Neuroaxial Anesthesia in Patients on Anticoagulants

Essay

Submitted for the partial fulfillment of Master Degree
in Anesthesiology

By

Ahmed Adly Mohamed Aly
M.B.B.CH

Under Supervision of

Prof. Dr. Raafat Abdel Azim Hammad
Professor and Head of the Department of Anesthesiology,
Intensive Care and Pain Management
Faculty of Medicine – Ain Shams University

Dr. Dalia Abdel Hameed Nasr Eldin
Assistant Professor of Anesthesiology, Intensive Care and Pain Management
Faculty of Medicine – Ain Shams University

Dr. Dina Salah Eldin Mahmoud
Lecturer of Anesthesiology, Intensive Care and Pain Management
Faculty of Medicine – Ain Shams University

Faculty of medicine
Ain shams university
2013

بِسْمِ اللّهِ الرّحمَنِ الرّحيمِ

[...رَبِّ أَوزِعنِي أَن أَشكُرَ نِعمَتَكَ
الَّتِي أَنعَمْتَ عَلَيَّ و عَلى والِدَيَّ
و أَنْ أَعْمَلَ صَالِحاً تَرْضَاهُ و أَدْخِلْنِي
بِرَحْمَتِكَ فِي عِبَادِكَ الصَّالِحِينَ]

صدق الله العظيم

النمل.. اية رقم ١٩

Acknowledgment

First and foremost, I feel always indebted to ***Allah****, the Most Merciful, Who gives me power to accomplish this work.*

I would like to express my deepest appreciation and sincere gratitude to ***Prof. Dr. Raafat Abdel Azim Hammad,*** *Professor and Head of the Department of Anesthesiology, Intensive Care and Pain Management, Faculty of Medicine – Ain Shams University, for his sincere help, constant encouragement, constructive criticism, and valuable guidance, I was truly honoured to work under his supervision.*

I wish also to express my great gratitude and utmost appreciation to ***Dr. Dalia Abdel Hameed Nasr Eldin,*** *Assistant Professor of Anesthesiology, Intensive Care and Pain Management, Faculty of Medicine – Ain Shams University, for her valuable suggestions and instructions during the progress of this work.*

I feel deeply indebted to ***Dr. Dina Salah Eldin Mahmoud,*** *Lecturer of Anesthesiology, Intensive Care and Pain Management, Faculty of Medicine – Ain Shams University, for her active cooperation, deep concern, enthusiastic encouragement, the effort and time she has devoted to the fulfillment of this work.*

I owe special thanks to ***my Family*** *for their care, patience and continuous encouragement.*

Ahmed Adly Mohamed Aly

List of Contents

Subject *Page No.*

List of Abbreviation

Abbrev.	*Meaning*
ADP	: Adenine nucleotides peptide
AHF	: Anti-hemophilic factor
AHG	: Anti-hemophilic globulin
aPTT	: Activated partial thromboplastin time
AT	: Anti-thrombin
ATP	: Adenosine triphosphate peptide
cAMP	: Cyclic Adinosine mono-phosphate
CO	: Cyclo-oxygenase
CT	: Clotting time
CYP	: Cytochrome P
d	: Day
DVT	: Deep venous thrombosis
EMC	: Extracellular membrane
HIT	: Heparin induced thrombocytopenia
HMW	: High molecular-weight
hrs	: Hours
IgG	: Imuno-glopin G
INR	: International normalized ratio
ISI	: International sensitivity index
ITC	: Intrinsic tenase complex

List of Abbreviation *(Cont…)*

Abbrev.	*Meaning*
Kg	: Kilogram
LMWH	: Low molecular weight heparin
mg	: Milligram
min	: Minute
mo	: Month
NO	: Nitric oxide
NSAIDs	: Non-steriodal anti-inflamatory drugs
PAI	: Plasminogen activator inhibitor
PAR	: Protease activated receptor
PE	: Pulmonary embolism
PF	: Platelet factor
PT	: Prothrombin time
PTA	: Plasma thromboplastin antecedent
PTC	: Plasma thromboplastin component
rt-PA	: Recominant tissue-type plasminogen activator
SPCA	: Serum prothrompin conversion acceleration
TAFI	: Thrombin achievable fibrinolysis inhibitor
TF	: Tissue factor
TFPI	: Tissue factor pathway inhibitor
U	: Unit

