CLOSTRIDIAL GANGRENE: A CASE REPORT

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INTRODUCTION:

IN AN ERA OF sophistication when antiseptic and asepsis is more or less taken for granted, physicians today tend to neglect the time tested principles in primary wound care.

A case was recently encountered where a traumatic wound following primary surgical care led to clostridial infection of one half of the body, associated with severe toxaemia together with renal and cardiac decompensation. A review of the literature is presented and this led us to the belief that a similar case has not been reported before.

CASE REPORT:

On 2nd January 1978, a 26 years old Chinese male was admitted to the district hospital, Batu Pahat. He had sustained a deep parang inflicted injury on the postero medial aspect of the left thigh in a coconut plantation.

When brought to the hospital, he was in a state of shock and was resuscitated with seven pints of blood. At an emergency exploration a very deep linear incised wound about a foot long was found in the middle third of the left thigh, exposing cut muscles and vessels. After adequate debridement and hemostasis, primary wound closure was done leaving a corrugated drain in situ. Six hourly intramuscular injection of Penbritin 500 mg. was instituted simultaneously.

On 4th January 1978, the patient was referred to the Department of Surgery at General Hospital, Kuala Lumpur. At the time the limb was tense and painful with sluggish venous return. The accompanying laboratory results showed: Hb -9.5mg.% blood urea -67mg.% sodium -137.5 me/L potassium = 4.2 me/L chloride -103.6 me/L.

On physical examination he was toxic and pale but not jaundiced. His extremities were cold and clammy and the left leg was oedematous with cyanosis of the nail beds. The dressing was noted to be drenched with a foul smelling serosanguinous discharge. A repeat laboratory investigation on admission revealed:- Hb - $10.2\text{gm.}_{\odot}^{\circ}$ PCV - 28%and a total leucocytic count of 2,000 cm.

A second emergency exploration of the wound was performed. It revealed massive tissue necrosis with an intact sciatic nerve and a femoral artery. The femoral vein could not be visualised. After extensive debridement of the wound and generous insuffulation with hydrogen peroxide the wound was dressed and left open. Release incisions were made over the calf to combat the intense swelling of the leg.

On 5th January 1978, a venogram study showed a complete obstruction of the femoral vein at its middle third. The laboratory results now showed:-Hb - 9.2gm.% PCV - 33% and a total leucocyte count of 10,000.

On 6th January 1978, the patient was referred to the orthopedic unit whence on examination, he was found to have crepitus in the left leg associated with marked toxaemia and tachycardia that did not correspond to his temperature. The classical serosanguinous discharge and foul smell permeated the



Figure 1

Note air in tissue spaces in the venogram.

room. A diagnosis of gas gangrene was made and the patient was immediately started on intravenous injection of crystalline penicillin 6 mega units 6 hourly. Empirically, intravenous polyvalent gas gangrene antitoxin was also given in divided doses of 75,000 units 6 hourly for a single day. Blood study at this stage showed:- Hb - 8gm.% PCV -28% ESR - 110mm/1st hour with an urine output of 400 mls. in 24 hours.

On 7th January 1978, the urinary output fell further to 292 mls./24 hours and serum analysis revealed urea concentration of 282 mg.% and potassium of 7.2 me/L. The urine was loaded with red blood corpuscles and pus cells. Palpable crepitus in the leg became more pronounced and swab from the discharge revealed gram negative rods and gram positive cocci and grew both Klebsiella and Enterobacter. The tissue culture subsequently yielded a growth of Clostridium welchii, Pseudomonas and Klebsiella. A high above knee guillotine amputation was done under general anaesthesia and in view of his increasing ureamia and hyperkalemia he was referred to the urology unit in the immediate post operative period. He was put on Resornium A retention enema, a restricted protein diet and a strict fluid input/output chart.

On 8th January 1978, the patient was noted to have developed a soft boggy diffuse swelling of the left side of the body, extending from the region of the stump up to the left axilla associated with crepitus. His blood urea continued to rise to 292 mg.% with serum Creatinine of 11.5 mg. Peritoneal dialysis was started during which he was given one unit of fresh packed cell transfusion. The haemoglobin continued to fall despite the blood transfusions.

