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Management of Acute Coronary Syndrome

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Abstract

Acute coronary syndrome is a common cause of acute heart failure, and having both illnesses increases the chance of short-term death compared to having just the acute coronary syndrome. Acute coronary syndrome might be difficult to diagnose in patients with accute heart failure Electrocardiograms can be muddled by pre-existing abnormalities, and cardiac biomarkers are typically increased in individuals with chronic or acute heart failure, even if they do not have an acute coronary syndrome. In individuals with acute heart failure, it's critical to differentiate between temporary or limited myocardial injury and primary myocardial infarction caused by vascular events. This paper presents many clinical situations to aid in the diagnosis of acute coronary syndrome as a cause of acute heart failure and attempts to offer doctors with tools to aid in the differentiation between these illnesses. The interpretations of ECG and biomarker findings, as well as imaging approaches that may aid in the diagnosis process, are discussed. Regardless of electrocardiographic or biomarker data, guidelines advocate a rapid invasive strategy for patients with acute heart failure and acute coronary syndrome. Patients with acute coronary syndrome and acute heart failure should be managed pharmacologically according to guidelines specific to each of these syndromes, with priority given to time-sensitive treatments in both cases. To better define the care of patients with a combination of acute coronary syndrome and acute heart failure, more research on these patients is needed.

Keywords: Acute Coronary Syndrome; Heart Failure; Electrocardiograms

Introduction

Despite normal medical therapy, such as long-term antiplatelet medication with aspirin and an adenosine diphosphate–receptor inhibitor, patients with an acute coronary syndrome are nonetheless at risk for recurrent cardiovascular events. This risk could be linked to extra thrombin production in these patients that lasts beyond the acute stage. As a result, researchers have started looking at the effect of oral anticoagulants in the aftermath of an acute coronary syndrome [1]. Patients who were treated with the anticoagulant warfarin in addition to aspirin had better cardiovascular outcomes. However, difficulties with drug administration and the risk of bleeding have limited the use of long-term warfarin in these individuals.

Similarly, following a myocardial infarction, treatment with the factor IIa inhibitor ximelagatran improved cardiovascular outcomes; however the drug was linked to hepatotoxicity. Rivaroxaban is an anticoagulant that works by inhibiting factor Xa directly and specifically. Factor Xa initiates the coagulation cascade's last common pathway, which leads to the synthesis of thrombin, which catalyzes other coagulation-related processes and increases platelet activation [2].

The study enrolled 3491 patients with a recent acute coronary syndrome in a phase 2 dose-finding trial. Rivaroxaban was tested at total daily doses ranging from 5 mg to 20 mg and reduced the composite end point of death, myocardial infarction, or stroke with the lowest hazard ratios seen at the lowest twice-daily doses, whereas there was a dose-dependent increase in bleeding events when compared to placebo [3]. Based on these findings, we established ATLAS ACS 2–TIMI 51, a phase 3 study to assess twice-daily rivaroxaban at doses of 2.5 mg and 5 mg as supplementary therapy in patients with a recent acute coronary syndrome, with the goal of identifying a clinically viable low-dose regimen.

Recent Acute Coronary Syndrome

Despite medical treatment, people with an acute coronary syndrome are nevertheless at risk of recurrent cardiovascular attacks. In individuals with a recent acute coronary syndrome, rivaroxaban significantly reduced the primary efficacy end point of death from cardiovascular causes, myocardial infarction, or stroke. Individual components of death from cardiovascular causes and myocardial infarction showed a directionally consistent benefit, whereas stroke did not. The benefits of adding rivaroxaban were seen regardless of whether patients had a STEMI, NSTEMI, or unstable angina, as well as across different geographical regions [4].

Similarly, both rivaroxaban doses significantly lowered the primary efficacy end point, with the twice-daily 2.5 mg dose demonstrating a survival advantage as well. In terms of safety, rivaroxaban at two doses increased the incidence of severe bleeding and cerebral haemorrhage when compared to placebo, but not by a substantial amount. Rivaroxaban at a lower dose caused less bleeding than at a larger dose.

