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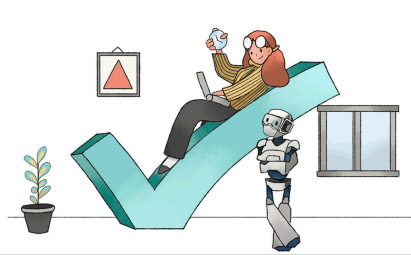
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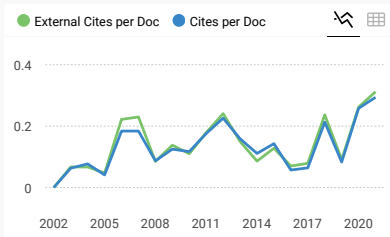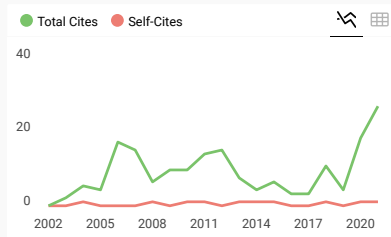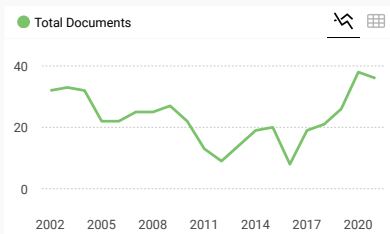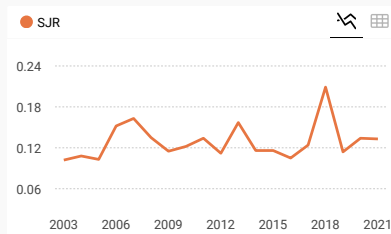| PUBLICATION TYPE	ISSN	COVERAGE	INFORMATION
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SCOPE

Critical Care and Shock has its origin in the regular discussions of a small circle of intensivists from the US, Europe, Japan, and Indonesia who pioneered the international conference of critical care medicine, better known as the Indonesian-International Symposium on Shock and Critical Care, which is held annually in Indonesia since 1994. It was thought at that time that it would be worthwhile to publish a journal in critical care medicine as part of the effort to support and promote the annual conference and to share the latest advances in critical care with the potential readers in Western Pacific region that might complement favorably to the conference. The first issue of Critical Care and Shock appeared in June 1998 featuring the articles mostly from the guest speakers of the annual Indonesian-International Symposium on Shock and Critical Care. From its beginning Critical Care and Shock has been the official journal of the Indonesian Society of Critical Care Medicine. By 1999, at the Council meeting of Western Pacific Association of Critical Care Medicine (WPACCM), it was approved to adopt Critical Care and Shock as the official journal of WPACCM. Also, as of the February issue of 2001, Critical Care and Shock has become the official journal the Philippines Society of Critical Care Medicine. At present, Critical Care and Shock is enjoying increasing readership in the countries of the Western Pacific region, and welcome the submission of manuscripts from intensivists and other professionals in critical care around the globe to be published in its future issues. Critical Care and Shock is published bimonthly in Februarys, Aprils, Junes, Augusts, Octobers, and Decembers.

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Month: June 2019

Anesthesia and intensive care management in acute ischemic stroke patient

Abstract

Acute ischemic stroke is a brain functional disorder, which cause high disability and mortality rate worldwide, the second most common cause of dementia, and the third leading cause of death. It has enormous clinical, social, and economic implications and demands a significant effort from both basic scientists and clinicians in the quest for understanding the underlying pathomechanisms and producing suitable preventive measures and successful therapies. Management of acute ischemic stroke has been revolutionized by the introduction of several interventions, such as prehospital and stroke unit care, intravenous tissue plasminogen activator therapy within 4.5 hours of stroke onset, aspirin therapy within 48 hours of stroke onset, decompressive craniectomy for supratentorial malignant hemispheric cerebral infarct, and more recently endovascular therapy for anterior circulating stroke. Also, special attention in management of vital systemic physiological variables, including oxygenation, blood pressure, temperature, and serum glucose. In line with this, the role of neuroanesthesiologists and neuro critical care in managing acute ischemic stroke become more prominent.

Authors: Hamzah, Prananda Surya Airlangga, Abdulloh Machin, Nancy Margarita Rehatta

Do Asian physicians manage hypertensive crisis properly? A Southeast Asia survey analysis

Abstract

Purpose: **Hypertension remains a common cause of morbidity and mortality worldwide, and proper management can prevent death. Over the last few decades, several changes in definitions, clinical manifestations, and management have occurred. We aimed to investigate the extent of knowledge about these changes, as the pertain to acute elevations of blood pressure among physicians in Southeast Asia.**

Methods: **A cross-sectional survey was created and validated. The survey included 25 questions about the definitions of hypertensive urgencies and emergencies, clinical presentations, ideal rate of blood pressure reduction, and other questions. Surveys were distributed at the Asia Pacific Symposium held in Indonesia in August 2017. Descriptive analysis was conducted using IBM SPSS Statistics™ version 25.0 (IBM Corporation, Armonk, NY).**

Results: **A total of 145 surveys were completed by physicians from India, Indonesia, South Korea, Philippines, Singapore, and Sri Lanka. Of them, only 49.6% (n=72) knew the normal blood pressure cut-off limits, and 19.3% (n=28) did not recognize the differences between hypertensive urgencies and emergencies. Moreover, 53.7% (n=78) of respondents was not aware that hypertensive urgency can be completely asymptomatic. Sixty-four point eight percent (n=94) indicated that acute**

cerebrovascular accidents were the most common end-organ damage from hypertensive emergencies. In addition, only 44.1% (n=64) were aware of the ideal rate of blood pressure reduction, and 50.3% (n=73) considered sublingual nifedipine as an appropriate choice in hypertensive crisis management.

Conclusions: A significant percentage of physicians in Southeast Asia lack knowledge about hypertensive crisis definitions, clinical presentations, and management.

Authors: Abbas Alshami, Johanan Luna, America Avila, Salim Surani, Joseph Varon

Corticosteroids and gastrointestinal bleeding in critical care: a systematic review and meta-analysis

Abstract

Background: Current belief suggests that patients receiving corticosteroids have an increased risk of bleeding from stress ulceration and that these patients should receive stress ulcer prophylaxis. This issue is important as many ICU patients receive corticosteroids and the pharmacologic agents used for stress ulcer prophylaxis are associated with adverse events.

Aim: The goal of this systematic review and meta-analysis was to compare the rate of clinically significant (overt) gastrointestinal (GI) bleeding in critically ill patients receiving corticosteroids versus placebo.

Methods: We searched PubMed, Embase, and the Cochrane database from inception through December 2018. In addition, the bibliographies of selected articles were reviewed for relevant studies and included if inclusion criteria were met. Included studies were randomized, placebo-controlled and blinded studies that compared treatment with corticosteroids for any indication in a patient population that included only ICU patients. Primary outcome of interest was rate of clinically significant GI bleeding in patients treated with corticosteroids versus placebo. Results were expressed as risk ratio (RR) with accompanying 95% confidence interval (CI). Heterogeneity, sensitivity analysis, and risk of bias were explored. In addition, we did a subgroup analysis according to the use of "low-dose" (<400 mg hydrocortisone or equivalent/day) versus "high-dose" corticosteroid.

Results: Thirty-five studies, which enrolled 16,659 patients, met inclusion criteria and were analyzed. Significant GI bleeding was recorded for 355 patients (overall rate of 2.1%). Summary data demonstrated no difference in the risk of GI bleeding between those treated with corticosteroids versus placebo (RR 1.08; 95% CI 0.88-1.33; p=0.46) with minimal heterogeneity between studies (Q statistic p=0.86, I^2=0%). Similarly, there was no significant difference in the risk of GI bleeding in either the low (RR 1.04; 95% CI 0.78-1.38) or the high dose groups (RR 1.13; 95% CI 0.84-1.53) and in those studies at low risk of bias (RR 1.16; 95% CI 0.91-1.49) and those at a high risk of bias (RR 0.88; 95% CI 0.6-1.28).

