Epistaxis Due to Platelet Dysfunction in a Healthy Person During Office Reconstruction

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Abstract—A healthy 63-year-old male developed severe unprovoked recurrent nose bleeding over a 40-day period. He had no other symptoms and his past-medical and family histories were non-contributory. His physical examination, complete blood counts, and coagulation profile were normal. Three weeks into the illness, a platelet function screen was found abnormal. Hematology consultation did not find a cause for his thrombasthenia. After a short hospitalization for severe epistaxis, recurrent nosebleeds stopped. Several months later, the platelet function screen was normal. Post-hoc review of history found that the onset and duration of his nose bleeding and platelet dysfunction coincided with office reconstruction, which generated a strong aromatic smell. He was physically closer to the reconstruction site than unaffected co-workers. There was a temporal relation between the intensity of exposer to office environment and the intensity of bleeding. It is probable that the cause of his platelet dysfunction was an inhaled unidentified toxic chemical(s). The management of his epistaxis and risk of inhalation of toxins as a cause of platelet dysfunction are discussed.

Index Terms—Air Pollutants; Diagnoses and Laboratory Examinations; Environment and Public Health

I. INTRODUCTION

Critical analysis of patient history generally offers useful insights into pitfalls of the clinical management, suggests novel etiology of the disease, or unmask hidden aspects of the health care. We analyzed history of one such patient with epistaxis, which provided insights into the management and probable etiology of a common problem.

II. CASE PRESENTATION

A previously healthy 63-year-old health-care-worker developed spontaneous recurrent severe bleeding from nostril which he self-treated with local pressure. He was not taking any medication, was not consuming alcohol, and had no allergy or any other illnesses. He had no past-medical or family history of bleeding. He then went on a two-week trip abroad during which he had two episodes of severe epistaxis; he was seen by ear, nose and throat (ENT) specialist who found no abnormality on examination. The complete blood cell count (CBC), prothrombin time (PT), and activated partial thromboplastin time (aPTT) were normal. Upon his return from the trip, three weeks since the first epistaxis, nosebleeds returned. He was examined by two ENT specialists who found no abnormalities. Physical examination by his internist was normal. All nose bleedings were controlled with application of the nasal vasoconstrictor,

moistening ointment, and local pressure. He was able to continue his office work.

Repeated CBC, PT, and aPTT were normal. Platelet filtration assay (PFA-100) showed significantly prolonged closure times in the collagen/epinephrine and collagen/ADP channels on three different occasions (Fig. 1). His von Willebrand factor antigen and activity were normal on two separate testing. Anti-platelet antibody test was negative. Study of platelet aggregation was performed twice and showed normal pattern with collagen and non-specific pattern with ADP, epinephrine and arachidonic acid. Two weeks after his return from a trip abroad, the severe epistaxis reoccurred and was stopped with nasal tamponade. After a short hospitalization followed with a four-day home stay, the recurrent bleedings stopped. Several months later, PFA-100 tests were normal (Fig. 1).

Post-hoc review of 40-day long illness showed that the bleeding coincided with the onset and duration of office reconstruction, which generated a non-irritating aromatic smell; air contained no visible dust. The reconstruction work involved part of the floor of office building, was isolated from office workers with provisionally made panel-walls and plastic sheets, and whole area was aerated thorough the central ventilation system. The patient office door was physically closer to the entrance into reconstruction zone ($\sim 2 \text{ meters}$) than any of unaffected co-workers in the department. There was also temporal relation between the exposer to office environment and patient symptoms (Fig. 1).



Fig. 1. Temporal and physical proximity of the patient to the presumed source of exposure and the sequence of bleeding (red flags)and abnormal platelet function test results.

We conclude that a probable cause of his platelet dysfunction and epistaxis was an inhaled unidentified chemical.

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III. DISCUSSION

The history of recurrent unexplained nosebleeds in an apparently healthy individual deserves consideration of its etiology and clinical management.

A. Etiology Aspect

The evidence that an environmental factor was inhaled and caused his platelet dysfunction and nose bleeding is circumstantial. Nonetheless, it is sufficient to generate a hypothesis based on inhalation of an unidentified toxin (especially aromatic toxic chemicals) and temporal and physical associations between his symptoms and the environmental source. Unfortunately, health assessment and platelet function testing for reconstruction workers are not routinely available. The number of compounds currently used in construction materials that could be released in to air is big. These potentially toxic chemicals are increasing in number, and most are untested or presumed harmless.

Review of literature shows environmental factors associated with epistaxis include temperature, humidity, and air pollutants. Temperature, humidity, and their sudden changes are inconsistently shown to be risk factors (1-5). Similarly, among air pollutants – O3, CO, SO2, NO2, and particulate matter (PM10) – only O3 was firmly associated with epistaxis (1,6-8) We found no study that links inhalation of toxins (or any other materials) from construction sites to platelet dysfunction (or nose bleeding). Thus, our patient history suggests that a toxic inhalation could be an environmental risk for epistaxis due to platelet dysfunction. This hypothesis could be tested by studying platelet function in a large cohort of construction workers, and in a case-controlled study of reconstruction and office workers.

B. Management Aspect

Review of 40-day long history of our patient illness suggests that he should have had platelet function testing earlier (not three weeks after the first episode of nose bleeding). Epistaxis is a frequent medical problem managed most often by general and family physicians, internists, pediatricians, ENT specialists, and hematologists. Our patient was seen in two countries by 11 physicians, five ENT and six hematology consultants; incidentally, their medical training and post-training experience spanned six countries on three continents. Recent literature reviews indicate that epistaxis is very common emergency in many health care settings caused by variety of factors, such as trauma, drugs, alcohol consumption, environment factors, platelet dysfunction, hematological and other diseases, and most often unexplained (9-12). Listing of platelet dysfunction as a cause of epistaxis, however, could be confusing. Platelet function is impaired by drugs, alcohol, and in hematological and other diseases that are known to cause epistaxis. In our patient, epistaxis was unexplained (idiopathic) until platelet function was tested and found to be abnormal (Fig. 1). Consequently, all patients with severe or recurrent (≥ 2) nose bleeding should have platelet function tested for two reasons. First, in patients considered to have 'idiopathic' epistaxis this test needs to be normal and must be considered an obligatory diagnostic criterion. Second, in patients with other risk factors for epistaxis, abnormal platelet function would confirm an effect of (etiological relation) drugs or alcohol; it may also indicate a hematological or other systemic disease. *Caveat emptor* in hematology workup of patient swith epistaxis: platelet count (CBC), platelet function screen and coagulation tests (PT and aPTT) should be considered a standard investigation in all patients with recurrent epistaxis.

IV. CONCLUSION

Platelet function test should be performed in all patients with severe or recurrent epistaxis. The normal platelet function test should be considered an obligatory criterion for diagnosis of idiopathic epistaxis. History of inhalation of aromatic or exposure to other toxins should be explored in patients with nosebleeds

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