



جامعة عين شمس  
كلية الطب  
قسم التخدير و العناية المركزة

## التحديثات في الرعاية الحرجة لنزيف المخ

رسالة مقدمة توطئة للحصول على درجة الماجستير في الرعاية المركزة

مقدمة من الطبيب  
**عمرو حنفي محمد حنفي**  
بكالوريوس الطب والجراحة (٢٠١١)  
جامعة القاهرة

تحت إشراف  
**أ.د. باسل محمد عصام نور الدين**  
أستاذ التخدير والرعاية المركزة  
كلية الطب – جامعة عين شمس

**د. وليد عبدالله إبراهيم**  
مدرس التخدير والرعاية المركزة  
كلية الطب – جامعة عين شمس

**د. جون نادر نصيف**  
مدرس التخدير والرعاية المركزة  
كلية الطب – جامعة عين شمس

كلية الطب  
جامعة عين شمس  
٢٠١٨

## المحتويات:

- المقدمة

- الهدف من العمل

(١) الباثوفسيولوجية للضرر الناتج عن نزيف المخ.

(٢) تشخيص نزيف المخ و التصوير الدماغى.

(٣) المعالجة الطارئة و طويلة المدى.

- الملخص

- المراجع

- الملخص العربي

## الملخص العربي

تتمثل أهمية الإصابة بنزيف المخ في ارتفاع معدل الوفيات حيث أن أعراض الإصابة تتنوع من عجز عصبي طفيف إلى مضاعفات شديدة وعدم الاستقرار القلبي وارتفاع الضغط داخل الجمجمة مما قد يؤدي إل الوفاة و يستند التشخيص إلى الأعراض السريرية والفحوصات المعملية والتصوير الذي يشمل الأشعة المقطعية والرنين المغناطيسي وتصوير الأوعية.

تقديرات المعدل السنوي للإصابة نحو ١٦-٣٣ حالة لكل ١٠٠,٠٠٠. وهناك العديد من الحالات المرضية الكامنة المرتبطة بنزيف المخ مثل ارتفاع ضغط الدم و تمدد الأوعية الدموية. و تشمل عوامل الخطورة التي قد تؤدي للإصابة مرض السكرى، السكتات الدماغية السابقة، التدخين، الكحول، بعض الأدوية و خاصة الوارفارين والأسبرين، أمراض الدم و الكبد و الأورام.

نزيف المخ هو حالة طبية طارئة. التشخيص السريع و الإدارة المنتبهة لمرضى نزيف المخ أمر بالغ الأهمية لأن تدهور الحالة في وقت مبكر شائع في الساعات القليلة الأولى. و أكثر من ٢٠ ٪ من المرضى يعانون من انخفاض في درجة الوعي < ٢ نقطة في الفترة بين تقييم الخدمات الطبية الطارئة قبل دخول المستشفى و التقييم الأولي في قسم الطوارئ.

الأشعة المقطعية بدون استخدام الصبغة هي الأداة الأكثر توفراً و بالتالي يشيع استخدامها في أقسام الطوارئ. و يعتقد أنها شديدة الدقة في الكشف عن نزيف المخ بنسبة ١٠٠ % و يستخدم الرنين المغناطيسي في المتابعة لتحديد الأسباب الثانوية لنزيف المخ مثل التشوه الشرياني و أورام المخ.

يجب أن تتركز الإدارة الأولية لنزيف المخ على تقييم قدرة التنفس ، المجرى الهوائى للمريض، وعلامات زيادة الضغط داخل الجمجمة و ضغط الدم. يجب العمل على استقرار العلامات الحيوية للمريض على أسرع وجه، وفقاً للمبادئ التوجيهية (ATLS). المرضى الذين يعانون من نزيف المخ غالباً في حاجة لحماية مجرى الهواء عن طريق تركيب امبوبة حنجرية بالقصبية الهوائية.

و يعد الحفاظ على وظيفة القلب الحيوية أحد أهم أدوار الرعاية العامة لنزيف المخ، و يجب العمل على منع حدوث مضاعفات نزيف المخ بما في ذلك الالتهاب الرئوى، سوء التغذية، جلطات أوردة الساق، الانسداد الرئوي و تقرحات الفراش.