List of Abbreviation *(Cont…)*

Abbrev.	*Meaning*
UFH	: Unfractioned heparin
VKER	: Vitamine k epoxide reductase
VTE	: Venous thrombo-embolism
vWF	: Von Willebrand factor
wK	: Week
%	: Percentage

List of Tables

List of Figures

Introduction

The choice of regional anesthesia may offer considerable advantages over general anesthesia for various surgical procedures or for certain patients or in certain settings. It provides several advantages over systemic opioids, including superior analgesia, reduced blood loss and need for transfusion, and decreased incidence of graft occlusion ***(Llau et al., 2007).***

New challenges in the management of patients undergoing neuraxial block have arisen, as thromboprophylaxis which has an established role in the perioperative period, while increasingly more patients presenting for surgery are already receiving anticoagulants. Performing a neuraxial blockade in these patients, with appropriate adjustments of their anticoagulant treatment, has generally been safe. However, due to the severity of a hematoma within the rigid space of the central nervous system, there is an urgent need for a systematic approach in parallel with the increasingly "mandatory" use of anticoagulants. Concern for patient safety in the presence of potent antithrombotic drugs has resulted in avoidance of regional anesthesia ***(Douketis et al., 2002).***

When patients in this category require surgery, anesthetic management is challenging, as the risk of a thromboembolic event during interruption of these drugs needs to be balanced

against the risk of bleeding. the anesthetist should have a clear understanding of the pharmacokinetics of these drugs, not only when timing the insertion of neuraxial blocks but also in the removal of indwelling catheters ***(Barret et al., 2010).***

Aim of the Work

The aim of this work is to review the issue of performing a neuraxial block in patients receiving anticoagulants. Management of both the anticoagulant therapy and the neuraxial block will be discussed.

Chapter (1)
Physiology of Hemostasis

Hemostasis:

Hemostasis is the process of forming a clot in the walls of damaged blood vessels and preventing blood loss while maintaining blood in a fluid state within a vascular system ***(kim et al., 2010).***

A collection of complex interrelated systemic mechanisms operate to maintain a balance between coagulation and anticoagulation ***(kim et al., 2010).***

The classic theory of hemostasis:

Can be initiated by either two distinct pathways.

The intrinsic pathway** can be initiated by events that take place within the lumen of the blood vessels. The intrinsic pathway requires elements (clotting factors, Ca^{++}, platelet surface, etc.) ***(Dahlback and Villoutreix 2005).

The extrinsic pathway** is the other route to coagulation. It requires Tissue factor (tissue thromboplastin), the intrinsic and extrinsic pathways converting to a common pathway at a point where factor X is activated ***(Bates and Weitz 2005).

The modern theory of hemostasis:

Hemostasis term means prevention of blood loss by activating several mechanisms:

1. Vascular constriction.
2. Formation of platelet plug.
3. Formation of blood clot as a result of blood coagulation.
4. Eventual growth of fibrous tissue into blood clot to close the vessel permanently ***(kim et al., 2010).***

1-Vascular constriction

Immediately after blood vessel has been cut or ruptured, the trauma to the vessel wall itself causes the smooth muscle in the wall to constrict; reduces the flow of blood from the ruptured vessel ***(Arthur et al., 2006).***

For the smaller vessels, the platelets are responsible for much of the vasoconstriction by releasing a vasoconstrictor substance, (e.g. thromboxane A_2.The more severely a vessel is traumatized, the greater the degree of vascular spasm ***(Guyton and Hall 2006).***

The spasm can last for many minutes or even hours, during which time the processes of platelet plugging and blood coagulation can take place ***(kim et al., 2010).***

2-Formation of platelet plug

If the cut in the blood vessel is very small, small vascular holes develop through each day-the cut is often sealed by platelet plug, rather than by blood clot ***(kim et al., 2010).***

Mechanism of platelet plug

The platelet membrane is characterized by numerous receptors and a surface-connected open canalicular system serving to increase platelet membrane surface area as well as to provide rapid communication between the platelet interior and external environment ***(Miller et al., 2006).***

Under normal circumstances, platelets do not bind vascular endothelium. However, when injury exposes extra cellular membrane (ECM), platelets undergo a series of biochemical and physical alterations characterized by three major phases:

A) Adhesion

Exposure of subendothelial matrix proteins (i.e., collagen, vWF, fibronectin) allows for platelet adhesion to the vascular wall.

Absence of either vWF (von Willebrand disease) or glycoprotein Ib/factor IX/factor V receptors (Bernard-Soulier syndrome) results in a clinically significant bleeding disorder ***(Chen and Lopez 2005).***