Gingival bleeding as a presenting sign of primary fibrinogenolysis

To the Editor:

A 52-year-old man presented with a 1-week history of oral cavity bleeding and was referred to the Department of Periodontics. There were neither systemic diseases nor hematopathology in his past medical history. There was no history of smoking, alcohol abuse, or medication intake. Intraoral examination revealed poor oral health care with a calculus level of +++ using the Volpe-Manhold Index. All gingiva were inflamed. The sulcus bleeding index of most teeth was 3 to 4. Laboratory evaluation revealed a hemoglobin level of 148 g/L, white blood cell count of 5.1 x 10^9/L, and platelet count of 134 x 10^9/L. Prothrombin time (PT) was 14.00 seconds, thrombin clotting time 16.90 seconds, and international normalized ratio 1.13. Activated partial thromboplastin time (aPTT) was 30.10 seconds. Because all of these indexes were normal, we prescribed supragingival scaling.

Scaling resulted in increased bleeding, including active bleeding of all gingival papillae and gingival margins. Bleeding was not controlled by either compression or periodontal dressing. Additional measures taken included suturing iodoform packs and injecting p-aminomethyl benzoic acid (Pamba), but neither of these were effective. On further inquiry, the patient reported having a history of hemospermia during the preceding 3 months and hematuria during the preceding month.

The patient was then transferred to the Department of Hematology. Further hematologic evaluation revealed a fibrinogen (Fib) level of 1.1 g/L (normal: 2-3 g/L), and fibrin degradation product (D-dimer) level of 3.2 mg/L (normal <0.2 mg/L). In light of these additional findings a preliminary diagnosis of “primary fibrinogenolysis with unknown primary infection” was made. Based on the urologic history, additional laboratory examinations and adjunctive tests were performed. These revealed total prostate-specific antigen (PSA) of 62.7 μg/L (normal <4.0 μg/L) and free PSA of 9.3 μg/L (normal <0.8 μg/L), indicative of prostate cancer. Positron-emission tomography/computerized tomography and pathologic examination confirmed the diagnosis of “metastatic prostatic carcinoma.”

Primary fibrinogenolysis is characterized by spontaneous activation of plasminogen into plasmin, causing generalized fibrinogenolysis with abnormal production of fibrinogen/fibrin degradation products (FDP), and degradation of fibrin, fibrinogen, and coagulation factors V, VII, VIII, IX, and XI, complement components, and other plasma proteins. Fibrinolytic activity of plasmin is initiated by plasminogen activators, including tissue plasminogen activator (t-PA) and urokinase (u-PA), and is downregulated by plasminogen activator inhibitors 1 and 2 and plasmin inhibitor α2-antiplasmin.

Primary fibrinogenolysis has been reported to occur in various conditions, including shock, trauma, surgical procedures, acute leukemia, and severe liver diseases. Primary fibrinogenolysis can also be caused by neoplasia, such as prostatic cancer, breast cancer, and renal cell carcinoma. In these instances, an excessive amount of plasminogen activators might be released into the blood from body stores (mainly endothelial cells) and may exceed the capacity of inhibitors. Tumor tissue or cells may also contain plasminogen activators, especially u-PA.

Coagulation profiles in these patients usually show: 1) normal platelet count and plasma levels of clotting factors, except for factors V and VIII, which are more sensitive to the proteolytic action of plasmin; 2) normal or slightly prolonged PT and aPTT; 3) depletion of plasma Fib and increased plasma levels of FDP and D-dimer. Of these, the decreased Fib and increased FDP and D-dimer levels have diagnostic significance. Coagulopathy should always be considered in periodontal clinical work. With an increasing incidence of malignancy, primary fibrinogenolysis secondary to certain cancers may first present in a periodontal clinic.

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REFERENCES

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