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Herbal Drugs used in the Treatment of Stroke

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ABSTRACT: Stroke is one of the most important causes of mortality and morbidity in the world. Prevention and effective treatment of stroke is of the utmost importance. Cerebral ischemia causes disturbances in a variety of cellular and molecular mechanisms, including oxidative phosphorylation, membrane function, neurotransmitter release, and free radical generation. Thrombolytic therapy is the most effective therapeutic strategy for the prevention of brain injury and reduction of mortality in patients with cerebral infarction. However, a combination of established thrombolytic therapy and effective neuronal protection therapy may have more beneficial effects for patients with cerebral infarction. Because clinical trials of pharmacological neuroprotective strategies in stroke have been disappointing, attention has turned towards approaches that include herbal drugs that can be used in limiting the neurological damage associated with stroke. Herbal drugs may be used as a prophylactic treatment in patients with a high risk of stroke. This review focuses on putative mechanisms underlying the beneficial effects of herbal drugs in patients with stroke and on the possibility of herbal drugs increasing the therapeutic time window in patients with cerebral ischemia.

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INTRODUCTION:

A stroke is the sudden onset of weakness, numbness, paralysis, slurred speech, aphasia, problems with vision and other manifestations of a sudden interruption of blood flow to a particular area of the brain. The ischemic area involved determines the type of focal deficit that is seen in the patient [1,2].

A Transient Ischemic Attack (TIA) is similar to a stroke, but the interruption of blood flow is temporary. The clot resolves sporadically. The symptoms are relatively the same as a stroke but last less than 24 hours, whereas stroke symptoms persist for greater than 24 h [17].

The primary pathophysiology of stoke is an underlying heart or blood vessel disease. The secondary manifestations in the brain are the result of one or more of these underlying diseases or risk factors. The primary

pathologies include hypertension, atherosclerosis leading to coronary artery disease, dyslipidaemia, heart disease, and hyperlipidaemia. The two types of strokes that result from these disease states are ischemic and haemorrhagic strokes [1-3]. The objective of the review is to have a thorough study on stokes.

Non-reducible Risk Factors:

The possibilities of a stroke occurring increases with age. For every decade (10 years) over the age of 55, the possibility of a stroke occurring doubles. A patient that is 75 years of age has four times the risk of having a stroke compared to someone who is 55 years old. Of all strokes that occur in people, approximately 65% occur in those who are over the age of 65. Those who have had a stroke or TIA are more likely to have another stroke or transient ischemic attack. Approximately 60% of strokes occur in patients who have had a previous TIA. Strokes generally occur more often in males than females, until the age of 55; after age 55 the risk is the same for both men and women. The occurrence of stroke is higher in the African-American, Hispanic, and Asian-Pacific Islander population than in other ethical backgrounds. Patients who have immediate family members (mother, father, or sibling) that have had a stroke or TIA are at greater risk for having a stroke or TIA than those who do not have a family history with these events. People who have diabetes are also at greater risk of stroke that those without diabetes.

Reducible Risk Factors:

Lower your high blood pressure. Hypertension (high blood pressure) is the number one most treatable risk factor for stroke. You can help prevent a TIA or stroke considerably by working to lower your blood pressure. Lowering cholesterol levels may decrease the risk of stroke. By working to lower your cholesterol, you can help prevent a TIA or stroke. Stop smoking. If you stop smoking, you can decrease your risk for stroke to that of a non-smoker within two to five years.

Common Symptoms:

These symptoms usually occur suddenly without warning. Most patients are going about their normal daily activities and suddenly notice:

- A weakness or numbness in the face, arm, or leg.
- A change in the vision of one or both eyes that occur suddenly with no known cause.
- A severe sudden headache that cannot be explained by any injury or other cause.
- A quick onset of dizziness, loss of coordination/balance, or other problems walking.
- A sudden problem talking or expressing thoughts and words.

These are the most common signs and symptoms to be aware of and reasons to seek immediate medical attention. The symptoms most often affect only one side of the body but may affect both sides. If you see someone or you have any of these or other symptoms seek immediate medical attention – do not wait to see if it goes away.

Other Symptoms

A sudden loss of consciousness or moments of fainting or convulsions (seizures) without any known cause. Nausea, vomiting, or fever that occurs suddenly (within minutes or hours) that cannot be explained by any other cause betes may also help to decrease your risk of stroke.

