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OSTEOSYNTHESIS INDUCED PULMONARY EDEMA AN OVERVIEW AND CASE STUDY

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ABSTRACT
We covered the etiology, pathophysiology, clinical manifestation, diagnosis, and classification of infection following fracture osteosynthesis with implants in the first section of the article; this illness is referred to as osteosynthesis -associated infection (OAI). Generally, a prolonged course of antibiotics is required. OAI permits implant retention and fracture bealing despite infection. There are five standard treatment approaches that vary based on the degree of infection, fracture healing status and host role. They are debridement with implant retension, implant removal, conversion of fixation, suppression therapy and npn-operative therapy, each therapeutic pathway and its tough scenarious are explained along with the decision making process that led to them.

KEYWORDS: *Osteosynthesis, implants, infection, pulmonary edema.*

I. INTRODUCTION

Plate osteosynthesis is one possible treatment. A dynamic and biological fixation that permits micromotion at the fracture gap while maintaining bone perfusion is generally acknowledged as the means of achieving bone healing. Adolescent tibial and femoral fractures with incompletely closed growth plates, distal tibial and femoral fractures, and certain midshaft fractures are among the conditions that call for a dynamic plate osteosynthesis. There are significant benefits to open-reduction-and-plate fixation, even if intramedullary nails are an effective treatment for many lower limb shaft fractures. These benefits include a decreased risk of nonunion, anterior knee discomfort, and malalignment. Micromotion and fixation strength at the fracture gap might be affected by the surgeon executing the plate osteosynthesis.[1]

An infection that develops following surgical fracture repair using implants inserted within the body is referred to as an osteosynthesis-associated infection (OAI). Treatment aims to eliminate infection, promote fracture healing, maintain bodily function, and stop the condition from returning. One Treatment is very customized and presentation is diverse.[2]

The presence of union, the time elapsed between fixation and infection, and mechanical stability are the three most crucial considerations for formulating an OAI treatment plan. Other leading considerations include the type of implant, the existence of collections, non-viable bone, graft or substitute, defects in soft tissue and bone, joint involvement, host state, and treatment response. Referrals to subspecialized centers with greater equipment are ideal for patients in need of surgical treatment.[3] and acute myocardial infarction), valvular function (moderate to severe aortic/mitral regurgitation and stenosis), and rhythm (atrial fibrillation with rapid ventricular response, ventricular tachycardia, high degree, and third-degree heart block).

II. ETIOLOGY

Pulmonary edema can be roughly categorized into two types

CARDIOGENIC

Pulmonary edema caused by cardiogenic or volume overload occurs when the hydrostatic pressure of the pulmonary capillaries rises quickly. This is commonly observed in conditions affecting the left ventricle's systolic and diastolic function (acute myocarditis, including non-ischemic cardiomyopathy and acute myocardial infarction), valvular function (moderate to severe aortic/mitral regurgitation and stenosis), and rhythm (atrial fibrillation with rapid ventricular response, ventricular tachycardia, high degree, and third-degree heart block).

NON-CARDIOGENIC

Lung damage that increases pulmonary vascular permeability results in the migration of protein-rich fluid

into the alveolar and interstitial compartments, which causes noncardiogenic pulmonary edema.[4]

III. PATHOPHYSIOLOGY

In the pathophysiology of FRI, biofilm development on the surface of foreign material is essential. Since bacteria in biofilms are more resistant to medicines than planktonic ones, systemically administered antibiotics typically fail to produce the required therapeutic effects. This is the reason that both surgical and antibiotic treatments must be used in conjunction for the effective management of FRI. In the event of an open fracture, FRI typically develops exogenously as a result of the trauma itself, during the installation of the fixation device, during disrupted wound healing, or during late soft tissue coverage. Infections with hemoglobin are uncommon. Polymicrobial infections are common (20–35%) and mostly affect individuals who have had open fractures.[5]

III. MANAGEMENT

Two primary concepts of surgery can be used to attain goals

I. Antibiotic therapy in conjunction with irrigation, debridement, and implant retention.

II. Implant exchange or removal (in one or more phases) coupled with the use of antibiotics.[6]

Deep tissue cultures were taken during the initial, comprehensive surgical debridement of the afflicted soft tissue and necrotic bone that was administered to each patient in the study. Repeated surgical debridement was performed as necessary. When primary closure was not possible, continuous vacuum therapy was used to promote the healing of secondary wounds and provide temporary covering. In this study, minor, non-infected skin abnormalities that are left untreated are regarded as "open" in terms of the time it takes for them to close. Free flaps or a skin transplant were to be considered in cases of significant soft tissue damage. Implant removal and the initiation of an alternative form of stabilization at the fracture site were only taken into consideration when infection persisted despite numerous surgical debridements and there was no chance of infection control.[7]

