

Utilization of Antiplatelet in Ischemic Stroke

Abstract

Objective: A review of the literature on antiplatelet medications for the prevention of primary and secondary strokes, including their mechanism of action, price, and reasons for their lack of benefit. Sources of data: The databases MEDLINE, Cochrane Reviews, and PubMed were used to gather the articles (1980-2021). We looked at abstracts from scientific meetings. Ischemic stroke, aspirin, clopidogrel, dipyridamole, ticagrelor, cilostazol, prasugrel, and glycoprotein IIb/IIIa inhibitors were among the search terms.

Selection of a study and data collection: Both original and review articles written in English were evaluated. Multiple nations' guidelines were looked at. Two authors independently evaluated the articles. Synthesis of data: There is a lot of evidence that aspirin and clopidogrel can be used to prevent secondary strokes. These medications work better together to prevent future strokes in the acute phase (the first 21 days after the initial stroke), but long-term combination therapy is linked to higher rates of bleeding. Failure of antiplatelet therapy is influenced by genetic polymorphisms and poor adherence. In some racial groups, antiplatelet agents like cilostazol may be better than clopidogrel and aspirin, but more research is needed on more diverse ethnic groups. Relevance to clinical practice and patient care: The available data on the use of various antiplatelet medications after stroke are presented in this review. Topics for future research include dual therapy, recurrence following the start of secondary preventative therapy, and these topics.

Conclusions: Even though there is a lot of evidence to support the use of certain antiplatelet medications after a stroke, personalized therapies still have a lot of room for improvement. Screening patients for platelet polymorphisms that cause antiplatelet resistance and incorporating more racially diverse populations into randomized trials are two examples of these.

Keywords: Stroke • Antiplatelets • Aspirin • Clopidogrel • Cilostazol • Prasugrel • Ticagrelor • Ticlopidine

Introduction

Ischemic stroke is, by far, the most common cause of stroke worldwide, accounting for 10 times more strokes than hemorrhagic strokes in higher income countries, but with much less difference observed in lower income countries. Although the rate of stroke deaths is decreasing, it is believed that up to 50% of stroke-related deaths are attributable to poorly managed modifiable risk factors.¹ Over the course of the past 25 years, there has been a global reduction in the rate of death and age adjusted. There is a lot of evidence that treating hypertension, hypercholesterolemia, diabetes, smoking, and cardiac arrhythmias like atrial fibrillation can reduce the number of strokes that occur and recur [1].

We have compiled a summary of the evidence for each antiplatelet medication used to treat ischemic stroke in this review article. When confronted with recurrent stroke, we have then provided recommendations for alternative native therapeutic options. In the final section, we discuss the potential developments in antiplatelet therapies for ischemic stroke.

Discussion

Aspirin

Aspirin is an essential World Health Organization (WHO) medication that is generally well

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tolerated and inexpensive. It is also used in the acute phase and in secondary prevention of ischemia-related stroke, either alone or in combination therapy with other antiplatelet agents.¹² Aspirin is the antiplatelet agent that has received the most research attention. Aspirin exerts its effects by irreversibly inhibiting cyclo-oxygenase (COX), which inhibits the synthesis of the procoagulant thromboxane A₂ (TXA₂) and reduces platelet aggregation. The effectiveness of aspirin in the acute phase of ischemic stroke has been demonstrated by numerous studies.⁷ For a period of two to four weeks, a meta-analysis of 40 000 patients from the International Stroke Trial (IST) and the Chinese Acute Stroke Trial (CAST) compared aspirin (160-300 mg) to either a placebo or no medication [2, 3].

Clopidogrel

Clopidogrel is an antiplatelet drug of the second generation made from thienopyridines. The hepatic cytochrome P450 system metabolizes it into its active form from a prodrug. The active metabolite prevents ADP-mediated activation of the downstream glycoprotein IIb/IIIa complex, reducing platelet aggregation, by acting as an irreversible inhibitor of the P2Y₁₂ class of Adenosine Diphosphate (ADP) receptors on the surface of platelets. Hereditary polymorphisms in the proteins associated with clopidogrel digestion add to variety in light of clopidogrel between individuals. Clopidogrel is authorized for the administration of ischemic stroke in both the intense stage assuming patients are known to be ibuprofen unfavorably susceptible and as long haul optional anticipation [4, 5].

