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Emergency Management of Patient under Antiplatelet Drug Therapy: A Case Report

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Abstract: ***Introduction: Adequate hemostasis is crucial for the success of emergency treatment, since bleeding problems can give rise to complications associated with important morbidity-mortality. The emergency treatment of patients who tend to have an increased risk of bleeding due to the use of anticoagulant and/or antiplatelet drugs raises a challenge in the daily practice of dental professionals. Adequate knowledge of the mechanisms underlying hemostasis, and the optimized management of such patients, are therefore very important issues. Purpose: The aim of this science report is to show a case of emergency management of patient under antiplatelet drug therapy. Material and methods: The clinical case is showed from adult male, 56 years old, which accidentally have lacerated wound on his tongue while he was slipped. He had been treated under antiplatelet drug therapy for his cardiac disease since 5 years ago. Based on adequate clinical medical historyand laboratory examination we will discuss the clinical approach and the best emergency management, including treatment consideration for the patient. Conclusions: Patient medication indicated for the treatment of background disease should not be altered or suspended unless indicated by the prescribing physician. Emergency managementofpatient under antiplatelet drug therapy, consists of wound debridement and suturing of lacerated wound under local anesthetic agent, hyaluronic acid gel, and hemostatic gargle have showed successful outcome.***

Keywords: anticoagulants, antiplatelet drugs,hemostasis, lacerated wound

1. Introduction

An emergency is a situation that poses an immediate risk to health, life, property, or environment.Most emergencies require urgent intervention to prevent a worsening of the situation. Some emergencies cause an immediate danger to the life of people involved, butmany emergencies are not necessarily immediately threatening to life, but might have serious implications for the continued health and well-being of a person or persons (though a health emergency can subsequently escalate to life-threatening).

Hemostasis is a defense mechanism composed of a series of independent biological systems that aim to preserve vascular integrity and avoid blood losses, while ensuring optimum fluidity throughout the circulatory system.[1,2]Hemostatic alterations have a broad range of potential causes, including deficiency states, hereditary and metabolic alterations, cancer, etc. However, at present, the most frequent cause of blood coagulation disorders in developed countries is the use of drug substances.[4] Many drug products are available for the prevention of thromboembolic events; it is therefore very important for dental professionals to know these products, their mechanisms of action, and the measures of caution required in order to prevent complications.

Tissue damage is generally associated to vascular injury resulting in more or less profuse bleeding.[2] Vascular endothelial rupture exposes different proteins of the subendothelial tissue layer to the bloodstream, triggering three different hemostatic mechanisms, such as : vasoconstriction phase, platelet phase, and plasmatic phase.[2-4]

Antiplatelet drug (antiaggregant) is a member of a class of pharmaceuticals that decrease platelet aggregation[1] and inhibit thrombus formation.Antiplatelet or platelet aggregation inhibitor drugs inhibit the aggregation of platelets, thereby avoiding platelet clot formation and suppressing the first hemostatic phase. [4]In contrast, anticoagulant drugs inhibit the enzyme vitamin K reductase, which mediates conversion of vitamin K epoxide to its active form. As a result, the formation of coagulation factors dependent upon this active form is inhibited, and the coagulation process is blocked.[4]

Prevention and treatment of arterial thrombosis is essential in patients with certain medical conditions whereby the risk of thrombosis or thromboembolism may result in disastrous consequences such as heart attack, pulmonary embolism or stroke.[2] Patients who require the use of antiplatelet drugs are: stroke with or without atrial fibrillation, any heart surgery (especially prosthetic replacement heart valve), Coronary Heart Disease such as stable angina, unstable angina and heart attack, patients with coronary stent, Peripheral Vascular Disease/Peripheral Arterial Disease and apical/ventricular/mural thrombus.[2]At clinical level, the end result following surgery is the same in both cases (antiplatelet and aticoagulant), i.e., hemostatic alteration that may result in bleeding which can prove difficult to control.The aim of this science report is to show a case of emergency management of patient under antiplatelet drug therapy.

2. Material and methods

Case Report

A 56 years old male patient came with bleeding from mouth. ± 3 hours prior to admission when the patient was taking a

bath, he was slipped and fell down with his tongue accidentally bitten. There was no other injury on his body. Medical history: The patient was diagnosed with coronary heart disease 5 years ago. He had undergone cardiac catheterization procedure and had been treated under antiplatelet drug therapy since then. The patient has been given 5 kinds of medicine telmisartan, cetirizine, spironolactone,bisoprololand aspirin by his cardiologist for treating his cardiac disease.

