

Cerebrovascular Accidents (Stroke) Types and its Medical Management

ABSTRACT

Strokes are a leading cause of morbidity and mortality across the world, in fact the third leading cause after heart diseases and cancer. Additionally, among the survivors of stroke, one-third suffers from permanent disabilities. Strokes can be classified broadly as ischemic and hemorrhagic, which account for 80% and 20% of total respectively. The prognosis of cerebrovascular accidents depends on quick diagnosis of the type, followed by appropriate and fast management. I conducted this review using a comprehensive search of MEDLINE, PubMed and EMBASE, from January 1990 to March 2017. The following search terms were used: stroke, cerebrovascular accidents, ischemic stroke, hemorrhagic stroke, stroke types, management of stroke, rehabilitation, CVA prevention. The most critical part about approaching a stroke patient is to identify the type of stroke, whether hemorrhagic or ischemic, as each type requires a different guideline of management. Also, time is the key in preserving neuronal function and preventing further damage. At the same time, the general population must be educated about methods of preventing stroke by making positive lifestyle changes.

Keywords: Stroke, Cerebrovascular accidents, Ischemic stroke, Hemorrhagic stroke, Stroke management

INTRODUCTION

One of the leading causes of morbidity and mortality is strokes (also known as cerebrovascular accidents CVA) attributing to significant negative consequences on the society.¹ Strokes are defined by the World Health Organization (WHO) as an acute, focal or diffuse, dysfunction of the brain, originating from vessels and lasting for a period longer than a day. This definition, thus, will include intra-cerebral hemorrhages, subarachnoid hemorrhages, ischemic strokes, and cerebral venous sinus thrombosis.² CVA has been considered to be the third most common cause of death in the developed world following cardiovascular diseases and malignant tumors. It is estimated that about seven hundred thousands are affected annually by CVA.³ Moreover, it is estimated that of stroke survivors, up to thirty percent will suffer from significant permanent morbidities and up to twenty percent will need rehabilitation programs.⁴ Therefore, significant budget of healthcare is needed to cover these expenses. Mortality from CVA can reach fifteen percent during the initial admission, and up to twenty five percent within the first month. Most patients will eventually return to their baseline status. However, up to thirty percent will develop significant permanent morbidities.⁵ In clinical practice, CVAs (excluding subarachnoid hemorrhages) are grouped into two main groups: ischemic and hemorrhagic. Most CVA cases (up to 80%) are ischemic, with hemorrhages being responsible for the remaining 20%. Hemorrhagic strokes cause brain damage in two main ways: ischemia due to the pressure of adjacent structures, and direct neuronal injury from the hemorrhage. Ischemic strokes originate mainly from a distant embolism or atherothrombotic occlusion.⁶ Embolisms most commonly originate from the left atrium in cases of atrial fibrillation 'AF' or the left ventricle in cases of heart failure or myocardial infarction 'MI'. The prognosis of CVA and the long term effects depend significantly on immediate diagnosis along with proper management. Determining the type of stroke is essential to properly plan the management of the patient.⁷

METHODS

Data sources and search terms

I conducted this review using a comprehensive search of MEDLINE, PubMed and EMBASE, from January 1990 to March 2017. The following search terms were used: stroke, cerebrovascular accidents, ischemic stroke, hemorrhagic stroke, stroke types, management of stroke, rehabilitation, CVA prevention

Data extraction

Two reviewers have independently reviewed the studies, abstracted data and disagreements were resolved by consensus. Studies were evaluated for quality and a review protocol was followed throughout.

DISCUSSION

Stroke types

The two main types of CVAs are ischemic and hemorrhagic. An ischemic stroke occurs as a result of the occlusion of a cerebral vessel that blocks 80% or more of the vessel. On the other hand, a hemorrhagic stroke occurs following a vessel rupture.⁶ Hemorrhagic strokes are further subdivided into intracranial hemorrhages 'ICH' or subarachnoid hemorrhages 'SAH'. Predisposing factors that significantly increase the risk of developing a hemorrhagic stroke include myocardial infarctions,

