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Current studies of cerebral infarction

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Abstract. Cerebral infarction, also known as stroke, is a serious medical condition resulting from the interruption of blood flow to the brain, either due to blockage or rupture of blood vessels. This interruption leads to localized ischemia and oxygen deprivation in the brain tissue, causing hypoxic-ischemic cell death and subsequent damage to brain function. Cerebral infarction is a leading cause of mortality and morbidity worldwide, particularly among the elderly, but recent research suggests that younger individuals are also increasingly affected. According to reports from the World Health Organization, cardiovascular diseases, including cerebral infarction, account for millions of deaths annually, posing a significant public health threat. The incidence of cerebral infarction among the younger generation is particularly noteworthy, with a significant proportion of patients aged 15-49 suffering from this condition. This trend underscores the need for greater awareness and prevention efforts across all age groups to address this global health challenge.

Keywords: Cerebral infarction, Symptoms, Thrombolytic injection

1. Introduction

Cerebral infarction, commonly known as a stroke, occurs when there is a blockage in the cerebral blood vessels, leading to localized ischemia and oxygen deprivation in the brain tissue. The obstruction is often caused by factors such as thrombus formation, the buildup of lipid plaques, or the rupture of aneurysms. These conditions impede blood flow, disrupting the supply of oxygen and nutrients to specific regions of the brain, resulting in hypoxic-ischemic cell death. Cerebral infarction is one of the most common and leading causes of mortality worldwide among cardiovascular diseases (CVD). CVD encompasses a range of diseases affecting the heart, brain, and body blood vessels, these diseases pose a significant public health threat due to their increasing incidence, imposing substantial burdens on both society and individuals [1]. According to reports from the World Health Organization, CVD claims the lives of approximately 18 million people annually, making it one of the primary global health challenges [2].

Cerebral infarction, commonly known as a stroke, occurs when blood flow to a part of the brain is interrupted, leading to tissue damage and impairment of brain function. This interruption is typically caused by a blockage or narrowing of the blood vessels supplying the brain (ischemic stroke) or by the rupture of a blood vessel in the brain (hemorrhagic stroke). Cerebral infarctions were once considered rare among young individuals [3] with a higher frequency observed among the aged. However, contemporary research indicates that young people are not uncommon among victims of cerebral infarctions. Thus, nowadays many younger generations are suffering from Cerebral infarction. In

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contrast to the elderly patients, there is a striking contrast among younger patients. According to the cohort study by CDC, from age 15-49, around 16% of people suffer from cerebral infarction. From age 50-69, the incident rate is approaching 22%. And with the elderly older than 70 years old, the incident rate is approximately 62%. The incidence of stroke is significantly higher between the vernal equinox and the autumnal equinox. Moreover, there is an increasingly serious trend of strokes occurring among younger individuals [4].

Cerebral infarction (cerebral infarction) typically occurs due to the obstruction of cerebral blood vessels, leading to localized ischemia and hypoxia, consequently resulting in damage to brain tissue. An infarct is an area of necrosis (tissue death) due to the blood vessel blockage. Common causes of cerebral infarction include thrombus formation, formation of lipid plaques, rupture of aneurysms, and others. These conditions obstruct blood flow, interrupting blood supply to a specific part of the brain, and causing hypoxic-ischemic death of brain cells [5]. Cerebral infarction can cause varying degrees of damage to specific parts of the brain, depending on the location and severity of the infarction. Mild cerebral infarction may only result in localized functional impairment, while moderate or severe infarction can lead to severe neurological deficits or permanent damage.

2. Symptoms

Symptoms of mild cerebral infarction may include mild headaches, mild neurological impairments (such as slight difficulty in speech or mild weakness in limbs), blurred vision, and others [5]. Symptoms of moderate cerebral infarction may be more pronounced, including severe headaches, difficulty in speech, limited limb movement, partial paralysis, and so on. Severe cerebral infarction may result in severe neurological deficits, such as complete paralysis, loss of speech ability, loss of consciousness, and others.

Currently, despite numerous studies on cerebral infarction, there is still no cure. Moreover, as a silent killer, early detection and timely medical treatment of cerebral infarction are particularly important. Furthermore, most middle-aged and elderly people suffering from this condition have a high risk of paralysis, and the prognosis of this disease is not optimistic. This not only torments the patients themselves but also imposes a significant burden on their families. Since immediate medical attention is crucial to minimize brain damage and prevent complications, studies for its mechanism and cure are significant to the world [6].