# **Updates in Critical Care Management of Intracerebral Hemorrhage**

Essay

Submitted for partial fulfillment of master degree in  
Intensive Care

Presented by

**Amr Hanafy Mohamed Hanafy**  
(M.B., B.ch – 2011 Cairo University)

Supervised by

**Prof. Dr/ Bassel Mohamed Essam Nour Aldin**

Professor of Anesthesia and Intensive Care  
Faculty of Medicine - Ain Shams University

**Dr/ Waleed Abdalla Ibrahim**

Lecturer of Anesthesia and Intensive Care  
Faculty of Medicine - Ain Shams University

**Dr/ John Nader Nassif**

Lecturer of Anesthesia and Intensive Care  
Faculty of Medicine - Ain Shams University

Faculty of medicine  
Ain Shams University  
2018

## **ACKNOWLEDGMENT**

*All thanks to ALLAH making me capable of finishing this work.*

*I would like to express my deepest thanks and appreciation for **Prof. Dr. Bassel Mohamed Essam Nour Aldin**, professor of Anesthesia and Intensive Care, faculty of Medicine, Ain Shams University for his valuable support and encouragement.*

*I would like also to express my deepest thanks and appreciation for **Dr. Waleed Abdalla Ibrahim**, Lecturer of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University for his kind support and supervision.*

*I would like also to express my deepest thanks to **Dr. John Nader Nassif**, Lecturer of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University.*

*Last but not least, I would like to express my deepest thanks, gratitude and love for my family.*

## **CONTENT**

- Introduction
- Aim of the study
- Chapter (1): Pathogenesis of intra cerebral hemorrhage.
- Chapter (2): Diagnosis of intra cerebral hemorrhage.
- Chapter (3): Acute and long term management.
- Summary
- References
- Arabic Summary

## **LIST OF FIGURES**

Figure number	Page
Diagram 1; showing causes of non traumatic intra cerebral hemorrhage.	6
Diagram 2; showing sites of intra cranial hemorrhage.	7
Figure 3; CT brain showing left intra cerebral hematoma with mass effect and shift of midline structures.	15
Diagram 4; Showing most common sites of cerebral aneurisms.	20
Figure 5; Axial MR images show a hyperacute hematoma in the right external capsule and insular cortex in a known hypertensive patient.	23
Figure 6; CT images with spot sign, demonstrating extravasation and hematoma expansion.	24
Algorithm 7; Showing how to manage intra cerebral hemorrhage once suspected.	31



## **LIST OF TABLES**

Table number	Page
Table 1; Risk factors causing intra cerebral hemorrhage.	8
Table 2; Univariate analysis of in-hospital complications on 3-month mortality in young patients with intra cerebral hemorrhage.	17
Table 3; Showing ideal management of different types of intra cranial hemorrhage.	42
Table 4; Applying classification of recommendations and levels of evidence for intra cerebral hemorrhage management.	44
Table 5; Showing Class 1 recommendations for intra cerebral hemorrhage management.	48

## **LIST OF ABBREVIATIONS**

**AVM:** Arterio-venous malformation.

**BP:** Blood pressure.

**BUN:** Blood urea nitrogen.

**CBC:** Complete blood count.

**CT:** Computed tomography.

**CVP:** Central venous pressure.

**DVT:** Deep venous thrombosis.

**ECG:** Electro cardio gram.

**EMS:** Emergency medical services.

**FFP:** Fresh frozen plasma.

**ICH:** Intra cerebral hemorrhage.

**ICP:** Intracranial pressure.

**INR:** International normalized ratio.

**IVC:** Inferior vena cava

**IVH :** Intra ventricular hemorrhage.

**MRI:** Magnetic resonance imaging.

**OAC:** Oral anti-coagulant.

**PE:** Pulmonary embolism.

**VKA:** Vitamin k antagonist.

**SAH:** Subarachnoid hemorrhage.

## **INTRODUCTION**

Intra cerebral hemorrhage (ICH) is associated with substantial morbidity and mortality. The varied clinical presentation of ICH, ranging from minor neurological deficits to fatal herniation syndromes arises from parenchymal damage, elevated intracranial pressure and cardiopulmonary instability. Diagnosis is based on clinical presentation, laboratory investigations and imaging, which include computed tomography (CT), magnetic resonance imaging (MRI) and angiography. (*Magistris et al 2013*)

Estimates of the annual incidence range from 16 to 33 cases per 100,000. There are many underlying pathological conditions associated with intra cerebral hemorrhage; hypertension, amyloid angiopathy, ruptured saccular aneurysm and vascular malformation account for the majority of cases. Other important risk factors for acute intra cerebral hemorrhage include any recent trauma, prior strokes, diabetes, smoking, alcohol, certain drugs especially warfarin, aspirin and other anticoagulants, hematologic disorders, liver diseases, neoplasms and infections. (*Kase 2012*)