Parenteral anticoagulants are used in concert with antiplatelet medicines during the early therapy of an acute coronary syndrome. Antiplatelet medicines have, nevertheless, been the cornerstone of antithrombotic therapy following hospital discharge [5]. Although secondary prophylaxis with oral anticoagulation has demonstrated to be beneficial to the cardiovascular system, the regimens have been limited by a number of factors. The anticoagulant rivaroxaban was studied in individuals who had recently experienced an acute coronary syndrome, and the study's primary efficacy end goal was reached. Rivaroxaban, a factor Xa inhibitor, has a predictable pharmacokinetic profile and has not been linked to an elevated risk of hepatotoxicity. Rivaroxaban has been studied in a variety of therapeutic situations, including venous thromboembolism prevention and treatment, as well as stroke prophylaxis in atrial fibrillation [6].

Our research looked at two modest dosages of rivaroxaban in patients who had recently experienced an acute coronary syndrome. The 2.5 mg dose of rivaroxaban reduced the primary efficacy end point when compared to placebo, as well as the risk of death from cardiovascular causes (relative reduction of 34%; absolute reduction of 1.4% points) and any cause (relative reduction of 34%; absolute reduction of 1.4% points) (relative reduction, 32% ; absolute

reduction, 1.6% points). The 2.5 mg dose of rivaroxaban was found to have a nonsignificant but directionally consistent benefit for myocardial infarction and a significant reduction in the risk of stent thrombosis, suggesting that increased thrombin activity may play a role in these events.

Rivaroxaban has been compared against an active comparator (e.g., warfarin or enoxaparin) in previous research, and the bleeding rates were identical in both groups. The rates of bleeding were much greater in patients taking rivaroxaban in our research, when the comparator was placebo, as expected [7]. When compared to placebo, both rivaroxaban doses increased the risk of bleeding, however the lower rivaroxaban dose resulted in less bleeding than the larger dose. Other than bleeding events, the combination rivaroxaban and placebo groups had similar rates of adverse events.

Other novel factor Xa and IIa inhibitors, in addition to rivaroxaban, have been studied in patients who have had an acute coronary syndrome. Rivaroxaban, apixaban, dabigatran, and darexaban all demonstrated a dose-dependent increase in bleeding in phase 2 studies. Rivaroxaban and apixaban both showed signs of reducing cardiovascular events in the ATLAS ACS–TIMI 46 and Apixaban for Prevention of Acute Ischemic Events 1 (APPRAISE-1) trials (NCT00313300). APPRAISE-2 (NCT00831441) then compared apixaban to placebo in a phase 3 trial, finding that adding 5 mg of apixaban twice daily to antiplatelet therapy increased the number of major bleeding events without a significant reduction in the rate of recurrent ischemic events in patients with an acute coronary syndrome [8].

Some of the discrepancies between our findings and those of APPRAISE-2 could be attributed to the patient groups. Patients having a history of ischemic stroke or transient ischemic attack who were to be treated with aspirin plus a thienopyridine were specifically excluded from the study, as this group has not shown to benefit from higher doses of antithrombotic medication.

In the Apixaban for Reduction in Stroke and Other Thromboembolic Events in Atrial Fibrillation (ARISTOTLE) trial (NCT00412984), a 5 mg dose of apixaban twice daily was examined in both patients with atrial fibrillation and those with an acute coronary syndrome in APPRAISE-2 [9]. The majority of participants in the trials testing rivaroxaban for stroke prophylaxis or treatment of venous thromboembolism got at least 20 mg per day. The doses of rivaroxaban used in our trial were a quarter or half of the 20 mg dose [10]. In the end, the lower dose of rivaroxaban provided a survival benefit, but not the greater amount.

The numerical increase in fatal bleeding linked with the higher dose of rivaroxaban helps to explain this observation. Other repercussions of nonfatal bleeding, on the other hand, could have contributed to this conclusion. Inverse dose–response correlations with cardiovascular events were also reported in ATLAS ACS–TIMI 46 and RUBY-1 (a phase 2 evaluation of darexaban placebo in individuals after an acute coronary syndrome) (NCT00994292). As a result of our research, as well as critical findings from APPRAISE-2, ATLAS ACS-TIMI 46, and RUBY-1, extremely low dosages of an oral anticoagulant appear to be the most beneficial in individuals with a recent acute coronary syndrome.

Finally, rivaroxaban treatment reduced the risk of death from cardiovascular causes, myocardial infarction, or stroke in individuals with acute coronary syndromes across the board. This positive effect was accompanied by an increase in bleeding rates. There was no substantial increase in the rate of fatal bleeding, and the 2.5 mg rivaroxaban dose given twice daily reduced overall and cardiovascular mortality. As a result, the combination of rivaroxaban to very-low-dose anticoagulation may be a new therapy option for individuals with a recent acute coronary syndrome.

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