MULTIPLE ORGAN FAILURE

An Essay Submitted in Partial Fulfilment of Master Degree in General Surgery

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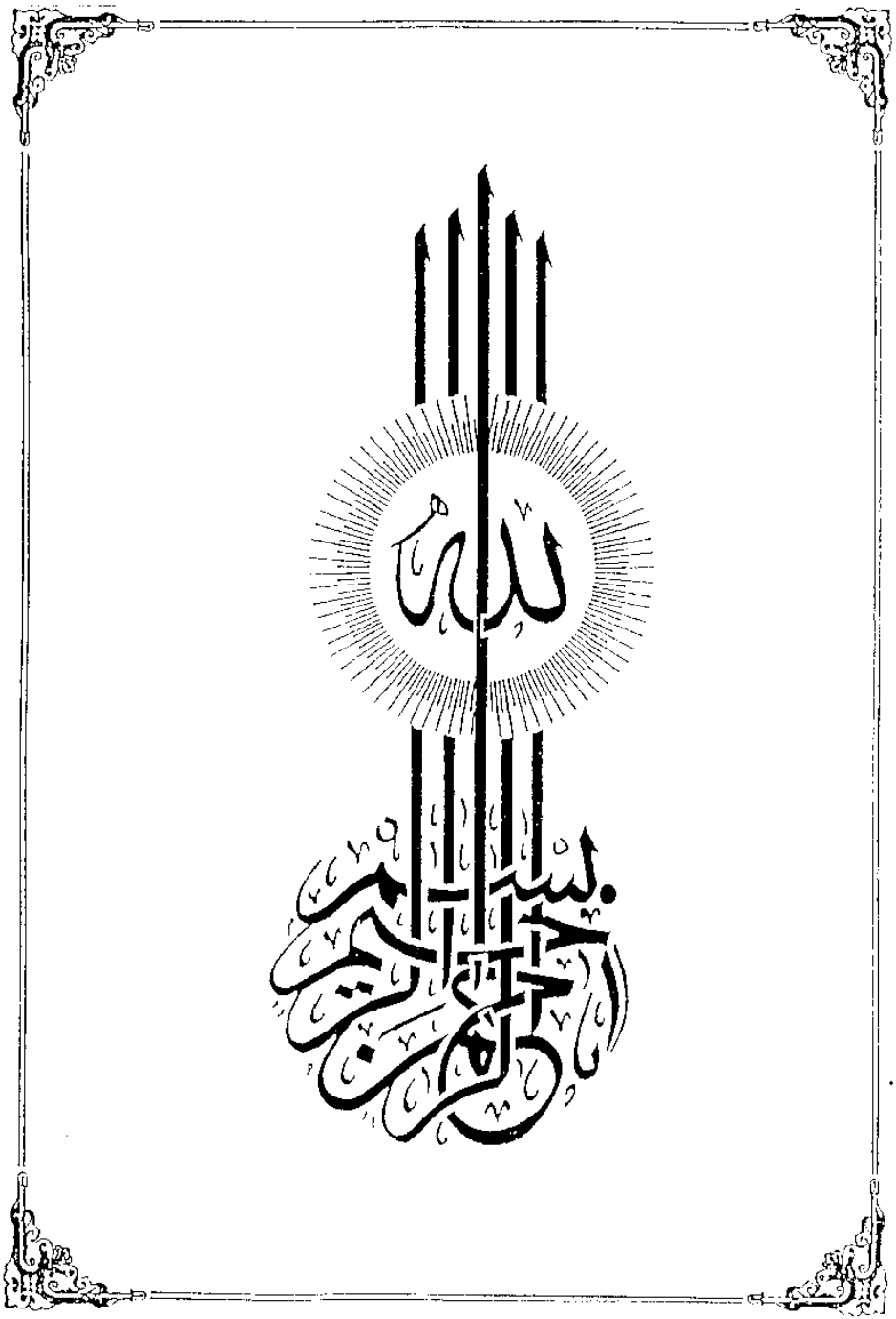

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TO MY FAMILY

INTRODUCTION

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The syndrome of multiple organ failure is the final common pathway for a number of clinical problems, including severe multiple system trauma, an operation with complications, infection such as peritonitis, inflammatory processes such as pancreatitis, and severe illness with limitations in organ function or cardiac output secondary to ageing, arteriosclerosis, or chronic disease. Multiple organ failure is thus a general intensive care unit syndrome and one of medical progress because it has appeared or develops only because of our technologic and clinical capapilities in supporting patients with organ dysfunction. ***(Arthur E. Baue 1991).***

Organ failure definitions were given by ***Faist E. et al., 1983 and Border J. R. et al., 1987*** as follows :

Pulmonary failure : there are three degrees of severity of pulmonary insufficiency, depending on the intensity of respiratory therapy required (P I- 1, 2 & 3).

PI - 1 is present when FIO_2 is 0.40 and there is no need for P.E.E.P 0-10. (FIO_2 : fraction of inspired oxygen) PI-2 is present when FIO_2 is > 0.40 and there is need for P.E.E.P. 0-10. PI-3 is present when FIO_2 is more than 0.5 and there is need for P. E. E. P. more than 10.