The physical examination revealed there was lacerated wound at his dorsal tongue region with 2x 1x 0.5 cm in size, irregular edge, muscle based. Laboratory examination revealed there was a normal platelet count, and increase levelof PT, APTT test.

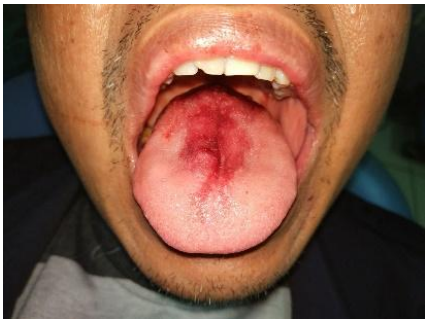

Figure 1: Lacerated wound of the dorsal tongue region

We treated the patient under local anesthetics agent. The clot was removed and also debridement of the necrotic tissue. The tongue was repaired by layers with simple interrupted Polyglactin 910 4.0 sutures for deeper area and Silk 4.0 for superficial area. With a time of surgery 25 minutes. We prescribed hyaluronic acid gel, hemostatic gargle, Cefadroxil500 mg two times daily for 5 days, and dexamethasone 0,5 mg three times daily for 3 days. The patient was discharged 30 minutes post operatively since no active bleeding had been seen.

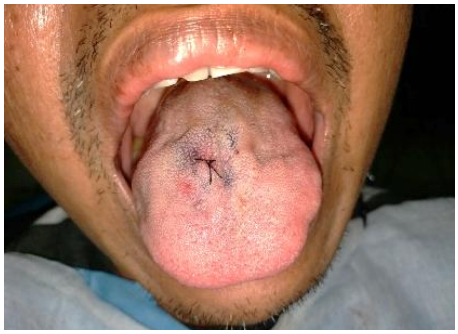

Figure 2: Complete repair showing appropriate continuity of the tongue and there was no signs of active bleeding

3. Discussion

Tissue damage is generally associated to vascular injury resulting in more or less profuse bleeding.[2] Vascular endothelial rupture exposes different proteins of the subendothelial tissue layer to the bloodstream, triggering three different hemostatic mechanisms, such as : vasoconstriction phase, platelet phase, and plasmatic phase[2-4].

Vascular or vasoconstriction phase: Vasoconstriction of the damaged blood vessel occurs immediately afte vascular injury, mediated by the vascular smooth muscle, and reduces blood loss from the damaged vessel. It does have two important effects: it reduces blood loss and triggers the second phase, facilitating platelet adhesion secondary to exposure of the subendothelial collagen fibers and basal membrane of the damaged blood vessel wall.[2]

Platelet phase, or platelet clot formation: The purpose of this phase is to form a primary hemostatic clot composed of aggregated platelets.[2] Platelet main function is to maintain vascular integrity and form a platelet clot in the event of vascular damage.[4] The normal count is between 150,000 and 400,000 platelets/mm3, and the platelet maturation sequence lasts 3-5 days.[4] The platelets adhere to the subendothelial collagen exposed as a result of vascular damage, and aggregate to form a platelet clot[4] that contributes to reduce blood loss.

Plasmatic phase, or production of fibrin that stabilizes and reinforces the platelet clot (coagulation): This phase is characterized by a complex series of proteolytic reactions known as the coagulation cascade. The classical cascade comprises two pathways: extrinsic and intrinsic, which in turn merge to form a common pathway.[1]The plasmatic coagulation phase involves the transformation of fibrinogen (soluble) into fibrin (insoluble), mediated by thrombin – a proteolytic enzyme formed by the activation of prothrombin, following sequential activation of the coagulation factors.[1]The intrinsic pathway is started by the activation of factor XII through contact with the subendothelial tissues in the damaged zone. The extrinsic pathway in turn is started when blood comes into contact with the tissue thromboplastin released by the damaged tissues, with activation of factor VII. [1,2]Finally, the blood clot is dissolved in the fibrinolytic phase. When the damaged vascular wall is repaired, activated factor XII facilitates the conversion of an inactive plasma molecule to its active form, called kallikrein. The latter in turn catalyzes the conversion of inactive plasminogen to the active molecule plasmin - an enzyme that digests fibrin, yielding degradation products and promoting dissolution of the clot. Once fibrin degradation has been completed, plasmin is quickly neutralized by antiplasmin. [2,4]

