Vasoplegic Syndrome

Essay

Submitted for Partial Fulfilment of
M.Sc. Degree in **ICU**

By

Assaad Mohammed Sheta

M. B. B. Ch. Cairo University

Supervised by

Prof. Madiha Metwally Zidan
Professor of Anaesthiology and ICU
Faculty of Medicine - Ain Shams University

Dr. Mohammed Sedky Mahmoud
Assistant Professor of Anaesthiology and ICU
Faculty of Medicine - Ain Shams University

Dr. Hany Ahmad Abd El Kader
Lecturer of Anaesthiology and ICU
Faculty of Medicine - Ain Shams University

Faculty of Medicine
Ain Shams University
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Assaad Mohammed Sheta

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List of Abbreviations

(ACE)…….……Angiotensin converting enzyme

(ACTH)……...Adreno corticotrophic hormones

(ALI)……...…….….Acute lung injury

(ANP)……..……..…….…..Atrial natriuretic peptide

(ARDS)……...……Adult respiratory distress syndrome

(ATP)……..……Adenosine triphosphate

(AVP)…….……..……..Vasopressin

(CCD)…….…….…… Charge-coupled device

(cGMP)…….....……Cyclic guanosine monophosphate

(CGRP)……..….Calcitonin gene-related peptide

(CO)…….……Carbon Monoxide

(CPB)…….…….….Cardiopulmonary bypass

CSE……....….…….(cystathionine γ-lyase) Enzyme

(DAG)…….…….…….….Diacylglycerol

(DIC)…….……..…Disseminated intravascular coagulation

(DIH)……..……...Dialysis-induced hypotension

(ECC)…….…….……Extracorporeal circulation

(ECG)…….…….Electrocardiography

(EEG)…….……...…..…Electroencephalography

(EF)…….……...……..…..Ejection fraction

(eNOS)……..Endothelial Nitric Oxide Synthase

(ET)……...…...……..……Endothelin

(GC)……...……..Guanylate cyclase

(HIT)…….……...Heparin-induced thrombocytopenia

(HMGB-1)……..……..…High mobility group box 1

(H2S)……..…….……Hydrogen sulfide

(ICU)……..…….…....Intensive Care Unite

(IF-γ)……..……...……Interferon gamma

(i NOS)…….……Inducible form of Nitric Oxide Synthase

(K^+-ATP)..Adenosine triphosphate sensitive potassium channels

(K-Ca channels)…..Potassium channels sensitive to cytosolic calcium

(IP_3)……..…….……...….Insitol trisphosphate

(LEDs)……...…….…Light-emitting diodes

(LPS)…….……..….Lipopolysaccharides

(LVAD)…….…...…Left Ventricle Assist Device

(MAP)…….……..Mean Arterial Pressure

(MB)…….…..……..Methylene blue

(MLC)……..……..……Myosin light chain

(MLCP)…….……Myosin light chain phosphatase

(MODS)……Multiple Organ Dysfunction Syndrome

(MOF)……..……..……..Multy Organ Failure

(mPTP)……...….Mitochondrial permeability transition pore

(NE)…….…….…..Norepinephrine

(nNOS)……..….Neuronal Nitric Oxide Synthase

(NO)……..…….……...Nitric oxide

(NOS)……Nitric oxide synthases

(NRP)…….……..‘No-return point’