Pulmonary failure is defined as :-

PI-2 or PI-3 for a period of at least 72 hours, the need for 40 : 50% or more oxygen, or the need for P. E. E. P.

Cardiac failure :

High filling pressures with an inadequate circulation and arrhythmias.

Kidney failure :

Serum bilirubin level greater than 3.0 mgm% for 48 hours with elevation of glutamate dehydrogenase (GLDH) to greater than 10 mu/ml (twice normal value).

Coagulation system failure :

Thrombocytopenia with fewer than 60,000 platelets, with prothrombin index declining and the need for clotting factors to be given for longer than 24 hours.

G.I.T. failure :

Bleeding from the upper GIT which is endoscopically confirmed and required the replacement of two or more units of blood over 24 hours period.

There are two different patterns of development of multiple organ failure.

1) Single phase :

Rapid development of multiple organ failure (M.O.F.):

After trauma or shock the patient repidly develops M.O.F. and either recovers or dies in a brief period of time following injury.

2) Two phase pattern of M.O.F. development, with a lag phase this is a more typical and common pattern :

This usually begins with the gradual development of pulmonary insufficiency, often associated with manifist hypovolemic shock coagulation problems and renal insufficiency may have been evident.

There is then an interval period of stability. This is suddenly interrupted by the rapid development of sepsis mostly pneumania. This is then the starting point of censecutive organ failure with worsening lung function being followed by renal insufficiency reaching its peak with liver failure and coagulation problems.

CHAPTER I
AETIOLOGY OF MOF

CHAPTER "I"
AETIOLOGY

There are a number of aetologic factors the presence of which predispose to multiple organ failure *(Faist E. et al., 1983).*

Septic Shock

It is a commonly recognized clinical entity, defined as a systemic infection accompanied by hypotension that is not explained by hypovolemia or intrinsic cardiac disease. In most cases blood cultures are positive during these episodes.

The bacteria most frequently responsible are gram negative enteric bacilli. It has been pestulated that endotoxins (the lipo-polysaccharides coat of gram negative organsisms) are the cause. However, an indistinguishable clinical disease can be caused by gram positive bacteria, viruses & yeast.

The upper G.I.T. is an important reservoir of the organisms causing sepsis associated with the development of multiple organ failure. *(Marshall JC. & Meakins JL. 1993).*

Marshall JC, Christou NV & Meakins JL. have made a study to determine the association between proximal (GI) colonization

and the development of intensive care unit (I.C.U.) acquired infection and multiple organ failure (MOF) in a population of critically ill surgical patients.

Methods of Study :

A prospective cohort study of 41 surgical ICU patients was undertaken, specimens of gastric and upper small bowel fluid were taken for quantitative culture, the severity of organ failure was quantitated by a numeric score.

Results :

One or more episodes of ICU-acquired infection developed in 33 patients and involved at least one organism concomitently cultured from the upper GI tract in all but 3.

The most common organisms causing ICU - acquired infection : Candida, streptococcus faecalis, Pseudomonas and coagulase negative staphylococci, were also the most common species colonizing the proximal GI tract.

Gut colonization corrolated with the development of invasive infection with one week of culture for pseudomonas (90% VS 13% in non colonized patients, $P < 0.0001$) or staphylococcus epidermidis (80% VS 6%, $P < 0.0001$), a weaker association was seen for colonization with candida infections associated with GI colonization

included pneumonia (16 Patients), wound infection (12 patients), urinary tract infection (11 patients), recurrent (tertiary) peritonitis (11 patients) and bacteremia (10 patients) . ICU mortality was greater for patients colonized with pseudomonas 70% VS 26%, P = 0.03).

Organ dysfunction was most marked in patients colonized with one or more of the following : Candida, Pseudmonas , or S. epidermidis.

Most bacteria causing gm (-ve) sepsis are normal commensals in the GIT from there they may spread to contiguous structures as in peritonitis after gut perforation and they may migrate from the perineum into the urethera & bladder . Gm (- ve) bactremia follows infection in a primary focus, usually the genito urinary tract , biliary tree, gastrointestinal tract or lung, less commonly, the skin , bones and joints.

In burn patients and patients with leukemia the skin or the lungs are often portals of entery. In about 30% of cases notably in patients with debilitating diseases, cirrhosis and cancer, no primary focus is apparant. Gm (+ ve) bacteremias usually cross from the skin or respiratory tract.

Sepsis activates specific and non specific proteolysis in different humoral systems such as the complement, fibrinolysis and coagulation system as well as kellikerine cascade.

These provoke release of mediators known to stimulate cellular elements like platelets, granulocytes and macrophages.

Activated phagocytes are known to release numerous tissue damaging and toxic mediators. These are responsible for organ failure *(Schlag and Redl, 1987).*

Mediators of M.O.F. Caused by Septic Shock :
1. Kinins

They are polypeptide which increase capillary permeability, dilate small blood vessles and depress myocardial function *(Ledingham, 1979).*

Hagman factor (Factor VIII) initialy thaugh to relate solely to intrinsic coagulation system, has shown to have a central role in activation of kinins, initiation of the coagulation and fibrinolytic systems and activation of the complement, particularly during septic shock *(O' Donnell et al., 1976).*