal myonecrosis shows chains of streptococci and masses of leukocytes. When clostridia are present as saprophytes, the smear shows a few gram-positive rods and a few tissue cells along with the usual bacterial flora.

The first principal in the management of clostridial myonecrosis is radical debridement of all necrotic tissue to remove the load of toxins entering the systemic circulation.General supportive measures include blood transfusions, albumin, and blood volume expanders to counteract the pathological effects caused by circulating toxins. Fluid and electrolyte balance must be regulated carefully. Continuous nursing care in strict isolation should be instituted. Appropriate parental nutrition should be instituted since the catabolic process induced by sepsis and starvation are associated with nitrogen loss that if un-replaced could be expected to cause death from starvation. [7] Antibiotic therapy must be instituted without delay at the time of diagnosis. Penicillin is the drug of choice and is given parenterally in doses of 20,000,000 units daily. Other drugs that have been advocated in combination with penicillin include chloramphenicol and chlortetracycline. If gram-negative organisms are also present, streptomycin or kanamycin may be given. The role of polyvalent clostridium antitoxin is debated.

It has been shown that neutralizing circulating exotoxin does not affect myonecrosis-toxin interaction which is the lethal mechanism of gas gangrene. Parenteral administration of antitoxin may fail to reach necrotic, ischemic areas where clostridial incubation and exotoxin production are occurring. On the other hand some authorities state that antitoxin has definite therapeutic value. [8,9,10]Polyvalent clostridium

antitoxin should be administered as an adjuvant to surgical debridement and antibiotic drugs , to patients who are not sensitive to a test dose of the equine antitoxin.

Hyperbaric oxygen in the therapy of gas gangrene is a recent advancement. There are multiple series of case reports of patients so treated with excellent response[11, 12]. Hyperbaric oxygen has been shown to be bacteriostatic rather than bactericidal[13] therefore hyperbaric oxygen is a useful ancillary treatment but should not replace surgical incision and debridement.

SPECIFIC CHALLENGES IN THE MANAGEMENT OF LARGE DEFECTS CREATED DUE TO ABDOMINAL WALL GAS GANGRENE

Several points relative to massive abdominal wall defects created as a result of radical debridement need to be addressed-

- Peritoneum widely exposed is not viable
- Continued bleeding from divided muscle is a common problem after extensive debridement for gas gangrene.
- Uncertainty that the infection has been controlled leads to a decision against immediate grafting.
- Abdominal evisceration due to non-granulating peritoneum, and the resulting generalized peritonitis leads to poor outcomes in terms of patient survival.

Table 2: Case reports showing the use of temporary prosthetic placement to the cover the defect to tackle the above problems.

NAME OF THE STUDY	IMPORTANT CONCLUSIONS
Moroggann, morain and eraklis[14]	The peritoneal layer was left intact and was reinforced with a sheet of Mersilene mesh external to it taking appropriate steps to prevent evisceration. On the third postoperative day windows were made in the Mersilene and split-thickness skin grafts applied to the peritoneum. For the following 30 days the original peritoneal grafts were debrided as necessary and immediately replaced by grafts on the underlying viscera. As the grafts gained strength, the mesh was gradually cut away; eventually, permanent coverage was obtained in all areas and the mesh was completely removed.
Mladick et al.[15]	Case report of a patient in whom the entire abdominal wall including peritoneum was excised. Closure was accomplished by allowing the exposed visceral peritoneum to granulate for 3weeks, then placing split thickness grafts on the granulations. This procedure was entirely successful, and is the first report of a patient surviving an abdominal wall excision of this magnitude.
Schmitt [16]	Mesh is sutured across the defect, granulations are allowed to grow through interstices of the mesh, and split grafts are placed on them. Complications of infected sinus formation and excessively delay in grafting because of "gathering" or "corrugation" of the prosthetic material, makes this method less desirable than temporary use of a prosthetic mesh.
McNally.[17]	Reconstructive closure with removal of the mesh before grafting has also been performed successfully
Kenneth Eng et al.[18]	Described a case of extensive clostridial myonecrosis of the anterior abdominal wall in an elderly patient after anterior resection of the sigmoid colon. After extensive resection of the abdominal wall, the defect was bridged by omentum and reinforced with Marlex mesh. Mesh skin grafts were applied to the granulation tissue which formed rapidly from the underlying omentum. This prosthetic abdominal wall proved to be durable despite subsequent reoperation for recurrent carcinoma.
John Phillips et al.[19]	Debridement is carried out through parallel incisions with maximal preservation of skin and subcutaneous tissue. Marlex mesh is used temporarily until the infection is completely controlled. The skin and subcutaneous tissue are then re-approximated, giving excellent wound coverage and markedly shortening the hospital stay.

CONCLUSION:

Clostridial myonecrosis in a young previously healthy patient without any history of trauma or surgery is rarely reported. Therefore a high index of suspicion is required for diagnosing this condition.Patients often present with overwhelming septic shock despite aggressive resuscitative measures the prognosis remains guarded.These patients with signs of overwhelming sepsis but abdominal wall rigidity even in absence of a known pathology merit prompt surgical exploration. Finding of characteristic foul smelling “sickly sweet” brownish fluid with gram positive spore bearing rod shaped bacteria is sufficient for diagnosis & indication for prompt institution of penicillin with or without anti gas gangrene serum. Aggressive debridement of all necrotic tissue is essential for the management of these patients.

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