Treatment [1,2]:

Thrombolytic Therapy:

Thrombolytic therapy is the use of drugs to break up the clot that is causing the disruption in blood flow to the brain. It is imperative that you immediately go to the hospital when you first notice the warning signs of a stroke. The length of time between the first warning signs and the time you get to a hospital may directly affect your recovery. Patients who present to the hospital within 3 hours of the first sign of a stroke have the possibility to receive alteplase (Activase®). Activase® is a clot-buster. It breaks-up the clot to restore blood flow to the area of the stroke. There are many factors that determine whether or not a patient is able to receive thrombolytic therapy. One of these factors is the amount of time between the onset of symptoms and presentation to the hospital. If you get to the hospital within the 3 h time frame and the doctor determines you are able to receive this clot-buster, you may have a better recovery.

Antiplatelet Agents – Mild Blood Thinners:

Platelets are blood cells that are help the blood clot (stick together) and prevent bleeding. When the body has a cut, scratch, bruise, or bleed, platelets go into action and begin to work. They can be thought of as materials (like bricks or blocks) that aggregate (link together/ stack up) to form this clot. These platelet cells need thromoxane A2, adenosine, vitamin K specific clotting factors (chemicals produced by the body) to make them aggregate (stick) together. These chemicals can be thought of as the glue that holds the blocks

together to make the clot. However, in patients who have had a TIA or stroke, the blocks don't need to stick together as much because this causes the blood to be too thick (like adding flour to milk when making a cake batter it makes it thicker and harder to stir or pour) and possibly form a clot that can't fit through the vessels. So, doctors often place stroke/TIA patients on blood thinners to decrease the possibility of the body forming another clot in the blood, which may lead to another TIA or stroke.

Aspirin:

Aspirin is used for prophylaxis of TIA and/or stroke except in patients with an allergy to aspirin or salicylates. The mechanism of action for aspirin's stroke prevention is the inhibition of prostaglandin synthesis action to prevent the formation of platelet-aggregating substance thromboxane A2.The usual dose for this indication in adults is 50 to 325 mg/day. Aspirin should be taken with food, milk, or large glass of water to decrease GI problems. Monitor for signs of bleeding.

Aspirin and Dipyridamole – Aggrenox®:

Aggrenox® is used to reduce the risk of stroke in patients who have had a TIA or completed ischemic stroke due to thrombosis, except in patients with hypersensitivity to dipyridamole or aspirin. The mechanism for its antithrombotic action is the additive antiplatelet effect of the two drugs. The aspirin portion works the inhibition of prostaglandin synthesis action to prevent the formation of platelet-aggregating substance thromboxane A2, while the dipyridamole inhibits adenosine uptake into erythrocytes, endothelial cells, and platelets.

One capsule (aspirin 25mg and dipyridamole 200mg) twice a day is the usual dose in adults. The capsule should be swallowed whole (not chewed or crushed), and can be taken with or without food. Most patients experience a severe headache when initiating therapy due to the vasodilatation of dipyridamole. The headache should ease and resolve after the body adjusts to the treatment. In the meantime, acetaminophen (Tylenol) is the treatment of choice for the headache.

Clopidogrel – Plavix®:

Clopidogrel is used to reduce future atherosclerotic events (stroke) in patients with a recent stroke. The drug's mechanism is it blocks the adenosine phosphate (ADP) receptors, which prevents fibrinogen binding to the receptor. This decreases the ability of platelet adhesion and aggregation. The usual dose for stroke prevention is 75mg once a day, and can be taken without food. It may be used as an alternative to aspirin containing products in patients allergic to aspirin or salicylates.

Clopidogrel and Aspirin:

The combination of Clopidogrel and aspirin is used to reduce future atherosclerotic events (stroke) in patients with a recent stroke or patients who had a stroke while on Clopidogrel. The mechanism of action for each drug is different. Clopidogrel blocks the adenosine phosphate (ADP) receptors, which prevents fibrinogen binding to the receptor, while aspirin inhibits prostaglandin synthesis action to prevent the formation of platelet-aggregating substance thromboxane A2. The usual dose is clopidogrel 75mg tablet and an additional aspirin 325mg tablet a day. Patients may need to take the medications with food, milk, or a full glass of water to decrease GI problems. Do not dispense in aspirin/salicylate allergic patients.