(NRTI)……..…..Nucleoside reverse transcriptase inhibitor

(NSCC)……...…… Nonselective cation channels

(PAF)……..…….…Platelet-activating factor

(PaCO2)……..…...Partial pressure of carbon dioxide

(PKC)……...……..……Protein kinase C

(PLC)……..…….Phopholipase C

(PTP)……..……..…Permeability transition pore

(ROC)……..…….Receptor-operated Ca^{2+} channels

(ROS)……...……..…Reactive oxygen species

(SDF)…….…..…..Sidestream dark-field

(SIRS)…...……..…Systemic Inflammatory Response Syndrome

(SlCO2)…….……..……Sublingual capnometry

(SOC)…….……...…….Store-operated Ca^{2+} channels

(St O2)……..……..……...Oxygen saturation

(SVR)……...……Systemic Vascular Resistance

(TNF) ……...……..Tumor Necrosis Factor

(TPR)……..…….Total peripheral resistance

(TRALI)……Transfusion-associated acute lung injury

(VIP)……..……Vasoactive intestinal peptide

(VOC)……..…….Voltage-operated Ca^{2+} channels

(V/Q)…….……Ventilation/ perfusion

(VS) ……..……....Vasoplegic Syndrome

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Introduction

Recognition of hemodynamic shock can be more challenging and will require the clinician to increase his or her scrutiny of the patient and either rule in or rule out hemodynamic shock with further clinical investigations. For patients who are normotensive, the measurement of serum lactate will often reveal the presence of hemodynamic shock in a patient whose clinical evaluation is equivocal. This entity of a normotensive patient with some clinical signs of hemodynamic shock with an elevated lactate is often referred to as "cryptic shock." In this condition, the patient is not yet in critical condition but cannot meet his body's oxygen demand resulting in evidence of significant anaerobic metabolism. Previous trials showed that presence of hemodynamic shock and a serum lactate concentration of > 4.0 mmol/L are associated with a mortality of 30 to 45% ***(Rosenthal et al., 2008).***

Vasoplegic syndrome is a severe case of vasodilatory shock. It is characterized by severe and persistent hypotension, tachycardia, normal or increased cardiac output, decrease in systemic vascular resistance, low filling pressure and poor or no response to fluid resuscitation. It is associated with cardiopulmonary bypass, severe sepsis,

anaphylaxis, and hemodialysis. It is also described in patients with chronic liver disease ***(Zidan, 2011).***

Vasoplegic syndrome is attributed to combination of endothelial injury, arginine-vasopressin system dysfunction and release of vasodilatory inflammatory mediators including TNFα, interferon gamma and IL 1 which promote vasodilation through increase in cGMP. cGMP causes vasodilation and decrease myocyte contractility with myocardial and vascular smooth muscle relaxation **(*Zidan, 2011).***

The use of vasoconstrictors is not free from adverse systemic effects such as malperfusion in areas of compensatory vasoconstriction (e.g. splanchnic territory). Although vasopressor drugs are frequently used to provide adequate MAP, there is no certainty about whether they are sufficient for obtaining the target MAP. It also remains debatable which of these blood pressure-increasing drugs is preferable ***(Gulmen et al., 2011).***

Aim of the Essay

The aim of this Essay is to review the prevalence of vasoplegic syndrome and show the pathophysiology and consequences of the syndrome and its management.

Recognition of Hemodynamic Shock

Shock is not just hypotension, but shock represents hypoperfusion of end organs. Normotensive patients can suffer from shock. The clinical manifestations of hemodynamic shock are related directly to the end organs that are not adequately perfused. Beside hypotension, the classic signs and symptoms of hemodynamic shock are tachycardia, tachypnea, cool and clammy extremities, oliguria, and delirium ***(Rosenthal et al., 2008).***

Current technology, which allows assessment of perfusion independent of arterial pressure, has shown that hypotension does not define shock. The most appropriate definition is the state in which profound and widespread reduction of effective tissue perfusion leads first to reversible, and then, if prolonged, to irreversible cellular injury ***(Kumar & Parrillo, 2008).***

In the absence of hypotension, the diagnosis of hemodynamic shock can be more challenging and will require the clinician to increase his or her scrutiny of the patient and either rule in or rule out hemodynamic shock with further clinical investigations. For patients who are

normotensive, the measurement of serum lactate will often reveal the presence of Hemodynamic Shock in a patient whose clinical evaluation is equivocal. This entity of a normotensive patient with some clinical signs of hemodynamic shock with an elevated lactate is often referred to as "cryptic shock." In this condition, the patient is not yet in critical condition but cannot meet his body's oxygen demand, resulting in evidence of significant anaerobic metabolism. Previous trials showed that presence of hemodynamic shock and a serum lactate concentration of > 4.0 mmol/L are associated with a mortality of 30 to 45% ***(Rosenthal et al., 2008).***

Indicators of hypoperfusion:

I-Vital signs (May be normal initially):

A) Heart rate:

Heart rate increases to maintain adequate cardiac output and oxygen delivery to injured tissues due to hypoperfusion. Reflex tachycardia can be caused also by hypotension, anxiety, hypoxia, hypercapnia, drugs, pain, temperature and direct irritating effect on the heart ***(Bonanno, 2011).***