When interruption or reduction in blood supply to the brain tissue, it causes hypoxia (oxygen deprivation) in the affected brain region. Without oxygen, brain cells cannot produce adenosine triphosphate (ATP), the energy currency of cells. Energy failure results in the impairment of cellular functions. The energy failure disrupts cellular ion gradients, leading to an influx of calcium ions into neurons [7]. Elevated intracellular calcium levels activate enzymes like phospholipases and proteases, causing cellular damage and programmed cell death. Additionally, apoptosis contributes to neuronal loss in the ischemic penumbra, the region surrounding the core infarct. Moreover, hypoxia triggers the release of excitatory neurotransmitters, such as glutamate, leading to excessive stimulation of neuronal receptors. This excitotoxicity exacerbates cellular damage and initiates a cascade of events that contribute to neuronal death. Besides, hypoxia triggers an inflammatory response characterized by the activation of microglia and the release of pro-inflammatory cytokines. Inflammatory mediators further contribute to tissue damage and neuroinflammation. In such a situation, the blood-brain barrier (BBB) is disrupted, leading to increased vascular permeability and oedema formation. In turn, BBB breakdown allows inflammatory cells and neurotoxic substances to infiltrate the brain parenchyma, exacerbating injury [8]. These pathological processes collectively contribute to the progression of cerebral infarction and the associated neurological deficits.

3. Methods

Currently, there are many methods for treating cerebral infarction. However, there are 2 methods for first aid for cerebral infarction: injecting fibrinolytic drugs and removing the thrombus with mechanical instruments. To implement these two measures, patients must go to the hospital early in

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the “golden hour”, the time from the stroke to the time of receiving thrombolytic drugs is < 4.5 hours from the onset, and the time to Mechanical thrombectomy was < 6 hours from onset. Before performing the above emergency measures, it is necessary to take computed tomography of the brain and cerebral blood vessels of the patient to determine the condition of the brain damage and locate the blocked blood vessels in the brain.

Thrombolytic injection: Thrombolytic injection is performed by inserting an arterial catheter into the site of the thrombus and then injecting the fibrinolytic agent and/or removing the thrombus from the vessel. Many studies have shown that arterial fibrinolytic therapy increases the clinical recovery rate in acute ischemic stroke. Thrombolytic drugs, such as tissue plasminogen activator (tPA), are administered intravenously to dissolve blood clots obstructing cerebral arteries. This treatment is most effective when initiated within a few hours of symptom onset but carries a risk of bleeding complications.

Mechanical Thrombectomy: In cases of large vessel occlusion, mechanical thrombectomy may be performed to physically remove the clot from the cerebral artery using catheter-based devices. In mechanical thrombectomy, physicians use catheters to introduce micro-sized devices into the occluded cerebral blood vessels to directly and physically remove the thrombus. These devices may include various catheters, stents, and specially designed-thrombectomy instruments. Once the thrombus is successfully cleared, blood flow can be restored to the affected brain tissue, thereby reducing or preventing further neurological damage and brain tissue necrosis. Mechanical thrombectomy is typically performed in the early stages of acute stroke to minimize its consequences and improve patient survival and recovery rates. This procedure is often combined with thrombolytic therapy and has been shown to improve outcomes in selected patients.

Antiplatelet Agents: Medications like aspirin, clopidogrel, and dipyridamole are commonly prescribed to prevent platelet aggregation and reduce the risk of recurrent stroke. Antiplatelet agents are medications used to prevent platelet aggregation, which plays a key role in the formation of blood clots. They are commonly prescribed to reduce the risk of cardiovascular events, including stroke, heart attack, and peripheral artery disease. Medications such as aspirin, clopidogrel, and dipyridamole are examples of antiplatelet agents. Aspirin works by inhibiting the enzyme cyclooxygenase, thereby reducing the production of thromboxane A2, a potent platelet aggregator. Clopidogrel blocks the P2Y12 adenosine diphosphate (ADP) receptor on platelets, inhibiting their activation and aggregation. Dipyridamole works by inhibiting the breakdown of cyclic adenosine monophosphate (cAMP), leading to decreased platelet aggregation. These medications are often prescribed singly or in combination to individuals at risk of cardiovascular events, particularly those who have experienced a stroke or transient ischemic attack (TIA). Dual antiplatelet therapy, which combines two antiplatelet agents, may be recommended in some cases for enhanced efficacy, especially after certain cardiovascular procedures like coronary stenting. Dual antiplatelet therapy may be used in some cases for enhanced efficacy.