hypertension, and thrombolytics use.⁸ Clinical presentation of hemorrhagic strokes can vary on a case basis. Generally, patients present with a headache of an acute onset, along with vomiting and severely high blood pressure. These clinical manifestations are accompanied with focal neurological signs that occur in minutes. These acute manifestations have been found to relate mostly to hemorrhagic strokes, although they can sometimes occur with any other type of stroke.⁹ On the other hand, an ischemic stroke can originate from three main etiologies: thrombosis, hypo-perfusion, and embolism. Thrombosis is considered to be the most common cause. In contrast to hemorrhagic strokes, clinical manifestations of ischemic strokes progress relatively slowly (over hours) and vary in severity. Clinical manifestations of an ischemic stroke can include paralysis, paresis, ataxia, vomiting, and eye gaze. The specific site of the lesion will determine the symptoms and signs that will appear on the patient.⁶ The main prognostic factor that will diminish the rate of complications and permanent morbidities is early diagnosis of stroke, along with the determination of the type and etiology of the stroke. A thorough history with a proper physical examination will help determining the type of the stroke.¹⁰ However, imaging is still required to establish a diagnosis. The imaging modality of choice to distinguish a hemorrhagic from an ischemic stroke is non-contrast CT scan. However, it is not always available in hospitals and emergency departments. Therefore, studies have attempted to give protocols to be able to distinguish between the two types based solely on clinical manifestations. Characteristics that help determining the type include focal versus diffuse symptoms, positive versus negative symptoms, and gradual versus sudden onset of symptoms. Moreover, some studies suggested that pupil size and eye gazes can help distinguish between types.¹¹

Risk factors for stroke

Several risk factors have been linked to a higher risk of developing CVAs. These risk factors are either modifiable or non-modifiable. Non-modifiable risk factors include a positive family history of strokes, age, male gender, and black or Hispanic races.¹² On the other hand, other risk factors can be modified with behavioral changes, leading to a significantly decreased risk of a stroke. In fact, up to 80% of strokes can be attributed to these modifiable risk factors. Risk of stroke has also been linked to genetic predisposition, with several studies studying this issue. However, studies are still needed in this area to establish the clinical significance of these genetic associations.¹³ The presence of these previously mentioned risk factors will negatively impact vessels functionally and structure-wise. The structure of vessels is affected in cases of atherosclerosis and arteries stiffening, narrowing, and thickening.¹⁴ These changes will later cause significant decline in the resting cerebral blood flow (CBF) along with significant dysfunction in CBF regulation mechanisms. For example, older patients with diabetes, hypertension, and/or dyslipidemia will have impaired regulatory mechanisms leading to an insufficiently perfused brain. Moreover, the endothelium loses its ability to adapt to changes in microvascular flow, leading to a significant mismatch between energy requirements and supplies.^{15,16}

Pathophysiology

The probability of developing a stroke in an individual is affected by both vascular risk factors, and other general predisposing factors that can act as a trigger. In some cases, there is a solid cause to which the stroke can be attributed. An example of this is a trauma to the neck that cause dissection and blockage of the internal carotid artery.¹⁷ However, in most other cases, this solid cause cannot be found. Moreover, it is still unclear how some factors like pregnancy, systemic infections, and mental stress predispose to a stroke.¹⁸ Some theories suggest that coagulation cascade activation and vascular inflammation can play a role in this. According to these theories, the vascular abnormalities along with coagulopathies will work together and with other predisposing factors to enhance the initiation of stroke development (either hemorrhagic or ischemic). The presence of leukocytosis, and elevates inflammatory markers (which is a poor prognostic factor) in the setting of acute stroke supports this theory. Overall, we still need further studies to be able to understand stroke triggers and predisposing factors, and to early identify high risk cases, as our knowledge is still limited.^{18,19} The brain is generally considered vulnerable to hypoxic injury when compared to other body organs. This is mainly because the presence of glutamate (a neurotransmitter) in high concentrations, and the relatively high metabolic activity. Thus, hypoxic injury can occur as a result of cerebral vascular occlusion from an emboli, or an in-situ thrombus. Distant emboli usually originate from the left atrium, left ventricle, or from the carotid artery (specifically from an atherosclerotic plaques). Within seconds of this occlusion, neurological manifestations will occur. However, permanent injury and cell death will develop over minutes to hours (or even days) according to the site of injury, its cellular ingredients, its vulnerability, and the extent of the lesion.²⁰ Another important factor affecting outcome is the vascular response to injury. An example of this is the absence of vasodilator (like NO), which will lead to a larger stroke. On the other hand, the early recovery of flow by lysis of the clot, will lead to decreased damage. Recombinant tissue plasminogen activator (tPA) is sometimes given to achieve this, and is in fact the only drug approved by FDA for this. However, only less than 10% of stroke patients qualify for tPA use, due to its large profile of side effects.²¹ It is associated with high risk of hemorrhage and other possible adverse events like damaging the blood brain barrier (BBB). Some regimens use combination therapies to decrease the rates of side effects and enlarge the therapeutic window of the drug.²² In cases of failure to recover blood flow in the injured vessels, it is not well understood until now whether the tissue could be rescued or not. Generally, following a vascular occlusion event, the brain tissue could stay viable for hours. Therefore, restoration of sufficient vascular blood flow as soon as possible will protect these tissues and possibly maintain their viability.²³

Management

Proper rapid management is essential to determine later outcomes, as the brain tissue is potentially under threat of death within the first few hours following the onset of ischemia. In severe cases, the core of the infarction will inevitably die due to dense

ischemia. However, surrounding tissue can survive if proper management is done. In this case, hemodynamic stability, proper oxygenation, and metabolic factors are essential to determine the outcome. Initial management includes achieving stabilization, and determining possible factors that will lead to negative outcomes.²⁴ The next step is to assess the eligibility of the patient to thrombolysis therapy (we will discuss this point later in this paper).