IV. PREVENTION

Compared to elective joint replacement, infection rates following internal fixation range from 2% (closed fractures) to 30% (open fractures). The location and severity of the injury, the amount of concurrent injuries, and the host's physiology all influence the chance of developing a functional retinal injury (FRI). A number of known risk factors for infection have been found, including male gender, diabetes mellitus, smoking, polytrauma, fractures of the lower extremities, and injury severity. There are currently no universally recognized guidelines for the prevention of infection in fracture care. But this problem was recently brought up at a consensus conference, which also produced some proposals. Three main areas appear to be significant, particularly in high-risk patients (i.e., open fractures): soft tissue covering, systemic and local.[8]

V. CASE PRESENTATION

A 55 years old female patient with a complaints of no urine output, breathing difficulty & dry cough and patient had a past medical history of type II diabetes mellitus for twenty three years, hypertension for four years and chronic kidney disease on hemodialysis for two years on regular treatment with history of ORIF with plate osteosynthesis. On admission the patient vital shows that temperature as normal, Blood pressure as 140/90 mmHg, pulse rate 90beats /minutes, respiratory rate 15 breath/minutes and saturated oxygen level as 85 % there was no signs of dizziness, chronic cough and swelling legs. the saturation level and respiratory rate was decreased. The laboratory investigation like leukocyte count, neutrophil, eosinophil, lymphocytes are neither sufficiently sensoitive nor specific to predict infection. After surgery the c-reactive protein (CRP) is elevated and returns to normal within weeks. Therefore, in the postoperative period, repeated measurement is important. The secondary increase of CRP after a initial postoperative decline is highly suggestive for infection.

Laboratory investigation	Patient value	Reference value
White blood cell	3000million /ml	4,000-11000 million/ml
Neutrophils	19%	40-80%
Basinophil	0.2%	0.5-1%
Monocyte	2%	3-8%
Lymphotes	10%	20-40%
Eosinophils	1%	1-3%
RBC	4.4 million/ml	4.5-6.0 million /ml
Erythrocyte sedimentation Rate	90mm/hour	0-20mm/hour
c-reactive protein	0.2mg/dl	0.8-1.3mg/dl

Based on the laboratory investigation it was diagnosed confirmed that acute pulmonary edema in response to his infection. The treatment goal for this patient to minimize the fluid accumulation in the lungsand the patient was advised with Inj. Cefoperazone + sulbactum 1.5gm, BD which initially given as empirical therapy. Inj. Pantoprazole 40mg, BD was given due to polypharmacy it decrease the gastric acid secreation. Tab. clonidine 0.1 mg TDS. Tab. prasazosin hydrochloride 2.5mg BD is alpha blocker., Tab. Sodium bicarbonate 650mg, TDS. T.

Rifaximin TDS, T. Isosorbide dinitrate OD, T. Taurine and acetylcystine BD. Tab. calcium andvitamin D3 TDS, T. Monteleukast sodium HS, T.Bisoprolol fumarate 5mg, OD, T. Levocanitone 500mg, BD. Neb. Levosalbutamol P/N, TDS. Neb. Budisonide P/N, TDS, Tab. anxit 0.5mg HS. Inj. paracetamol 1gm.

VI. CASE DISCUSSION

Initially the patient came with the compliance of no urine output, Breathing difficulty and dry cough and patient had a past medical history type II diabetes mellitus for twenty three years and hypertension for four years and chronic kidney disease on hemodialysis for two years on regular treatment. recently the patient had a open reduction and internal fixation (ORIF) with plate osteosynthesis. Due to this condition he has continued discharge medication Inj. Cefoperazone+ sulbactum was administered to address bacterial superinfection which may occurs due to hospitalization. Inj. Pantoprazole was given due to decrease gastric acid secreation due to polypharmacy. Tab. Prasazosin hydrochloride is also given to decrease the blood pressure. Tab. Sodium bicarbonate thrice a day for acid indigestion. T. Rifaximin was a own drug given to teart bacterial infection TDS, T.Isosorbide dinitrate was also a own drug which was OD, T. Taurine and acetylcystine BD was given for breathing difficulty. Tab. calcium and vitamin D3 was nutrition supplement because the already he had a chronic kidney failure on hemodialysis, T. Monteleukast sodium HS was given to treat breathing difficulty, T. Bisoprolol fumarate was given to treat hypertension, OD, T. Levocanitone 500mg,BD prescribed to prevent and lack of carnitine. Neb.Levosalbutamol P/N, TDS. Neb.Budisonide P/N, TDS to reduce the symptoms of breathing difficulty, Tab. alprazolam 0.5mg HS was given to sleep.

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