Conclusion: This meta-analysis did not identify a clinically significant difference in the rate of overt GI bleeding in critically ill patients receiving corticosteroids as compared to placebo. The role of stress ulcer prophylaxis in these patients remains uncertain.

Authors: Paul E. Marik, Mit P. Patel, Joseph Varon

The effect of N-acetylcysteine on the myeloperxidase and Tei index in patients with acute myocardial infarction

Abstract

Bacground: Myeloperoxidase (MPO) is a strong oxidant and toxic to microorganisms with excess production causing tissue damage. We aimed to determine the effect of N-acetylcysteine (NAC) 600 mg orally 3 times a day for 3 consecutive days on MPO levels

and left ventricle myocardial performance index (LVMPI/Tei index) in ST elevation myocardial infarction (STEMI) patients treated with fibrinolytics.

Methods: Pre- and post-design, single blind experimental randomized trial, conducted on 32 patients with STEMI at Intensive Cardiovascular Care Unit (ICVCU). The subjects were divided into 2 groups: 17 patients received 600 mg t.i.d NAC for 72 hours and 15 controls. MPO levels before and after 72 hours and Tei index 72 hours after NAC therapy were measured. Statistical analysis of MPO level and Tei index were analyzed with SPSS 22. Tei index was measured using the pulsed wave Doppler (PWD) and tissue Doppler imaging (TDI).*Results*: NAC administration showed decrease in the marker of MPO (112.76±57.28 vs 180.40±69.03, p=0.001) and delta MPO (-50.15±46.62 vs 12.06±108.65) 72 hours after NAC therapy compared with control. NAC improved the LVMPI value compared to the control group. Tei index examination using PWD (0.39±0.11 vs 0.49±0.08, p=0.005) and that using TDI (0.41±0.08 vs 0.57±0.08, p=0.001) showed improved values for NAC administration than those with controls.*Conclusion*: NAC 600 mg orally 3 times a day for 3 consecutive days can reduce MPO levels and improve diastolic function by decreasing LVMPI values.

Authors: Trisulo Wasyanto, Ahmad Yasa, Nuka Meriedlona

The effect of oral N-acetylcysteine on galectin-3 and global longitudinal strain in patients with acute myocardial infarction

Abstract

Objective: Galectin-3 (Gal-3) plays a big role in the development of cardiac fibrosis; however, its role in remodeling after acute myocardial infarction (AMI) has not received sufficient attention. Post-AMI measurements of global longitudinal strain (GLS) are beneficial in providing information about infarct area and remodeling. We aimed to determine the effect of N-acetylcysteine (NAC) on Gal-3 and GLS in AMI.

Design: This was a randomized, single-blind study with pre- and post-treatment evaluations performed from May 1 to August 31, 2018.

Setting: Dr. Moewardi Hospital, Indonesia

Patients: ST elevation myocardial infarction (STEMI) patients who received fibrinolytic therapy were randomly allocated to NAC and control groups.

Interventions: A total of 32 STEMI patients were administered fibrinolytic therapy (17 patients were administered standard therapy plus 600 mg NAC orally three times a day for 72 hours and 15 patients were administered standard therapy plus placebo as the control). Gal-3 samples were taken during admission and at 72 hours in both groups, while GLS measurement was only performed 72 hours after admission.

Measurements and results: Gal-3 levels in the NAC and control groups at admission were not significantly different; however, levels were significantly different after 72 hours (p=0.017). After comparing Gal-3 levels during admission and at 72 hours, the NAC group showed significant differences between Gal-3 levels at the time of admission and at 72 hours (p=0.0001); no difference was found in the control group. There were also significant intergroup differences in Gal-3 level changes (p=0.014). In the NAC group, a better and significantly different 72-h GLS value was obtained from that in the control group (p=0.023).

Conclusion: Supplementary therapy with NAC can reduce Gal-3 levels and GLS in AMI patients receiving fibrinolytic therapy.

Authors: Trisulo Wasyanto, Akhmad Jalaludinsyah, Ahmad Yasa

Analysis of sepsis and septic shock 3- and 6-hour management at resuscitation room in Dr. Soetomo General Hospital

Abstract

Objective: To provide a record of the implementation and outcome of surviving sepsis campaign 2016 at Dr. Soetomo General Hospital, Surabaya, Indonesia, such as 3- and 6-hour sepsis bundle compliance as a baseline and the Sepsis-related Organ Failure Assessment (SOFA) score after 48 hours of treatment. SOFA values were used to predict mortality in the hospital.

Design: This was an observational experimental study that used cross sectional design.

Setting: Resuscitation room in Dr. Soetomo General Hospital.

Patients and participants: A purposive sample was taken of patients older than 17-year-old suspected with sepsis or septic shock according to diagnosis criteria from Surviving Sepsis Campaign (SSC) 2016. Thirty-two patients, consist of 24 male and 8 female patients were included.

Interventions: After patients were suspected of sepsis or septic shock according to diagnosis criteria from SSC 2016, they were treated with 3- and 6-hour sepsis bundle, then the data were collected with questionnaire.

Measurements and results: We found 75% of patients received a 3-hour sepsis bundle in <3 hours, and 50% of patients had 6-hour sepsis bundle in <6 hours. The compliance rate of 3- and 6-hour sepsis and septic shock bundles reached 46.88%. SOFA scores before and after 48-hour management of sepsis had a significant increase with $p=0.001$ ($p<0.05$).

Conclusions: There were significant decreasing of SOFA values in baseline and 48 hours after the management of sepsis and septic shock in 81.25% patients (n=26). This result suggests that management of sepsis based on SSC 2016 contribute to the improvement of the patient's condition and better prognosis.

Authors: Arie Utariani, Bambang Pujo Semedi, Rizki Anestesia, Hamzah, Eddy Rahardjo, Elizeus Hanindito

Correlation between zinc plasma level to inflammation response of patient with ventilator in pediatrics intensive care unit

Abstract

Objective: Critical conditions arise when there is a threat or ongoing organ failure that disrupts the balance of the body's oxygen and physiological needs. Patients often require help, such as endotracheal intubation procedures, mechanical ventilation, and renal or liver replacement therapy, in place of impaired organ function. Usually, complications are more common in children than in adults. Zinc is one of the micronutrients that plays a vital role as an antioxidant and the role of defense immune modulators against systemic inflammatory response syndrome (SIRS). Outcome patient with ventilator depends on SIRS response to the disease. Meanwhile the study about zinc supplementation in Pediatric Intensive Care Unit (PICU) is limited. The main objective of this study was to look at the correlation of plasma zinc levels with inflammatory responses in children of 1-12 years old with ventilator in pediatric intensive care unit.

Design and setting: This study was prospective study in Sanglah General Hospital, Denpasar and was calculated by Pearson analysis to determine the correlation of variables plasma zinc with levels of pro-inflammatory cytokines interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-α) and continued by general linier model analysis.

Results: More than 70% sample was insufficiency zinc for the first 24 hours and after 72 hours, respectively. Correlation between plasma zinc and pro-inflammatory cytokines

were TNF-α; p<0.001 r: -0.91, and IL-6; p=0.013, r: -0.48 in 24 hours, and IL-6; p>0.05 r: 0.011, and TNF-α; p<0.01 r: 0.659 in 72 hours.

Conclusions: There were significant correlation in TNF-α and IL-6 measurement between zinc plasma insufficiency with pro-inflammatory cytokines in the first 24 hours and significant correlation in TNF-α in 72 hours. Therefore, zinc supplementation in 72 hours from patient admission to PICU may have important role to reduce morbidity in PICU.