Investigation

A CT scan of the brain must be initially done to all patients with an acute CVA to distinguish between the two types of a stroke (hemorrhagic or ischemic). It is crucial to know the type as subsequent management plans will depend on the type. Other benefits of imaging are to rule out other possible differential diagnosis and predict outcomes of the stroke. CT should be done immediately after the admission of the patient.²⁵ Brain MRI can replace CT because it has the following advantages: identifying stroke anatomy, assessing vascular blood flow, assessing brain tissue perfusion, distinguishing between old and new lesions, and identifying the presence/absence of carotid artery stenosis.²⁶ After imaging is done, physicians will determine the next step of evaluation based on imaging results, risk factors in the patient, the likelihood of recovery, and the age of the patient. Identifiable cause is more likely to be found in a younger patient, and this may be due to underlying inflammatory or clotting disorders or other diseases. Physicians are recommended to strictly follow guidelines when determining later investigations in each patient, and not to do any investigation that will not affect management of the patient.²⁷

Acute intervention

Two previous large clinical trials have found that aspirin (oral or rectal) will reduce the risk of later morbidity and mortality if started within the first 48 hours following the onset of ischemia. However, this effect is considered small and the number needed to treat is 77.²⁸ Before aspirin is administered, neuroimaging is highly recommended. Prior clinical trials did not find significantly improved outcomes when administering unfractionated heparin to stroke patients, even in cases of embolic strokes. Moreover, low-molecular-weight heparins have also been found to be ineffective in stroke patients. However, both unfractionated heparin and low-molecular-weight heparins can still be used in certain subgroups.²⁹ The use of thrombolytic therapy (alteplase recombinant) in the first three hours of onset of symptoms is proved to significantly improve outcomes and cause a complete (or nearly complete) recovery. Thrombolytic therapy within three to six hours of symptoms, on the other hand, has been found to be less effective. Generally, the use of thrombolytic therapy is still being studied, and several concerns are still being raised regarding its safety.³⁰

Complications after stroke

Complications of stroke are important factors that will affect later outcomes. Generally, the presence of fever, hyperglycemia, and/or hypertension is associated with poor outcomes. Therefore, high glucose levels should be controlled, and fever should be treated with paracetamol.³¹ However, it is not recommended to try to control blood pressure early following the stroke due to the possible worsening of outcomes that can be caused by anti-hypertensive drugs (especially calcium channel blockers). Complications of large strokes include swelling, edema, and herniation. These complications are generally fatal with no available approved treatment for them.³² About half of immobile stroke patients will develop deep venous thrombosis (DVT), pulmonary embolism, or another venous thromboembolic disease, if proper prophylaxis is administered. Compression stockings have been found to decrease the risk of developing a DVT in the general population, but this is not the case in stroke patients, and real effects of it are not well established. Generally, ischemic stroke patients are recommended to receive stockings, good hydration, aspirin, and early mobilization. In fact, early mobilization will further decrease the risk of other complications including respiratory infections, urinary tract infections, and bed sores. The use of urinary catheters is not recommended due to high rates of infection.^{33,34}

Rehabilitation

The main goal of undergoing rehabilitation programs is the restoration of function to the state prior to the stroke, and the reduction of stroke negative impact on both patients and caregivers. Rehabilitation is recommended to be started as soon as possible during the recovery phase, with properly assessing patients while still in the acute stroke unit. When patients become stable, immediate transfer to rehabilitation unit is recommended. Proper sufficient rehabilitation has been associated with significantly less risk of morbidity and mortality (number needed to treat is 12) along with shorter admission times. To establish proper rehabilitation, there should be good coordination between physicians, physiotherapists, speech therapists, psychologists, and nurses.³⁵

Stroke prevention

Other than proper blood pressure control, secondary prevention should be initiated as soon as possible following admission. Lifestyle and behavioral education should be offered to all patients. This includes recommendations of smoking cessation, salt intake reduction, alcohol cessation, weight loss, and exercise initiation. Aspirin should be prescribed as a long-term treatment to prevent further strokes.³⁶

CONCLUSION

I have seen in the review that the most critical part about approaching a stroke patient is to identify the type of stroke, whether hemorrhagic or ischemic, as each type requires a different guideline of management. Also, time is the key in preserving neuronal function and preventing further damage. At the same time, the general population must be educated about methods of preventing stroke by making positive lifestyle changes, and those who had a cerebrovascular accident, must be provided with proper rehabilitation therapy to maximize their quality of life.

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