Anticoagulants: Anticoagulant drugs, such as warfarin or direct oral anticoagulants (DOACs), are indicated for patients with atrial fibrillation or other cardiac conditions predisposing to thromboembolism. Warfarin is a traditional anticoagulant that works by interfering with the synthesis of clotting factors to prevent blood clotting, but it requires close monitoring of the International Normalized Ratio (INR) to ensure appropriate dosage. In contrast, Direct Oral Anticoagulants (DOACs) are a relatively new class of anticoagulant drugs with a more direct mechanism of action, stable dosing, and do not require frequent monitoring. The use of these medications can effectively prevent the formation of blood clots in the heart, thereby reducing the risk of serious complications such as stroke and improving the quality of life for patients [8]. These medications help prevent the formation of blood clots in the heart and reduce the risk of cerebral infarction.

4. Side Effects of Therapies

The treatments for cerebral infarction, while beneficial, can also carry certain side effects. Here are some potential side effects associated with each treatment: Thrombolytic Therapy (tPA): Risk of

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bleeding, including intracranial haemorrhage, particularly if administered outside the recommended time window or in patients with increased bleeding risk. Since tPA works by breaking down blood clots, it can increase the risk of bleeding, particularly in the brain (intracranial haemorrhage) or other organs. Therefore, patients receiving thrombolytic therapy must be carefully selected based on strict criteria to minimize this risk. Other potential side effects of thrombolytic therapy may include allergic reactions, such as hives or difficulty breathing, as well as nausea, vomiting, and fever. Sometimes, patients may experience a transient drop in blood pressure or an irregular heart rhythm (arrhythmia). Allergic reactions to the thrombolytic agent.

Mechanical Thrombectomy: Risk of vessel injury: During the procedure, there is a risk of injuring the blood vessel being treated. This could lead to vessel dissection (tearing of the vessel wall) or perforation (creating a hole in the vessel). These complications can cause bleeding and may require additional medical intervention to repair [9].

Formation of new blood clots: Despite removing the initial clot causing the stroke, there is a possibility of new blood clots forming either during or after the procedure. These new clots could potentially cause another stroke or other complications if they travel to other parts of the brain or body.

Complications related to anesthesia: Mechanical thrombectomy is typically performed under general anesthesia or conscious sedation to keep the patient comfortable and still during the procedure. However, anesthesia carries its own set of risks, including respiratory problems, allergic reactions, and cardiovascular complications.

Complications related to catheterization: The procedure involves threading a catheter through the blood vessels to reach the site of the clot in the brain. While this is generally safe, there is a risk of complications such as bleeding at the catheter insertion site, damage to blood vessels, or allergic reactions to contrast dye used during imaging.

Antiplatelet Agents: Increased risk of bleeding, particularly gastrointestinal bleeding or bruising. Gastrointestinal upset, including nausea, indigestion, or diarrhea. Allergic reactions, although rare.

Anticoagulants: Risk of bleeding, including intracranial hemorrhage, especially with high doses or in patients with underlying bleeding disorders.

Drug interactions with other medications. Skin reactions or allergic responses to the medication. Cerebral infarction is a complex and irreversible brain injury that requires new treatment approaches to improve patient survival rates and prevent paralysis or death. In recent years, new technologies such as nanomedicine have provided hope for the treatment of cerebral infarction [10]. However, the development of treatment methods still faces challenges.

5. Conclusion

The process of brain injury is complex and irreversible, necessitating the development of new treatments for cerebral infarction to improve patient survival rates and prevent progression to paralysis or death. In recent years, there have been promising prospects in the application of cerebral infarction treatments. The latest research includes relatively ideal nanomedicines, whose carriers possess characteristics such as long circulation time in vivo, immune evasion, targeted delivery, and targeted release. These properties hold the potential to become beneficial technologies for the treatment of cerebral infarction, expanding their value in the treatment of cardiovascular diseases. However, the development process poses significant challenges.

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