Authors: Dyah Kanya Wati, Lanang Sidiartha, Ketut Tunas, Andrie Setiawan

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Anesthesia and intensive care management in acute ischemic stroke patient

Hamzah, Prananda Surya Airlangga, Abdulloh Machin, Nancy Margarita Rehatta

Abstract
Acute ischemic stroke is a brain functional disorder, which cause high disability and mortality rate worldwide, the second most common cause of dementia, and the third leading cause of death. It has enormous clinical, social, and economic implications and demands a significant effort from both basic scientists and clinicians in the quest for understanding the underlying pathomechanisms and producing suitable preventive measures and successful therapies. Management of acute ischemic stroke has been revolutionized by the introduction of several interventions, such as prehospital and stroke unit care, intravenous tissue plasminogen activator therapy within 4.5 hours of stroke onset, aspirin therapy within 48 hours of stroke onset, decompressive craniectomy for supratentorial malignant hemispheric cerebral infarct, and more recently endovascular therapy for anterior circulating stroke. Also, special attention in management of vital systemic physiological variables, including oxygenation, blood pressure, temperature, and serum glucose. In line with this, the role of neuroanesthesiologists and neuro critical care in managing acute ischemic stroke become more prominent.

Key words: Acute ischemic stroke, acute ischemic stroke management, neuroanethesiologists, neuro critical care.

Introduction

Acute ischemic stroke (AIS) is a condition that causing focal and global brain physiology disfunction with high morbidity and mortality rate. (1,2) Focal AIS is caused by thrmbosis or cerebral arthery occlusion. (3) Nowadays, the amout of AIS patients are increasing and 15-20% of them may require intensive care management and monitoring. (1,4)

Recent AIS management has developed progressively, from intravenous administration of recombinant tissue-type plasminogen activator (rtPA) within 4.5 hours from the onset, until the other new arterial intervension such as intra-arthery thrombolysis, angioplasty stenting, and decompression craniectomy in malignant brain infarction that may induce severe brain oedema and mass-like effect. (5) There are some new level 1 evidence-based medicine regarding the successful outcome of endovascular treatment in anterior cerebral stroke. (6,7)

Rapid management in various systemic problems, such as blood pressure control, body temperature, blood glucose level, and oxygenation would need a closed monitoring by a neuroanesthetist - neuro critical care doctor in the ICU. Clinical improvement is an indicator of a success patient management. Neuroprotective concept that was proposed after many experimental researches before, failed to improve patient clinical outcome. This condition may occur because there are too

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many risk factors that associate with AIS, such as heterogeneity of AIS in human, and lacking of standardise clinical and preclinical research method. These last two decades research showed that endovascular intervention in AIS patients could significantly improve patient outcome. (2,8,9)

This review article would describe the intensive care management for AIS patients based on the latest studies. The writers hoped that through this article they can provide a treatment guidelines for AIS patients and open up research opportunities in this sector.

Stratification and etiology of AIS
Since 1980-1990, many brain imaging modalities have emerged, including echocardiography and doppler ultrasound, which help clinicians diagnose a lacunar infarct, big artheroscleotic plaque, and other source of cardioemboli stroke. (10) Stroke was classified by stroke data bank and Trial of ORG 10172 in Acute Stroke Treatment (TOAST) into:
- Large artery atherosclerosis
- Cardiac embolism
- Small artery occlusion
- Stroke of other determined etiology
- Stroke of undetermined etiology

This system helps the clinician to group AIS patients based on the etiology of stroke. TOAST stratification method is not only useful in clinical and epidemiological studies, but also in accomodating all AIS patients. (10,11)

Large artery atherosclerosis
Based on its pathological study, atherosclerosis lesions can be found in bifurcations and big arterial arches. The more proximal the lesion is, the more dangerous the atherosclerosis lesion becomes. Atherosclerosis lesion can cause intravascular stenosis, while the rupture of atherosclerotic plaque can induce inflammation process, which leads to complete occlusion of the blood vessel. (12)

One of the most common locations of large artery to become atherosclerotic is the carotid artery. At first, stenosis in the carotid artery is usually asymptomatic and preceded by infarction, which occurs in the left anterior descending artery (LAD). Perfusion disturbance in the carotid artery may interfere or cause an occlusion in the distal vessel(s) and increases the risk of infarction in the LAD. In carotid artery occlusion, the affected areas are usually supra-sylvii, frontal, central, and parietal area. (11,12)

The cause of perfusion disturbance is commonly emboli fragment, released from atheroma ulcers, which clogs the distal arteries. Another cause of perfusion impairment is a sudden blockage in the stenotic area, which causes decrease perfusion to the distal area. (13)

Cardioembolic stroke
Cardioemboli occur in about 14-30% of ischemic stroke patients. (14) Stroke that's caused by embolism mostly happens in the middle cerebral artery (MCA). (15) Blood clots in the heart are usually caused by blood flow stasis or clots in prostheses. (11,16)

In neuroimaging, cardioembolic strokes may appear in more than one artery. It is more common in the anterior circulation than the posterior one, but it can occur bilaterally. (11,12)

Small vessel disease
This mechanism happens due to the occlusion of microcirculation in the small blood vessels. (17,18) The etiology of this stroke subtype is the presence of lipohyalunosis in the vessel wall, while an increase in microatheroma also contributes to this pathology. (17,18) The most common location of this stroke is the deep region in the substantia alba, internal capsule, and its penetrating branches, pons, brainstem, and thalamus. Infarction caused by this mechanism is usually small (<1.5 cm) and the symptoms depend on the location in which the infarction occurs. The stroke is called the "lacunar syndrome" when characterized by: 1) pure motor symptoms, 2) pure sensory symptoms, 3) sensorimotor mixture, 4) ataxia hemiparese, and 5) dysarthria-clumsy hand syndrome. (12)

Other determined etiology
The causes that include in this category are extra-cranial artery dissection, strokes that are caused by vasospasm associated with subarachnoid hemorrhage (SAH), hereditary arteriopathy (such as Moyamoya disease), vasculitis caused by infections, and strokes caused by coagulopathy. (13)

Undetermined causes
Patients who have undergone complete screening for heart problems, large artery diseases, coagulopathy, and other possible conditions, but the cause of the stroke remains unknown. (11)

Pathophysiology of AIS
The pathophysiology of AIS is very complex, but in general there is a regional obstruction in cerebral blood flow resulting in an ischemic area in the

brain tissue (ischemic core), while the surrounding area experiences hypoperfusion known as the penumbra region. (19) The ischemic nucleus is considered to be an irreversible area and the penumbra is considered to be an area that has good potential for a complete recovery. (20,21)

AIS diagnosis
Diagnosis of AIS is generally made through careful and thorough history-taking, neurological, and physical examination, as well as radiological imaging. (22,23)

Anamnesis
The most important information to be obtained from the AIS history is the onset. When the last time the patient felt normal? If the patient is unconscious, when the last time the patient was symptoms free? Another history that needs to be explored is the condition around the patient when the symptoms appeared. Even though it is not always accurate, it can help the clinician to find another diagnosis or get rid of the differential diagnosis. It is also important to ask about the risk factors such as history of atherosclerosis, heart disease, history of drug abuse, migraines, seizures, infections, trauma, and pregnancy. Obtaining these data is crucial in determining the treatment options. (24)

Physical examination
After the primary survey is carried out, a further general physical examination is done to ascertain the potential causes of AIS, other comorbid factors, or other matters that might affect the selection of AIS therapy later.