Ticlopidine – Ticlid®:

Ticlopidine is used in patients to decrease the risk of stroke or the occurrence of another stroke. However, due to its life-threatening rheumatologic disorders, it should be reserved for patient's refractory to aspirin or allergic to aspirin. The mechanism of action is unique among the antiplatelet drugs because it specifically increases bleeding time. The usual dose is 250mg twice a day. It should be taken with food to decrease stomach upset. Starting the second week of therapy and through the third month of therapy, patients will need a complete blood count with differential every two weeks. The peak occurrence of thrombocytopenia (TTP) is 3 to 4 weeks after starting the medicine, with peak occurrences of neutropenia at 4 to 6 weeks, and aplastic anaemia incidences after 4 to 8 weeks.

Oral Anticoagulant – Stronger Blood Thinners:

***Warfarin – Coumadin*:**

Warfarin is used for the treatment and prophylaxis of pulmonary embolism, venous thrombosis, and thromboembolic disorders, and to prevent recurrences of TIA's. In stroke patients, warfarin is most often used to prevent a cardiogenic embolism due to atrial fibrillation. The usual therapy for the prevention of a cardio embolic TIA or stroke in patient with atrial fibrillation is long term oral anticoagulation with a target international normalized ratio (INR) of 2.5 (range of 2.0-3.0)

Warfarin's mechanism of action is interference with hepatic synthesis of vitamin K-dependent coagulation factors (II, VII, IX, and X). Foods high in vitamin K inhibit the effects of warfarin. Once patient is stabilized on warfarin, the patient should not change dietary habits. The patient needs to maintain a consistent amount of vitamin K (70-140mcg/day) in their diet. Foods that are high in vitamin K include: leafy green vegetables, pork and beef liver, and green teas. Patients should be instructed to avoid large amounts of alfalfa, broccoli, asparagus, Brussels sprouts, cauliflower, cabbage, kale, spinach, watercress, lettuce, and turnip greens, and to consult their pharmacist or doctor who monitors their warfarin therapy. Patients beginning warfarin will need to have weekly laboratory test done to evaluate and stabilize their therapy.

***Prevalence*:**

According to the World Health Organization, 15 million people suffer stroke worldwide each year. Of these, 5 million die and another 5 million are permanently disabled. High blood pressure contributes to more than 12.7 million strokes worldwide. In India 1.2 billion people suffering from stroke per year. Europe averages approximately 650,000 stroke deaths each year. In developed countries, the incidence of stroke is declining, largely due to efforts to lower blood pressure and reduce smoking. However, the overall rate of stroke remains high due to the aging of the population.

***Social burden*:**

A costly disease for individual, family and societal perspective. About half of stroke survivors have physical or cognitive impairment and therefore partial or complete dependency. Chronic disease, required long term therapy.

Benefits of Herbal medicine:

A fewer side effects as compared to allopathic medicine. Safer used for longer period of time as compared to allopathic medicine. Excellent prospective for treatment of ischemic stroke. Permanent cure of disease, but in allopathic medicine disease is not cure permanently and provide only instant relief to the patient Natural healing. Cost effective. More affordable and conventional medicine. Do not damage immune system but allopathic medicine may damage the immune system.

Rational

The allopathic drugs used for stroke treatment prevent or cure the stroke condition but it produces side effects. This chemical drug leads to psychological dependence and adverse behavioural effects. Therefore, herbal drugs used over allopathic drugs because it shows very less side effects as compare to allopathic medication**.**

The objective of the present review study is related to search herbal medicines and classify them according to its effect on modifying stroke behaviour that are effect on against the depression, effect on anxiety, and effect on High blood pressure and to identify the traditionally used herbal medicines in society which lacks any scientific claims.

HERBAL DRUGS FOR STROKE:

Punarnava:

***Botanical Name*:**

Its botanical name is *Boerhaavia diffusa*.

***Biological source*:**

It is obtained from whole herb/root of *Boerhavia diffusa* linn. belonging to family *Nyctaginaceae.*

***Chemical Constituents*:**

It consists of chemicals like b-sitosterol, a-2-sitosterol, palmitic acid, ester of b-sitosterol, tetracosanoic, hexacosonoic, stearic, arachidic acid, urosilic acid, hentriacontane, b-Ecdysone, and tricontanol.

