

Massive intracranial hemorrhage in trivial head trauma due to iatrogenic coagulopathy – A case report

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ABSTRACT

Anticoagulants are regularly prescribed for treating patients who had acute or chronic coronary episodes. The potential hazardous side effects are almost always not shared to such patients or their bystanders. Bleeding episodes that occur due to even trivial trauma can result in life threatening complications for such patients. A case report and discussions concerning this matter is presented in this paper.

Keywords: anticoagulants, intracerebral hemorrhage, subdural hemorrhage, trivial trauma

INTRODUCTION

Occlusive coronary artery disease is the usual etiology for acute coronary syndromes.¹ Stenting procedures or coronary artery bypass surgeries (C.A.B.Gs) are the principal treatment procedures for occlusion of coronary vessels. Following such treatment modalities, the patients are discharged with advice on anticoagulants or fibrinolytics along with antihyperlipedemics and diuretics. Earlier only the elder had to undergo this ordeal, but now anticoagulants are prescribed for all. The life expectancy of several patients would have been reduced but for the prompt and efficient intervention of the cardiologists.

However anticoagulant therapy always has its disadvantages, though less often, involves risk of life threatening complications such as bleeding into specific locations or severe bleeding following trauma. It gets worse in hypertensive patients. Intracerebral hemorrhage, gastrointestinal bleeding, hemarthrosis etc are some of the complications seen in patients on anticoagulants. Therefore it is not surprising that many physicians are reluctant to prescribe anticoagulant drugs in presence of risk factors especially in elderly patients.² Case report of a patient, undergoing treatment with anticoagulants, who had died due to severe intracranial bleeding following a trivial head trauma is reported.

CASE IN BRIEF

A 57 year old man was admitted with retrosternal discomfort. He had no past history of similar episodes. He was not diabetic. He was not on any chronic medications. He was a smoker and used to consume alcohol occasionally. At the time of admission, his blood pressure was 146/110mmHg. Laboratory investigations had revealed his serum total cholesterol as 269.5mg/dl and LDL fraction as 196mg/dl. ECG showed normal sinus rhythm with T inversion in lead I, aVL, V1 to V6 and troponin T was positive. He was initially managed with nitrates, antiplatelets, beta blockers, low molecular weight heparins and other supportive treatment. Later, coronary angiogram was done which revealed single vessel disease of Left Anterior Descending (L.A.D) branch of Left Coronary Artery. However, left ventricular function was adequate. Subsequently stenting of proximal portion of L.A.D was done; and the patient was advised to take Betaloc, Clopidogrel, Ecospirin and Atorvastatin drugs in adequate doses. He was instructed to not to stop medications without consulting the cardiologist but later he had never turned up for any reviews.

The patient met a road traffic accident while crossing the road and sustained head injury. He was unresponsive when presented at the casualty. There was bleeding from his left ear. His pulse was 56/min and blood pressure was 90/60 mmHg.

Glassgow Coma Scale was 3/15 (E1V1M1). Pupils were not reacting to light. Other systemic examinations revealed normal findings. There was a lacerated wound 6x3cms on the right side of occiput, outer to midline.

The patient was admitted in ICU. His investigation results on the day were: Hb- 11.8g/dl, Platelet - 109K/uL, Prothrombin Time with INR - 2.66s and APTT - 2.7s. The hemodynamics was deteriorating on subsequent days. He was started on antiepileptics and antiedema measures. Blood transfusions and inotropics were started to correct haemodynamics. But the patient succumbed to his injuries on third day of admission.

Since it was a medico-legal case the body was subjected to autopsy. There was thick subgaleal hematoma around the occipital region. A linear fracture 10cms long was present on the left side of occipital bone extending into temporal bone of same side. Brain (1300gms) was softened and edematous. Thick dark subdural and subarachnoid hemorrhages were seen diffusely on both cerebral hemispheres covering occipital, parietal, temporal and frontal lobes. The hemorrhage had also spread to the base of brain. The opinion as to cause of death was concluded as due to massive intracranial bleeding.

DISCUSSION

Massive fatal bleeding is a serious concern of any physician treating a patient with anticoagulants. Aspirin which inhibits platelet thromboxane A₂ production is the most widely used drug for primary and secondary prevention of atherosclerotic disease. Clopidogrel is an alternative antiplatelet agent that inhibits adenosine diphosphate (ADP) – induced platelet aggregation.⁶ This occurs through irreversible inhibition of P₂ nucleotide receptors on the platelet surface. So, both aspirin and clopidogrel inhibit platelet aggregation through independent mechanisms and they are used in combination especially after coronary stenting or acute coronary syndrome. Like aspirin, clopidogrel may cause adverse bleeding events via the antiplatelet action.

While prescribing antiplatelet risk attributable to treatment vis-a-vis with the established absolute benefits in cardiovascular outcomes. A meta-analysis of such treatment strategies have shown the combination of low-dose aspirin plus clopidogrel to be increasing the risks of bleeding as compared with either agent alone.⁷ In another randomized, double-blinded and placebo-controlled trial; life threatening bleedings were higher in the group of patients receiving aspirin and clopidogrel versus clopidogrel alone (2.6% vs 1.3%).⁸

There are several risk factors that cause spontaneous bleeding in individuals. Many reports have clearly demonstrated the rapid enlargement of hematomas in patients on warfarin therapy.^{9,10} In a study the investigators had found two fold mortality in aspirin users compared with nonusers of aspirin/warfarin. Here the deaths of aspirin users were attributable to Intracranial Hemorrhage (ICH).¹¹ The same scientists on another occasion had observed prolonged bleeding times on admission in ICH patients who had been using aspirin, and even after discontinuation of aspirin, since the effect of the drug on hemostasis continued for few more days.¹² In one Swedish Aspirin Low-dose Trial (SALT), there was a significant increase in the risk of fatal hemorrhagic strokes among aspirin (75mg/day) users.¹³ Several studies have revealed that decreased blood coagulability^{14,15}, increased blood pressure¹⁰, liver disease¹⁵, thrombocytopenia¹⁵ etc. are the other risk factors. It is clear that persons who experience head trauma have an increased likelihood of developing intracranial bleeding^{16,17} (especially subdural hematomas), and there is increased risk in persons taking anticoagulant agents.

CONCLUSION

Anticoagulants are a necessary evil for treatment of all acute and chronic coronary syndromes. The treating doctors need to educate the patients while administering them such anticoagulant agents regarding potential side effects. If not, the

patients become susceptible to fatal bleeding episodes. Treating a patient begins from educating them regarding the disease as well as the plan of treatment. Both the pros and cons of each treatment modality must be explained in detail to the patient. This is the cornerstone of Medical Ethics and Indian Medical Council Regulations.

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