Neurological examination
The initial neurological examination must be brief, but thorough. Every ischemic region would present a distinct symptom. A standardized neurological examination will help the clinician in performing neurological examination component precisely, quickly, and uniformly. The commonly used standard is the National Institute of Health Stroke Scale (NIHSS). Based on this scoring system, the clinical severity of the disease can be determined, where the higher the score the poorer the outcome would be. At the present, NIHSS (**Table 1**) is also one of the criteria for thrombolysis therapy and the best predictor of large vessel occlusion. (25) Stroke is one of the most common cause of disabilities in adult patients, therefore efforts have been made to quantify the level of disability in adults through modified Rankin Scale (mRS). (26) Values ranging from 0-2 in this table are considered as

good outcomes after an AIS episode. (27)

Diagnostic test
Some diagnostic tests need to be done routinely in all patients suspected with AIS to rule out other differential diagnoses, especially hemorrhagic stroke. Diagnostic tests can help the clinician to identify other comorbid diseases, to determine treatment options and to find out the possible complications that may occur. These tests may include blood glucose, serum electrolytes, kidney function tests, complete blood count with platelet count, cardiac markers, prothrombin time (PT), international normalized ratio (INR), activated partial thromboplastin time (aPTT), and electrocardiogram (ECG). (28)
Blood glucose levels are important to be checked before the administration of rtPA, because hypoglycemia can cause focal signs and symptoms that resemble AIS. (29) Platelet count and PT/INR in AIS patients are important especially in patients with thrombocytopenia, impaired blood clotting function, liver dysfunction, or patient with anticoagulant medication. (24)
Cardiac examinations such as electrocardiogram and cardiac markers are essential because of the strong association between stroke and cardiac abnormalities. An increase in cardiac markers occurs in 5 to 34% of patients, whereas an increase in troponin T is associated with severity, outcome and mortality. (24) Before giving thrombolysis therapy, the only laboratory test that need to be checked is blood glucose level, except in certain cases when the patient has blood coagulopathy, the doctor may need to order some more tests. (25)

Diagnostic radiological imaging
Brain imaging is not only capable of identifying the type and cause of stroke, but also distinguishing between ischemic and hemorrhagic stroke. In addition, it can also differentiate an irreversibly damaged tissue from areas that may be recovered, thus it is useful to determine the patient's therapy in emergency situation and to predict outcomes. Vascular imaging can identify the location and the cause of arterial obstruction, in addition to identifying high risk patients. (28)

Computed tomography scan (CT scan)
All patients suspected with transient ischemic attack (TIA) or stroke are recommended to get an emergency head CT scan. (25,27,28) Patients suspected with TIA are equally important to get a CT scan because up to 10% of TIA patients suffer from strokes in the next 48 hours. (28)

Stroke patients are more prioritized than other patients to get a brain imaging, because the outcome of the patient is determined by how soon the patient can get a proper treatment. Head CT scan for patients suspected with stroke has proven to be the most cost-effective diagnostic strategy. In the hyperacute phase, CT scan without contrast is the main investigative method to eliminate other possible diseases such as stroke-like symptoms (stroke mimics) and bleeding lesions, which will influence the physician decision regarding the patient management. That's why head CT scan is the first choice of radiological imaging modality in stroke patients. (25,27)

Initial CT in ischemic stroke will show some abnormalities such as hypodensity in more than 1/3 cerebral parenchyma in areas that receive blood flow from the middle cerebral artery, hypodensity in lenticular nucleus, blurring of the cortical sulcus, focal parenchymal hypodensity, loss of differences in gray matter in the basal ganglia area, large vessel hyperdensity (e.g. middle cerebral artery hyperdensity, which indicates the presence of intraluminal thrombus), and cerebral edema with blurring of cerebrospinal fluid space. (27,28) CT scan is very specific in identifying the signs of ischemic brain damage, but sensitivity is only 61%. Two-thirds of patients with moderate to severe strokes have marked ischemic changes within the first few hours, but less than 50% of patients with mild strokes have ischemic lesions that appear relevant on CT scans, especially in the first few hours after the onset. The initial changes in brain CT scan are correlated with patient poor outcome and the success rate of reperfusion therapy. However, currently there is insufficient evidence for signs of early stroke changes being the reason of delaying thrombolysis therapy. (25)

Sometimes, changes in the initial CT scan of AIS are missed, that's why a scoring system is developed to improve the detection of early ischemic changes. (30) One of the most widely used standardized methods is the Alberta Stroke Program Early CT Score (ASPECTS). (30) ASPECTS has been developed to recognize the early signs of ischemia. This scale assesses two axial pieces on the CT scan: the first one is at the level of the thalamus and the basal core, the other is at the basal level of the superior ganglia. The method of assessment is as follows: The middle cerebral artery is divided into ten regions. Subcortical structures have a score of 3, each divided into caudate, lentiform nucleus, and internal capsule. The cortex associated with the middle cerebral artery has a score of 7; 4 points derived from axial pieces at the level of the basal

ganglia for the insular cortex, area M1, M2, and M3; 3 points originating from subsequent cuts for regions M4, M5, and M6. One point is reduced for each area with signs of initial ischemia (hypodensity or edema). (31)

ASPECTS is very helpful in predicting patient outcomes, especially for endovascular management in the acute phase, but this scoring system cannot be applied to lacunar strokes, ischemia in the midbrain, or in other ischemic lesions involving arterial areas other than MCA. (27)

CT angiography

This method is used to evaluate the lumen of the intracranial artery and its branches. CT scans with contrast can give an insight of how contrast media is distributed in the area of the cranial blood vessels and show the clogged artery as having filling defects. This method can also assess the patency of the extracranial carotid system while seeking stenosis or a barrier that can cause AIS. Therefore, this method becomes important for the provision of potential therapy in AIS, especially for mechanical thrombectomy. (6) Noninvasive vascular imaging is recommended during the initial evaluation of patients who are planned to receive endovascular therapy, especially for patients with large vessel occlusion. But this diagnostic modality should not be a reason to delay thrombolysis therapy. (25)

CT perfusion

This method performs repeated imaging of the same cerebral parenchyma as bolus of intravenous contrast media is given. The goal of this imaging study is to map the overall volume of brain perfusion. The images need to be analyzed and interpreted where the cerebral ischemic area will appear hypodense. (32) This method can also estimate the cerebral blood flow (CBF), cerebral blood volume (CBV), and mean transit time (MTT) needed for blood to flow from the vascular compartment to cerebral tissue by quantitative analysis of contrast media kinetics. (33) CBF and CBV boundary values can be used to predict whether the brain parenchyma may survive or not. ASPECTS has been used in mapping CBF or MTT to identify the maximum expansion of ischemia region by assessing the absence of reperfusion, and the difference between CBV and CBF (or MTT) to identify the area of ischemic penumbra. (6)

Magnetic resonance imaging

Immediate CT scan is the most cost-effective imaging strategy for acute stroke patients, but it is insensitive for prolonged bleeding. That's where

magnetic resonance imaging (MRI) can be used as an alternative modality when non-contrast CT lesions are not seen or when the clinical location of stroke is unclear at the time of stenting. However, delaying the administration of thrombolysis therapy because of waiting for the MRI result is not recommended. (25)

MRI is time consuming in obtaining and reconstructing the images. It is less suitable for restless patients or patients with possibility of vomiting and aspiration. If needed, emergency life assistance should be provided during the MRI process, as AIS patients (especially those with severe stroke) tend to become hypoxic when lying supine during imaging process. Therefore, MRI is rarely used in emergencies and is not included in acute stroke protocols. However, MRI is a radiological modality that has 100% diagnostic accuracy in detecting bleeding lesions. In addition, a certain sequence such as gradient echo (GRE) can help physician in distinguish acute changes from chronic forms. (34)