Hypertensive vasculopathy is the most common etiology of spontaneous intra cerebral hemorrhage. Cerebral amyloid angiopathy is the most common cause of non-traumatic lobar ICH in the elderly, while vascular malformations are the most common cause of intra cerebral hemorrhage in children. (*Beslow et al 2010*)

Intra cerebral hemorrhage is a medical emergency. Rapid diagnosis and attentive management of patients with ICH is crucial because early deterioration is common in the first few hours after ICH onset. More than 20% of patients will experience a decrease in the Glasgow Coma Scale (GCS) score of  $\geq 2$  points between the pre-hospital emergency medical services assessment and the initial evaluation in the emergency department. (*Morgenstern et al 2010*)

The non-contrast CT is the most readily available tool providing rapid feedback and is thus commonly used in emergency departments. It is thought to be nearly 100% sensitive for detecting clinically relevant acute hemorrhages. Moreover, it may elucidate hematoma location and expansion and the presence of edema. MRIs are most frequently utilized as follow-up investigations to identify secondary causes of ICH, such as arterio-venous malformation (AVM), amyloid angiopathy, or associated neoplasm. (*Nentwich and Veloz 2012*)

Early neurologic deterioration within 48 hours after intra cerebral hemorrhage onset is not infrequent and is associated with a poor prognosis. Potential mechanisms include hemorrhage enlargement, development of hydrocephalus, and perilesional edema. The inflammatory response to the hemorrhage may also play a role. (*Rodriguez-Luna et al 2011*)

Intra cerebral hemorrhage is a neurological emergency and initial management should be focused on assessing breathing capability, patients airway, signs of increased intracranial tension and blood pressure. The patient's vital signs must be immediately stabilized according to ATLS guidelines. Patients with ICH are often unable to protect their airway and may need endotracheal intubation. (*Stone and Humphries 2011*)

Advances in acute care including investigative, diagnostic, and management strategies are important to improving outcomes for patients with ICH. Physicians caring for patients with ICH should anticipate the need for emergent blood pressure reduction, coagulopathy reversal, cerebral edema management, and surgical interventions. Good general care should be directed at maintaining vital cardiopulmonary function. Measures to prevent sub-acute complications of intra cerebral hemorrhage including aspiration, malnutrition, pneumonia, deep vein thrombosis, pulmonary embolism, pressure sores, orthopedic complications, and contractures are recommended. (*Koto et al 2014*)

The prognosis after ICH depends upon the location of hemorrhage (supra versus infra-tentorial location), size of the hematoma, level of consciousness, patient age, and overall medical health and condition. (*Gonzalez-Perez et al 2013*)

## **AIM OF WORK**

The aim of this essay is to discuss recent knowledge and evidence based studies regarding critical care management of intra cerebral hemorrhage.

## **PATHOGENESIS**

Intra cerebral hemorrhage accounts for 8-13% of all strokes and results from a wide spectrum of disorders. Intra cerebral hemorrhage is more likely to result in death or major disability than ischemic stroke or sub arachinoid hemorrhage. Intra cerebral hemorrhage and accompanying edema may disrupt or compress adjacent brain tissue, leading to neurological dysfunction. Substantial displacement of brain parenchyma may cause elevation of intracranial pressure (ICP) and potentially fatal herniation syndromes (*Magistris et al 2013*).

Non traumatic intra cerebral hemorrhage most commonly results from hypertensive damage to blood vessel walls (eg, hypertension, eclampsia, drug abuse), but it also may be due to autoregulatory dysfunction with excessive cerebral blood flow (eg, reperfusion injury, hemorrhagic transformation, cold exposure), rupture of an aneurysm or arterio venous malformation (AVM), arteriopathy (eg, cerebral amyloid angiopathy), altered hemostasis (eg, thrombolysis, anticoagulation, bleeding diathesis), hemorrhagic necrosis (eg, tumor, infection), or venous outflow obstruction (eg, cerebral venous thrombosis) (*Woo et al 2004*).

Non penetrating and penetrating cranial trauma are also common causes of intra cerebral hemorrhage. Patients who experience blunt head trauma and subsequently receive Warfarin or Clopidogrel are considered at increased risk for traumatic intracranial hemorrhage. According to one study, patients receiving Clopidogrel have a significantly higher prevalence of immediate traumatic intracranial hemorrhage compared with patients receiving warfarin. Delayed traumatic intracranial hemorrhage is rare and occurred only in patients receiving warfarin (*Nishijima et al 2013*).