In cases of suspected bleeding disorders, initial laboratory evaluations should include a complete blood count with platelet count, peripheral blood smear, prothrombin time , and partial thromboplastin time.[5]Five studies are important to the diagnosis of bleeding disorders: bleeding time (BT), platelet count, activated partial thromboplastin time (aPTT), prothrombin time (PT), and thrombin time (TT). If the platelet count alone is low, the cause is usually peripheral destruction of platelets, immunothrombocytopenia, or an abnormality of bone marrow production. An abnormal bleeding time alone suggests a platelet aggregation defect that is most likely due to medication. When the aPTT is the only abnormal test and the patient has a definite history of bleeding, one of the hemophiliac states is present. An abnormal PT, with or without an abnormal aPTT but with normal results in the other three tests, indicates an abnormal reduction in the vitamin K-dependent clotting factors (II,

VII, IX, X) or factor V. When the TT is abnormal, disseminated intravascular coagulation, the presence of plasma heparin, or a hepatopathy should be suspected.[6]

A patient's individual risk of bleeding complications is dependent on a variety of factors, including the type and combination of anticoagulants or antiplatelet drugs they are taking, their underlying health conditions and other medications that they be taking. The patient's medical history and details of the prescribed and non prescribed medication that they are taking should be noted at the start of each course of treatment and checked for any changes at each visit.[5]Therefore, a complete anamnesis of medical history should be taken by the clinician before starting the treatment procedure.

In this case, the patient was treated under single antiplatelet drug therapy (aspirin). Low dose-long term aspirin use irreversibly blocks the formation of thromboxane A_2 in platelets, producing inhibitory effect on platelet aggregation.[7]Patients who are on dual or combination therapies and are taking more than one anticoagulant or antiplatelet drug are likely to have ahigher bleeding risk than those on single drug therapies.[5] The clinical experience of dental professionals suggest that dual antiplatelet medication can lead to prolong bleeding following an invasive procedure. However once formed, the clot tends to be reasonably stable. Conversely, clinical experience suggests that for the patients taking anticoagulants, blood clots may form more quickly than with antiplatelet drugs but can also be more easily dislodge.[5] The use of sutures at the time of treatment, in addition to hemostatic agent, usually stabilizes the wound and may reduce the likelihood of prolonged or subsequent re-bleeding and the need for the patient to return for further treatment.[5]This patient showed complete repair of the wound and appropriate continuity of the tongue and there was no signs of active bleeding.

In the past decade, it has become clear that routine discontinuation of oral anticoagulant therapy for dentalprocedures is not supported by the scientific literature, as itmay put patients at unnecessary medical risk forthromboembolic events either from the cessation ofanticoagulant therapy or because of "reboundphenomenon.". This phenomenon is hypercoagulabilityowing to increased thrombin production or plateletactivation if therapy is abruptly discontinued, which candamaged prosthetic cardiac valves and even causethrombotic deaths in dental patients. So, unless seriousbleeding is anticipated, the therapy should be continued.[5]

For this case, we use dexamethasone as analgesic drug of choice because it has a potent anti-inflammatory properties and has no negative effect if being used simultaneously with antiplatelet agents. Association of new oral anticoagulants with otheranticoagulants, platelet inhibitors(Aspirin, Clopidogrel, Ticlodipine, Prasugrel,Ticagrelor, and others), and non-steroidal anti-inflammatorydrugs (NSAID) increases the bleeding risk.[8]The concomitant administration of nonselective reversible COX-1 inhibitors such as ibuprofen and naproxen may lead to impairment in the efficacy of aspirin.[7]For all patients taking anticoagulants or antiplatelet drugs, hemostasis should be achieved before the patients being discharged from care.[5]

4. Conclusions

Many management protocols have been developed, though in all cases a full clinical history is required, together with complementary hemostatic tests to minimize any risks derived from invasive treatment. Patient medication indicated for the treatment of background disease should not be altered or suspended unless it was indicated by the prescribing physician. Local hemostatic measures have been shown to suffice for controlling possible bleeding problems resulting from emergency treatment. Emergency managementofpatient under antiplatelet drug therapy, consists of wound debridement and suturing of lacerated wound under local anesthetic agent, hyaluronic acid gel, and hemostatic gargle have showed successful outcome.

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