***Pharmacological use*:**

Pharmacologically it shows immunomodulatory effects, immunosuppressive activity, hepatoprotective, analgesic, anti-inflammatory, and antioxidant activities.

***Use in stroke*:**

It is nerve rejuvenator and it is given in case of sciatica or nervous weakness or even paralysis condition. [15]

***Animal/human studies*:**

In 3-nitropropionic acid (NPA), sodium nitroprusside (SNP) induced oxidative stress in rat brain homogenates BDE treatment with dose significantly decreased the production of TBARS and increased the activities of antioxidant enzymes like catalase and superoxide dismutase along with increased concentration of non-enzymatic antioxidant, reduced glutathione (GSH). Similarly, BDE caused a significant decrease in the lipid peroxidation (LPO) in the cerebral cortex. Inhibitory potential of BDE against deoxyribose degradation shows that BDE can protect hydroxyl radical induced DNA damage in the tissues [18].

Bramhi:

***Botanical name*:**

Its botanical name is *Bacopa monnieri*.

***Biological source*:**
It consists of fresh leaves and the stem of the plant known as *Bacopa moniera* Linn. of the family *Scrophulariaceae*.

***Chemical constituents*:**
The best-characterized compounds in *Bacopa monnieri* are dammarane-type triterpenoid saponins known as bacosides, with jujubogenin or pseudo-jujubogenin moieties as aglycone units. Bacosides comprise a family of known analogs.
Other saponins called bacopasides I–XII have been identified more recently. The alkaloids brahmine, nicotine, and herpestine have been catalogued, along with D-mannitol, apigenin, hersaponin, monnierasides I–III, cucurbitacin, and plantainoside B.

***Pharmacological uses*:**
It was used as a brain tonic to enhance memory development, learning and to provide relief to patients with anxiety.

Use in Stroke:
Brahmi has been used in the Ayurvedic system of medicine for centuries. Traditionally, it was used as a brain tonic to enhance memory development, learning and to provide relief to patients with anxiety or epileptic disorders.
The bacosides aid in the repair of damaged neurons by enhancing kinase activity, neuronal synthesis, and restoration of synaptic activity, and ultimately nerve impulse transmission [16].

***Animal/human studies*:**
Bacopaside I treatment produced a significant reduction in neurological deficits at 22 and 70 h, and significantly reduced cerebral infarct volume and edema at 70 h, when compared with the ischemia group. Animals, that were orally treated with bacopaside I showed increased brain ATP content, energy charge (EC), total adenine nucleotides (TAN), nitric oxide (NO) level, $Na^{+}K^{+}$ATPase, and $Ca^{2+}Mg^{2+}$ATPase activity. Bacopaside I treatment also improved antioxidant enzyme activities including brain superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px), in varying degrees, compared with the ischemia group.
In addition, three doses of bacopaside I markedly inhibited the increase in MDA content in the brain [19].

Jatamansi:

***Botanical name*:**
The botanical name of Jatamansi is *Nardostachys jatamansi.*

***Biological source*:**
It consists of dried rhizomes, stolons, and roots of *Valeriana wallichii* belonging to the family *Caprifoliaceae*.

***Chemical constituents*:**
Alpha-patchoulenese, angelicin, beta-eudesemol, beta-patchoulenese, betasitosterol, calarene, calarenol, elemol, jatamansin, jatamansinol, jatamansone, n-hexacosane, n-hexacosanol, nardol, nardostechone, norsechelanone, oroselol, patchouli alcohol, seychelane, seychellene, valeranal, valeranone.

***Pharmacological uses*:**
Hepatoprotective activity, Antidepressant activity, Anticonvulsant activity, Antioxidant and stress relieving activity.

***Use in stroke*:**
Alcoholic and N-hexane extracts of jatamansi prevent lipid peroxidation and it is beneficial in stroke as it suppresses oxicalosive stress [4].

***Animal/human studies*:**
The anti-stress effect of the hydro-ethanolic extract of *N. jatamansi* was evaluated in reference to its antioxidant property. Wistar rats were divided into four groups naïve, stressed, T-200 and T-500 stressed with oral pre-treatment of *N. jatamansi* extract, Restraint of rats on metallic chambers for 4 h at 4°C was followed by sacrifice and assessment of stress-induced alterations in biochemical parameters, incidence and severity of ulcers. The *In-vitro* antioxidant activity of *N. jatamansi* was studied by measuring the free radical scavenging activity. *N. jatamansi* showed potent antioxidant activity and significantly reversed the stress-induced elevation of LPO and NO levels and decrease in catalase activity in the brain. *N. jatamansi* possesses significant anti-stress activity, which may be due to its antioxidant activity [20].