DWI and PWI
Diffusion-weighted imaging (DWI) is a method based on the ability of MRI to detect signals from the movement of water molecules based on differences in Brownian motion. (35) This method can detect anomalies associated with cerebral ischemia within 3-30 minutes of the onset of symptoms, when there are no abnormal changes in traditional MRI and CT scans. (35) DWI can distinguish between cytotoxic and vasogenic edema using apparent diffusion coefficient (ADC). The ADC can measure the level of water diffusion. In this context, hypointense signals in ADC mapping are in accordance with cytotoxic edema, while hyperintense signals represent vasogenic edema. This method also gives a predictive value of clinical and functional outcomes. (36) Tissues that appear abnormal on DWI are identified as having ischemic damage, where tissue with a slight reduced ADC can be permanently damaged; but, there is no ADC threshold can be relied upon to distinguish dead tissue from living tissue. (37) Perfusion-weighted imaging (PWI) can identify areas of ischemia through MRI by measuring the amount of contrast that reaches brain tissue. (38) Map of CBF, CBV, and MTT are processed through several stages of scanning in the same brain parenchyma area. This method can be used to assess whether the brain tissue is still salvageable or not, assess the severity of lesions, considering the choice of therapy where reversible ischemic brain damage is a key factor in selecting patients for reperfusion, and monitoring patients. (36) The pattern of missed match between DWI and PWI highlights the area of cerebral parenchyma that can still be saved. Specifically, while DWI detects irreversible-damaged brain parenchyma (ischemic nucleus), PWI can show hypoperfusion areas (ischemic penumbra region) by reducing the irreversible areas seen on DWI. (39)

MR angiography
This method could show any signs of stenosis or occlusion in extra- and intracranial vessels. The sensitivity and specificity in detecting vascular lesions vary from 86 to 97% for CT angiography and 62 to 91% for MRI angiography. Acute thrombolytic occlusion is usually shown as a hypointense image in a large vessel (middle or carotid cerebral artery). (40) However this method is not recommended for AIS patients who are admitted to the hospital in less than 6 hours after the onset. (25)

Neurosonology
Carotid color Doppler and transcranial Doppler are two noninvasive methods that can be used for neurovascular assessment. This method can assess serious vascular stenosis or identify the cause of a stroke such as vascular occlusion, or for carotid and vertebral arteries surgery. Transcranial Doppler works by utilizing the emission of low frequency pulsating sounds that can penetrate the bone, allowing identification of stenosis, occlusion, collateral circuits, and possible reperfusion after thrombolytic treatment.

This method has some advantages such as practical and as a bedside diagnostic modality, but it requires an experienced operator. The combination of duplex ultrasound imaging techniques and magnetic resonance angiography (MRA), which is relatively risk-free, can diagnose carotid artery stenosis. Digital subtraction angiography (DSA), which has 1-3% risk of stroke in patients with carotid lesions, might be used as well. (41,42) Transcranial doppler is the only technique that could detect circulating intracranial embolism, which is very common in patients with large arterial disease. (43) Symptomatic carotid artery stenosis is a strong independent predictor of recurrent acute ischemic stroke and TIA. That's why it should be used to evaluate antiplatelet therapy. (44)

Ultrasonography has some limitation such as the difficulty in finding a bone window and inability to provide a good image of posterior circulation thet need to be measure repeatedly. In this condition, CT angiography is preferred in clinical practice, especially in urgent situations. (28)

Conventional angiography
This imaging modalities is the main and most accurate technique for evaluating circulation such as detecting stenosis, occlusion, or dissection. Using cerebral angiography, we can study collateral circulation and the level of cerebral perfusion. Recently the usage of cerebral angiography is reduced due to the discovery of other accurate non-invasive alternatives such as CT angiography, MRA, and duplex ultrasonography. (41,42) Nowadays, angiography is used by neuroradiology interventionists to perform neuro-reperfusion therapy which combines pharmacologycal and mechanical therapies. (27,42)

Management
Determining indications of intensive care
All AIS patients are better treated in intensive care unit, more specifically the stroke unit, with standardized protocol. (45) The number of patient who require ICU care management keeps increasing, but there is still no concencus or guidelines for that. This consideration has relied only on hospital regulations. (46)
Studies showed that there is no evidence that managing AIS patients in ICU gives better outcome, especially in patients with minimum emergency. As an example, a retrospective study involving 138 AIS patients who entered two different hospitals with different regulations, showed no significant difference of length of stay. (47)
Monitoring and management of systemic physiology in the patient with acute ischemic stroke in the ICU is summarized in **Figure 1**. Management of intracranial issues and complications in acute ischemic stroke in the ICU is summarized in **Figure 2**.

Blood pressure and fluid management
More than 80% of AIS patients suffer hypertension (systolic blood pressure >140 mmHg). Some of the cases might be related to chronic hypertension, stress response, increase of intracranial pressure (ICP), or neuroendocrine. (48) Hypertension has been reported to be associated with increase of mortality during endovascular therapy, the same association was found with hypotension. (49) Data from International Stroke Trial showed that both hypertension and hypotension have damaging effects, with U-shaped graphic relationship, showing an increase of 3.8% mortality for every 10 mmHg increase above 150 mmHg systolic blood pressure, and 17.9% increase of mortality for every 10 mmHg decrease. (50) At the present, consensus states that target of systolic blood pressure is <185 mmHg and diastolic <110 mmHg before fibrinolysis begins. It is known that bleeding risks post fibrinolysis therapy will increase in patients with higher blood pressure, eventhough it has not yet been established at what level does the blood pressure start to increase the risk of bleeding. Therefore, within 24 hours post fibrinolysis the target systolic blood pressure is <180 mmHg and diastolic <105 mmHg. (25)
Currently, the suggested therapy is using labetalol 10-20 mg IV within 1-2 minutes, can be repeated once, or using nicardipine with intial dose 5 mg/hour IV with titration 2.5 mg/hour every 5-15 minutes (maximum 15 mg/hour). Afterward, blood pressure is checked every 15 minutes for 2 hours, continued by blood pressure check every 30 minutes for 6 hours, and every hour for the following 16 hours. If during observation systolic blood pressure >180-230 mmHg or diastolic blood pressure >105-120 mmHg, labetalol can be given 10 mg IV within 1-2 minutes, and continuous IV 2-8 mg per minute, or nicardipine with initial dose of 5 mg per hour IV, titrated to 2.5 mg per hour every 5-15 minutes (maximum dose 21mg per hour). If the systolic blood pressure remains >140 mmHg, sodium nitropruside IV can be administered. (25)
Some patients, such as those with moderate carotid stenosis, however, might gain advantage with hypertension. High blood pressure may give rise to cerebral blood flow in these cases, but further observations are required in establishing the advantage of this therapy. (46)

Reperfusion therapy
Reperfusion is currently the best option to restore blood flow to penumbra zone. Introduction of intravenous thrombolysis and endovascular therapy, such as intra-arterial thrombolysis, use of angioplasty/stenting, has changed management of AIS. Differential diagnosis of hemorrhagic stroke must be done first using CT scan without contrast. If AIS diagnosis has been established, and there is no contraindication, based on the standard therapy, intravenous rtPA is given within 3 hours after the onset of stroke (0.9 mg/kg, maximum dose 90 mg within 1 hour, with 10% of early dose given in the first minute), although risk-and-benefit considerations must also be taken. (25) This therapy showed better outcome when given earlier, and it has also reported to lower dissability on day 90 and 180 post AIS. (24) Furthermore, in 2015, there had been many new suggestions of therapy, such as systemic and endovascular therapies, use of mechanical thrombectomy (stentrievers), as well

as arguments of rtPA administration time limits. Current endovascular therapy can be summarized into:

1. CT scan is compulsary to eliminate hemorrage, no matter how small.
2. Rdiological imaging with contrast (CT scan or MRI) shows proximal occlusion from major artery branch and the anterior circulation.
3. Procedure must be done in a hospital, which is facilitated by cardiovascular intervention diagnostic installation, especially for performing stent retreiver.
4. Mechanical thrombectomy must be performed in less than 6 hours of period. (2)

Choices of anesthesia techniques during endovascular therapy
Anethesiologists play an important role in the management of endovascular AIS. The choice of anesthesia technique is made based on several factors, including airway, breathing, hemodynamics, level of consciousness, agitation, and ability to lie still. The choice has to be made quickly because 30-minute delay in starting thrombolysis may reduce 3-month success rate up to 10%. Endovascular therapy can be performed using general anesthesia (GA) or measured anaesthesia care (MAC), which uses local anethesia, with or without sedation. Retrospective studies so far showed that MAC gave better results compared to GA. This result, however, may be due to the fact that in GA group, many involve stroke in the posterior circulation, which is associated with poorer outcome. In addition, most of the patients in the GA group had worse conditions in the beginning. Therefore, more studies are required to determine the standard choice of anethesia technique during endovascular therapy.