Cilostazol

Because it is a selective inhibitor of phosphodiesterase 3, cilostazol prevents platelet aggregation by increasing the activation of intracellular cAMP. It is assumed that cilostazol has a weak antiplatelet effect in the acute stages of stroke treatment, but the combined vasodilatation and antiplatelet effect is thought to be the underlying mechanism leading to long-term stroke prevention. In addition to its action on platelet aggregation, cilostazol also dilates blood vessels [6, 7].

Antiplatelet Therapy Associated Stroke

The guidelines recommend aspirin, either

alone or in combination with dipyridamole or clopidogrel, for noncardioembolic stroke and TIA. However, in the clinical trials, no antiplatelet agent was 100% effective in preventing recurrent cerebrovascular events. Besides, the peculiarity of antiplatelet opposition has been well described. While a meta-investigation of observational examinations found proof for changing to a completely new antiplatelet blend after an intermittent occasion, with a decreased incidence of cardiovascular occasions on follow-up, there are no randomized controlled preliminaries to direct clinicians [8, 9].

Conclusion

One of the secondary preventive treatments for stroke that has received the most research and is still one of the most effective is antiplatelet agents. The majority of clinical guidelines worldwide are based on the well-established use of clopidogrel and aspirin in both acute and secondary prevention settings. Even though there is already a lot of evidence to support the use of antiplatelets after a stroke, more research may focus on how to personalize the prescription process. Screening patients for platelet polymorphisms that confer antiplatelet resistance or expanding the level of evidence from randomized controlled trials to be more encompassing all of the various racial groups afflicted by stroke [10].

REFERENCES

1. Lauria G, Gentile M, Fassetta G *et al.* Incidence and prognosis of stroke in the Belluno Province, Italy First year results of a community-based study. *Stroke*. 26: 1787- 1793 (1995).
2. Andersen Klaus K, Olsen Tom S, Dehlendorff C *et al.* Hemorrhagic and ischemic strokes compared. *Stroke*. 40: 2068-2072 (2009).
3. Topçuoğlu MA, Arsava EM, Ay H Antiplatelet resistance in stroke. *Expert Rev Neurother*. 11: 251-263 (2011).
4. Ellekjaer H, Holmen J, Indredavik B *et al.* Epidemiology of stroke in Innherred, Norway, 1994 to 1996 Incidence and 30-day case-fatality rate. *Stroke*. 28: 2180- 2184 (1997).
5. Smadja D, Cabre P, May F *et al.* ERMANICA: Epidemiology of stroke in Martinique, French West Indies Part I: Methodology, incidence, and 30-day case fatality rate. *Stroke*. 32: 274- 2747 (2001).
6. Martin CP, Talbert RL Aspirin resistance: an

- evaluation of current evidence and easurement methods. *Pharmacotherapy*. 25: 942- 953 (2005).
7. Harker LA, Kadatz RA Mechanism of action of dipyridamole. *Thromb Res Suppl*. 4: 39- 46 (1983).
 8. Sacco RL, Diener HC, Yusuf S Aspirin and extended release dipyridamole versus clopidogrel for recurrent stroke. *N Engl J Med*. 359:1238-1251 (2008).
 9. Halkes PH, Gray LJ, Bath PM Dipyridamole plus aspirin versus aspirin alone in secondary prevention after TIA or stroke: a meta-analysis by risk. *J Neurol Neurosurg Psychiatry*. 79: 1218-1223 (2008).
 10. Goto S Cilostazol: potential mechanism of action for antithrombotic effects accompanied by a low rate of bleeding. *Atheroscler Suppl*. 6: 3-11(2005).