On 12th January 1978, the haemoglobin was less than 3 gms. The blood urea at this time was 190 me/L, total leucocyte count of 27,000 and urinary output measured 100 mls./24 hours. To combat the acute renal failure a second peritoneal dialysis was instituted and he had to be given another unit of fresh packed cell transfusion. During this period he developed yet a further complication of congestive cardiac failure that needed digitalisation. By 15th January 1978, his renal functions started showing evidence of improvement. The urine output was 1050 mls./24 hours, serum creatinine 5 mg. and the boggy swelling with crepitation on the whole of the left side subsided. On 17th January 1978, the patient regressed to a state of mental depression with violent behaviour. Largactil in doses of 50 mg. b.d. on a sliding dose for one week helped to control this behaviourial disorder.



Figure 2 A mobile patient on crutches.

On 6th February 1978, under spinal anaesthesia autogenous split skin graft was applied over the granulating stump. On 21st February 1978, the skin graft was noted to have taken well. The blood urea was now recorded at 21 mg.% and serum creatinine at 1.4 me/L. Besides plasma expanders he received in all 16 pints of blood, and two sessions of peritoneal dialysis. On 3rd March 1978, he was discharged ambulant on crutches.

DISCUSSION:

In cases with history of trauma especially those sustained in an agricultural area the possibility of primary infection by clostridial organisms must always be borne in mind.

Relatively little attention is now given to gas gangrene and it is seldom remembered that fulminating gangrene can follow elective surgery or present as a primary infection. (Alterneier *et al.*, 1971). Hence, a high index of suspicion must be stimulated to be constantly aware of this threat.

In this case a significant point to note is that primary closure of the wound was done despite gross soil contamination. Also, that there was a lapse of 48 hours between the time of injury and the development of local complaints of severe pain. This would seem to confirm Mac Lennan's (1962) observation on clostridial infections and myonecrosis, that unsatisfactory primary surgical treatment and inadequate debridement are the routine findings in such cases.

The incubation period of Clostridium welchii varies between 24 hours and 3 days. The two factors that are needed to initiate the process are the contamination of tissue and tissue hypoxia and once initiated it follows a relentless course usually leading to a fatal end, as given in a communication by Demello (1970, 1974) and Hitchcock (1970). It is fortunate that while both these factors coexisted in this case, the end result was not fatal. However, other conditions are known to mimic gas gangrene and hence should be considered. These are as described by Michael Barzar of Boston; clostridial cellulitis, anaerobic streptococcal myonecrosis, necrotizing fascitis and synergistic necrotizing cellulitis. Of these infections gas gangrene exhibit the severest form of toxaemia, local pain and swelling.

Taylor (1954), Altemeier (1957) and Langley (1945) outlining the current regime for treatment for clostridial infections recommend that immediate exhibition of an antibiotic is a must even on a suspected diagnosis. Penicillin, being the drug of choice is given parentally in divided doses of 20,000,000 units daily.

Aldrette and Judd (1965) state that antitoxin have a definite value and to be effective the therapeutic dose of the polyvalent antitoxin should be given intravenously, at least 75,000 units and may be repeated every 4-6 hours according to the response of the patient. The prophylatic dose recommended is 25,000 units intramuscularly. (Martindale Extrapharmacopia 1975).

More recently, hyperbaric oxygen is considered. a useful adjunct in the treatment but it should be remembered that this must not replace surgical excision and debridement. This view is supported by Mc Swain, Sawyers and Lawler (1966). Case analysis of 133 instances of proven gas gangrene by Hitchcock (1975) record 9 deaths from cardiac failure, 8 from renal failure, 3 from progressive pneumonia, 1 from pulmonary embolus, 3 due to uncontrolled infection, 1 due to hepatic failure and 1 death resulting from toxicity of hyperbaric oxygen theraphy. This case was proven to be gas gangrene clinically and bacteriologically. The cardinal complication of acute renal failure as seen in this case could have been the result of hypovolaemic shock in view of the fact that massive fluid transfusions were needed to resuscitate the patient. The alpha exotoxins could possibly have further aggravated the situation. However, the accompanying ureamia secondary to renal failure was promptly recognised and adequately managed soon afterwards. The severe anaemia during the acute period despite the frequent transfusion of packed cells shows the devastating effects of the exotoxins - especially the alpha toxin that causes hemolysis with all its attending complications, and theta toxin that is both hemolytic and cardiotoxic. The cardiac failure may have been the result of the latter. The events in this case, conclude and reaffirm that surgery is the cornerstone in the treatment of gas gangrene and that decompression and proper debridement is mandatory as cited by DeHaven et al., (1971).

CONCLUSION:

This affidavit is presented to remind us of the ever present dreaded infection of gas gangrene and its devastating action on target organs distant from the site of primary infection. Furthermore, with early suspicion of the disease process the time tested surgical intervention combined with appropriate antibiotics and supportive measures still remain the sheet anchor in its management.

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