Oxygenation and ventilation management
Hypoxia is commonly found in AIS and very influential to the patient care outcome. It is, therefore, very important to identify the causes of hypoxia, which may include neurological and non-neurological factors, such as aspiration pneumonia, acute lung injury, pulmonary emboli, and central respiration arrhytmia.
Established studies so far showed that hyperbaric therapy (51,52) and routine oxygenation supplementation in non-hypoxic patients (41) worsen their stroke severity and condition. Current guidelines from American Stroke Association and European Stroke Organisation recommend oxygen supplementation only in patients with SpO2<94%, who can breathe on their own. (24,28)

Continuous monitoring of arterial oxygen with pulse oximeter is required in all AIS patients in ICU, while blood gas analysis is measured regularly during the use of mechanical ventilation, in order to observe and manage PaO2 (>80 mmHg) optimally. Hypocapnia (PaCO2<35 mmHg) is associated with poor outcome post stroke attack, therefore, normocapnia (PaCO2 35 to 45 mmHg) is the target for intubated patients. For patients with spontaneous ventilation, SpO2 is suggested to be maintained at >92%, (53) while SpO2 levels during endovascular therapy and ICU stay is maintained at > 94%. (1)
More than a third of patients with severe AIS need tracheostomy. Indications of tracheostomy including the use of long-term mechanical ventilator (for 2 weeks or more), severe dysphagia and/or bulbar palsies due to extensive brain and brain stem infarction. (54) Based on a systematical evaluation of 11 studies involving 886 patients, the best method to detect dysphagia in patients who are not intubated is through water test, which has 73%-98% sensitivity and 63%-76% specificity. This test is done by giving an aliquot of water, which has been determined before, to evaluate the ability to swallow using coughing, choking, and voice changing as markers, while continuous monitoring using pulse oximeter is done. (55) The time when tracheostomy is needed is debatable. Generally, tracheostomy is considered after 1 week use of mechanical ventilator in stroke patients. (56) However, some studies showed that patients who received tracheostomy 7 and 14 days after mechanical ventilator use didn't have any complications due to tracheostomy, using the length of ICU stay, sepsis severity, and bleeding as indicators. (54)

Glucose serum management
More than 40% of patients suffer hyperglicemia in acute phase of AIS. The high level of serum glucose is an indicator of disease severity, which is associated with increase of cortex toxicity, larger infarct volume, poorer outcome, and infection risk. (57) Pathophysiology of hyperglicemia associated with death in AIS has not been fully understood, but is suspected involving many factors, and the failure of recovery in ischemic penumbra zone to be the key. Other mechanisms, which are suggested to play a role including increase of coagulation and decrease of fibrinolytic activity, which inhibit recanalization, vasodilation inhibition, which causes reduced cerebral blood flow (CBF), reperfusion injury duse to oxydative stress and inflammation. (47) Hyperglycemic management af-

ter AIS currently targets serum glucose level at 70-140 mg/dl during ICU care. (1) Hypoglycemia in AIS patients must be dealt with when the serum glucose level reaches <60 mg/dl. (25) Suggested method in the present is by continuous glucose infusion to avoid extreme low serum glucose levels.

Temperature management and hypothermia therapy

Fever can be found among up to 35.5% AIS patients and is associated with poor outcome. (58) Infection, which causes the fever, must be found and treated, but it can also be caused by central inflammatory response without the presence of infection. Fever must be treated aggressively. Management may include the use of acetaminophen as the first line, metamizole, rapid cold saline (4 $^{\circ}$C) infusion, or automatic cooling system. Suggested target temperature is 35 $^{\circ}$C to 37 $^{\circ}$C in patients with endovascular therapy and <37.5 $^{\circ}$C in ICU patients. (1)

Different to hyperthermia, hypothermia in early care post AIS is associated with better outcome. (59) Therapeutic hypothermia (TH) has shown many neuroprotective effects. (60) However, there are some risks associated with TH, including higher pneumonia incidence, shivering, electrolyte imbalance, and cardiac dysfunction. (25) Additional airway management has become a challenge in doing TH after AIS, and may be an indicator itself to have an ICU treatment care. (61) Although local cooling (in head only) through nasal air flow, conduction through nasal catheter, air, gel caps, and other methods can reduce systemic risk, (62) there are also some reports of higher complication events and equal mortality with conventional therapy, which suggests the need of more research about its use outside trials. (63) In vitro studies showed that lower temperature (<37 $^{\circ}$C) may disturb the performance of rtPA, (64) therefore, more studies in clinical trials context are required to observe the effects of TH in thrombolytic therapy. (25)

Anticoagulation, antiplatelet therapy, and thromboprophylaxis

Anticoagulation, antiplatelet therapy, and thromboprophylaxis after AIS is made harder by intravenous thrombolysis and endovascular therapies. Data available in this topic is not specific for endovascular therapy or ICU treatment care. Moreover, the optimal agent and dose have not been clearly stated. Currently, the use of these agents are avoided during the first 24 hour after

thrombolysis, but this approach still needs further evaluation. (2)

Class I evidence supports the use of high dose aspirin (160-325 mg) in 24-48 hours after AIS begins. (24) However, this approach does not substitute endovascular therapy. Evidence supporting other antiplatelet such as clopidrogel is still weak. History of antiplatelet use also showed an increase of symptomatic ICH incidence. (65)

Heparin is used during endovascular therapy to minimize thrombosis and emboli risks due to catheterization. Moreover, combination of aspirin and clopidrogel are often used during endovascular stent insertion. (66,67) Until now there is no strong and consistent evidence stating the correlation between the use of oral anticoagulant and bleeding post AIS, including the new anticoagulants such as dabigatran and rivaroxaban. The choice to use of anticoagulants in these cases, therefore, is made by the doctors with considerations of its risks and benefits. (2)

Thromboembolism prevention can be facilitated by early hydration and mobilization. Graduated compression stocking does not reduce the incidence of thromboembolism. (68) Other studies showed that intermittent pneumatic calf compression reduced risks of deep vein thrombosis in 30 days after trial entry, with improvement of survivability in 6 months, but without significant increase of function and quality of life. (69)

The use of low molecular weight heparin (LMWH) in vein thromboembolism prevention is supported by class I evidence. The type and dose of LMWH chosen must be adjusted by local guideline. Current evidence showed that the best option is using subcutaneous enoxaparin 40 mg single dose as reported in PREvention of Venous thromboembolism after Acute Ischemic stroke with Low molecular weight heparin (PREVAIL) study. (70) There is no clear guideline on when to start giving LMWH, but reports suggested that it is better to start early with interlude of 24 hour minimum after reperfusion therapy to avoid intracranial bleeding.