Garlic:

***Botanical name*:**
Its botanical name is *Allium sativum*.

***Biological source*:**
Lehsun consists of the fresh compound bulb of *Allium*

sativum Linn belonging to the family *Liliaceae*.

***Chemical constituents*:**
Alliin is a sulfur-containing amino acid. Allicin is allyl sulfide. Polysulphide is responsible for the unpleasant smell of the oil. The amino acid present in garlic is Leucine, methionine, S-methyl cysteine, and S-allyl cysteine. Allyl propyl disulfide. Vitamins present in garlic are Vitamin A, B, C, and D. Other constituents present in the garlic are Fatty acid, mucilage, and albumin.

***Use in stroke*:**
The neuroprotective effect of garlic is associated with the control of free radical bursts induced by reperfusion preservation of the antioxidant enzyme, diallyldisulphate an active principal of garlic [4,5].

***Animal/human studies*:**
Reviewed human trials which were conducted since 1993. Only those trials which were conducted for a minimum period of two weeks and that addressed the following parameters had been included: (a) cholesterol-lowering effects, (b) inhibition of platelet aggregation (c) lowering of blood pressure, and (d) other cardioprotective properties. They reported that since 1993, 44 % of the clinical trials have indicated a reduction in total cholesterol and all seven clinical trials on the inhibition of platelet aggregation showed positive responses in both healthy subjects and subjects with CVD [21].

Turmeric:

***Botanical name*:**
Its botanical name is *Curcuma longa*.

***Biological source*:**
Turmeric consists of the dried rhizomes of *Curcuma longa* belonging to family *Zingiberaceae.*

***Chemical constituents*:**
Turmeric includes diarylheptanoids, a class including numerous curcuminoids, such as curcumin, demethoxycurcumin, and bisdemethoxycurcumin. Curcumin constitutes up to 3.14 % of assayed commercial samples of turmeric powder (the average was 1.51%); curry powder contains much less (an average of 0.29 %). The essential oils are present in turmeric, among which turmerone, germacrone, atlantone, and zingiberene are major constituents.

***Use in stroke*:**
Curcumin possesses multiple pharmacological properties (anti-inflammatory, anti-thrombotic, and anti-oxidative) and these properties further add to its anti-ischemic property. The anti-ischemic effect of curcumin is believed to be contributed by its free radical scavenging activity which is unique in having phenolic and diketonic groups present in its structure. The neuroprotective effect of curcumin is well documented over different neurotoxicants. These protective effects not only rescue the metabolite alterations but also improve brain edema, Evans Blue leakage, and infarct size during ischemic brain injury (stroke) [4,5].

***Animal/human studies*:**
The pre-treatment with curcumin or 30 min post-treatment significantly reduced brain water content and improved neurological outcomes following a moderately controlled cortical impact in mice. The protective effect of curcumin was associated with a significant attenuation in the acute pericontusional expression of interleukin-1β, a pro-inflammatory cytokine, after injury [22].

***Animal/human studies*:**
Nicotine evokes improvement in learning and memory mediated through NPYY1 receptors in AD-like conditions induced by colchicines in rats. In this study the cognitive functions were assessed by Morri's water maze task and on acute nicotinic administration, dose-dependent improvement in learning and memory in colchicines-treated rats was observed. Nicotine decreased escape latency and increased the time spent in the target quadrant as compared to the saline-treated rats [23].

CONCLUSION:

Herbal compounds have vast therapeutic potential and are used as a treatment measure for stroke. The review provides a compilation of herbal plants which are used for the treatment of stroke.

It also provides some plants which are used for the prevention of stroke and used for the treatment of common post-stroke symptoms. Because availability, lower cost, and fewer adverse effects of herbal compounds in comparison to synthetic ones make they an excellent choice in treating stroke. Further studies on investigating the mechanism of action and drug

development for stroke from such herbs may help in the therapeutic management of this chronic, morbid, and debilitating disease.

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