Bleeding management

Hemorrhagic transformation may occur among 5-6% of patients who receive intravenous rtPA and intra-arterial recanalization therapy, even in some patients who do not undergo reperfusion therapy. There was no significant difference of ICH incidence between patients who received intravenous thrombolytic therapy and endovascular therapy. (6,7,71-73) Presently, there is still no guidelines in managing hemorrhagic transformation, although combination of cryoprecipitate,

fresh frozen plasma, recombinant factor VII, and prothrombin complex concentrate can be used with local guidelines and expert's instructions. The administration of rtPA must be stopped immediately if hemorrhagic transformation happens and protamin should be given to patients who receive heparin during endovascular therapy. Consultation with neurosurgeon regarding the indications of the surgery may be needed, expecially in patients with extensive hematoma. (2)

Seizure and anticonvulsants
Unlike other acute brain injuries, convulsive seizure rarely happens after AIS, even in the cases related to thrombolysis. (74) Previously known seizure must be treated aggresively, but the role of anticonvulsants prophylaxis is yet to be elucidated. (75) There is no specific data describing seizure management during ICU treatment care or endovascular therapy in AIS, but in general evidence showed that the use of anticonvulsants is associated with long-term poor outcome. (76) However, most of the studies used phenytoin, therefore, it is unclear whether the same results should be expected with the new agents as well. Even though phenytoin is commonly considered as first line anticonvulsant. Levetiracetam is more preferable nowadays in medical centers. Non-convulsive seizures is also rarely found in AIS compared to other types of acute brain injuries. It is also still unclear if application of anticonvulsant therapy as medication or prophylaxis can better the outcome. Consensus reccommended the use of electroencephalography (EEG) to eliminate diagnosis of non-convulsive seizure in all AIS patients with change of consciousness levels, which is unexplainable and/or persistant. (76)

Role of neuromonitoring
Basic of neurological deterioration detection includes monitoring of conscious and cooperative patients, combination of clinical evaluation and neurologic physical examination, and radiological imaging. This also becomes the determinant of next steps in managing AIS. This applies to patients in the emergency room, reperfusion therapy patients, those treated in the ICU and in stroke unit. The intracranial pressure (ICP) of AIS patients, even those with extensive infarct and edema, is commonly normal (<20 mmHg). Routine examination of ICP is therefore not reccommended. (77)
Non-invasive neurological monitoring tools seem to be more relevant to use, where patients do not require sedation. One of the most useful tools is transcranial doppler ultrasonography, which can facilitate AIS diagnosis by detecting the response of acute MCA occlusion towards thrombolysis, aiding in determining patient's prognosis based on severity of arterial occlusion, evaluating brain blood flow and vasoreactivity, and when combined with continuous blood pressure measurements can evaluate cerebral autoregulations. (2)

Sugery
Approximately 10% of AIS involves extensive edema in the hemisphere, more commonly found in patients with internal carotid artery or middle cerebral artery or both occlusions. The most common symptoms are hemiplegia, head or eye deviation, aphasia, and/or contralateral neglect syndrome. Even the treatment with hyperventilation, manitol, barbiturate coma, and hypothermia, the mortality rate is still high (estimated between 50-78%). Decrease of consciousness is often found after 48 hours and followed by transentorial herniation during 48-96 hours afterwards. (27)
The role of decompressive craniectomy in patients with malignant infarct from MCA has been vastly studied. Indication for surgery is clear, (1) and this surgery for patients aged 18 to 60 years old with MCA malignant infarct within 48 hours after the first symptoms appear can reduce mortality up to 29-78% with improvements of patient's functions. (10) Although this surgery can increase patient's survival, and that the timing should be done as early as possible, often patients end up suffering from severe dissabilities. Therefore, careful considerations from both patients and their family must be taken.

Conclusion
Introduction of several interventions, including reperfusion therapy and decompressive craniectomy, have improved AIS management in the two decades. Furthermore, management of essential systemic physiological variables, such as oxygenation, blood pressure, temperature, and glycemic control, has been appreciated. In relation to class I evidence, which supported the role of endovascular therapy, management of AIS has become more aggressive, which involves greater contribution from anesthesiologists and intensive care. Many challenges and questions in managing AIS have not been successfully answered due to lack of data regarding AIS specific management using endovascular therapy and intensive care. Consensus guidelines in anesthesy care from endo-

vascular therapy has been published by the Society for Neuroscience in Anesthesiology and Critical Care. (53) In ICU setting, several matters that need careful observation include airway management, ventilation, optimalization from hemodynamic and fluid parameters, as well as monitoring and control of temperature, glycemic status, anticoagulant management, antiplatelet and thrombophylaxis, management of complications associated with reperfusion therapy, detection and management of seizure, and neurosurgeries. (1) Therefore, there are still plenty more specific studies needed for AIS management using intensive and endovascular therapies.

Table 1. National Institutes of Health Stroke Scale (NIHSS) (78)

Category	Score/Description		Date/Time Initials	Date/Time Initials	Date/Time Initials	Date/Time Initials	Date/Time Initials
1a. Level of Consciousness (Alert, drowsy, etc.)	0 = Alert 1 = Drowsy 2 = Stuporous 3 = Coma						
1b. LOC Questions (Month, age)	0 = Answers both correctly 1 = Answers one correctly 2 = Incorrect						
1c. LOC Commands (Open/close eyes, make fist/let go)	0 = Obeys both correctly 1 = Obeys one correctly 2 = Incorrect						
2. Best Gaze (Eyes open - patient follows examiner's finger or face)	0 = Normal 1 = Partial gaze palsy 2 = Forced deviation						
3. Visual Fields (Introduce visual stimulus/threat to pt's visual field quadrants)	0 = No visual loss 1 = Partial Hemianopia 2 = Complete Hemianopia 3 = Bilateral Hemianopia (Blind)						
4. Facial Paresis (Show teeth, raise eyebrows and squeeze eyes shut)	0 = Normal 1 = Minor 2 = Partial 3 = Complete						
5a. Motor Arm - Left 5b. Motor Arm - Right (Elevate arm to 90° if patient is sitting, 45° if supine)	0 = No drift 1 = Drift 2 = Can't resist gravity 3 = No effort against gravity 4 = No movement X = Untestable (Joint fusion or limb amp)	Left Right					
6a. Motor Leg - Left 6b. Motor Leg - Right (Elevate leg 30° with patient supine)	0 = No drift 1 = Drift 2 = Can't resist gravity 3 = No effort against gravity 4 = No movement X = Untestable (Joint fusion or limb amp)	Left Right					
7. Limb Ataxia (Finger-nose, heel down shin)	0 = No ataxia 1 = Present in one limb 2 = Present in two limbs						
8. Sensory (Pin prick to face, arm, trunk, and leg - compare side to side)	0 = Normal 1 = Partial loss 2 = Severe loss						
9. Best Language (Name item, describe a picture and read sentences)	0 = No aphasia 1 = Mild to moderate aphasia 2 = Severe aphasia 3 = Mute						
10. Dysarthria (Evaluate speech clarity by patient repeating listed words)	0 = Normal articulation 1 = Mild to moderate slurring of words 2 = Near to unintelligable or worse X = Intubated or other physical barrier						
11. Extinction and Inattention (Use information from prior testing to identify neglect or double simultaneous stimuli testing)	0 = No neglect 1 = Partial neglect 2 = Complete neglect						
TOTAL SCORE							

INITIAL	SIGNATURE	INITIAL	SIGNATURE	INITIAL	SIGNATURE

Figure 1. Monitoring and management of systemic physiology in the patient with acute ischemic stroke in the intensive care unit (1)

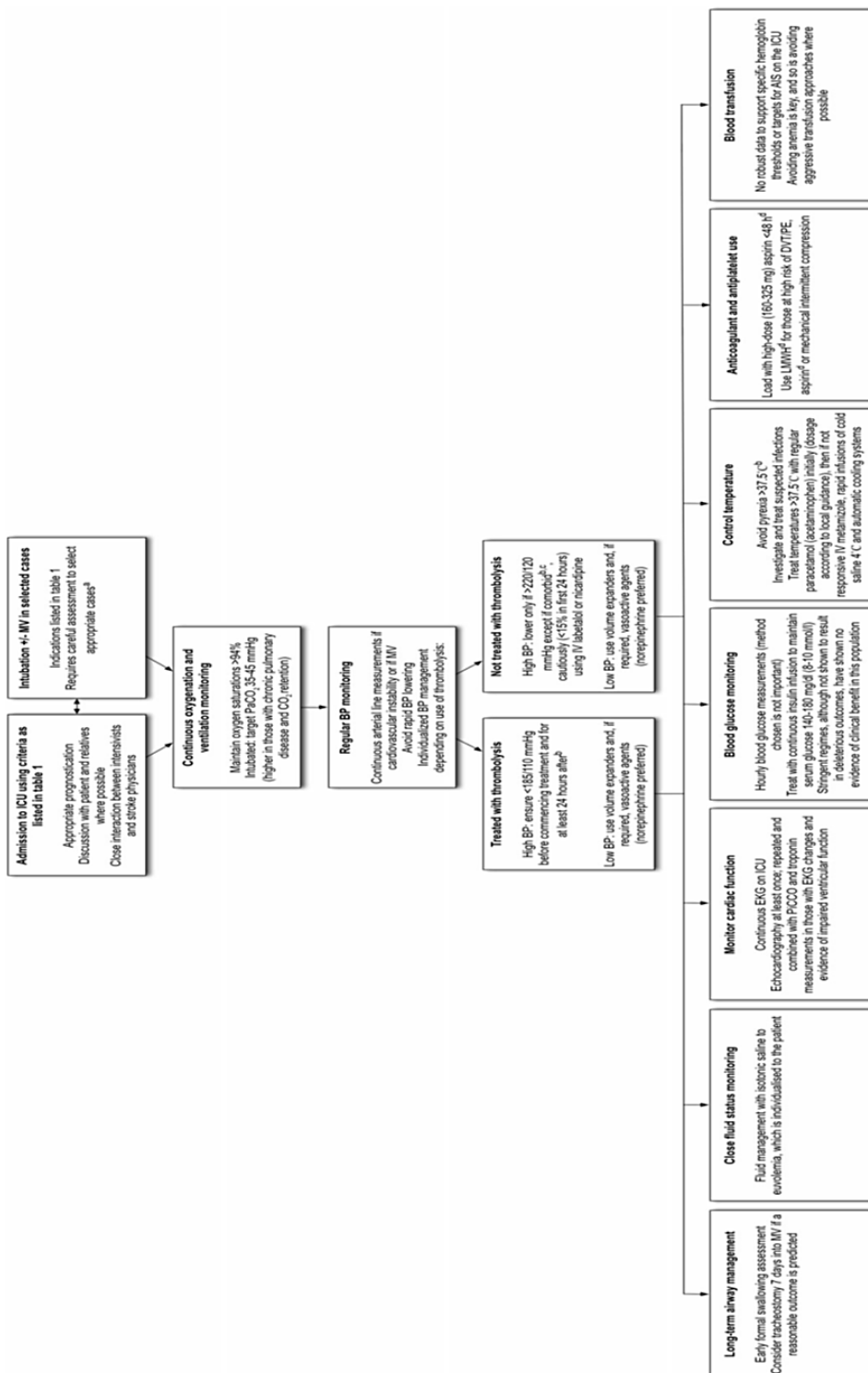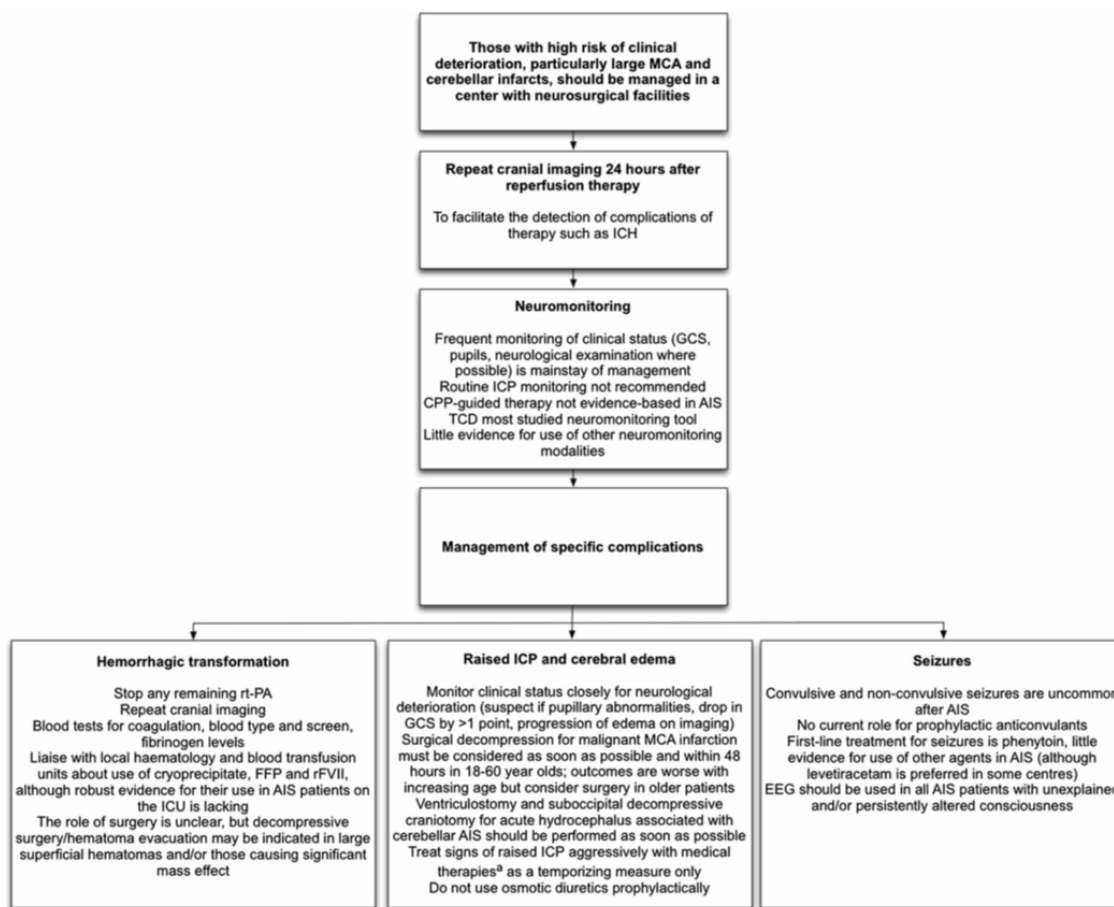**Figure 2**. Management of intracranial issues and complications in acute ischemic stroke in the intensive care unit (1)

Those with high risk of clinical deterioration, particularly large MCA and cerebellar infarcts, should be managed in a center with neurosurgical facilities

↓

Repeat cranial imaging 24 hours after reperfusion therapy

To facilitate the detection of complications of therapy such as ICH

↓

Neuromonitoring

Frequent monitoring of clinical status (GCS, pupils, neurological examination where possible) is mainstay of management
Routine ICP monitoring not recommended
CPP-guided therapy not evidence-based in AIS
TCD most studied neuromonitoring tool
Little evidence for use of other neuromonitoring modalities

↓

Management of specific complications

Hemorrhagic transformation

Stop any remaining rt-PA
Repeat cranial imaging
Blood tests for coagulation, blood type and screen, fibrinogen levels
Liaise with local haematology and blood transfusion units about use of cryoprecipitate, FFP and rFVII, although robust evidence for their use in AIS patients on the ICU is lacking
The role of surgery is unclear, but decompressive surgery/hematoma evacuation may be indicated in large superficial hematomas and/or those causing significant mass effect

Raised ICP and cerebral edema

Monitor clinical status closely for neurological deterioration (suspect if pupillary abnormalities, drop in GCS by >1 point, progression of edema on imaging)
Surgical decompression for malignant MCA infarction must be considered as soon as possible and within 48 hours in 18-60 year olds; outcomes are worse with increasing age but consider surgery in older patients
Ventriculostomy and suboccipital decompressive craniotomy for acute hydrocephalus associated with cerebellar AIS should be performed as soon as possible
Treat signs of raised ICP aggressively with medical therapies[a] as a temporizing measure only
Do not use osmotic diuretics prophylactically

Seizures

Convulsive and non-convulsive seizures are uncommon after AIS
No current role for prophylactic anticonvulants
First-line treatment for seizures is phenytoin, little evidence for use of other agents in AIS (although levetiracetam is preferred in some centres)
EEG should be used in all AIS patients with unexplained and/or persistently altered consciousness

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