Hemarthrosis of the Knees Following Streptokinase Therapy for Acute Myocardial Infarction

Eduardo Pimenta, Sônia M. A. Anti

São Paulo, SP - Brazil

A sixty-four-year-old male patient was studied who had acute coronary syndrome with ST segment elevation experienced bilateral hemarthrosis of the knees after administration of streptokinase and acetylsalicylic acid.

Streptokinase has well-known side effects, such as hypotension, bradycardia, allergic reactions, and bleeding. The most frequently observed hemorrhagic events, such as microscopic hematuria, hemoptysis and, in more severe cases, intracranial hemorrhage, occur at the puncture site. On the other hand, hemarthrosis involving both knees is a rare occurrence, and it has not been reported in the studies involving a great number of patients using thrombolytic medication [1,2].

This report describes, for the first time, the occurrence of bilateral knee hemarthrosis associated with the use of streptokinase in the acute phase of myocardium infarction.

Case Report

A sixty-four-year-old white male patient was admitted to the emergency room with the typical chest pain of acute coronary syndrome that had begun 30 minutes early. He denied risk factors for coronary disease, but he had had gout for 10 years with chronic and irregular use of nonhormonal antiinflammatory medication. A physical examination was unremarkable and the electrocardiogram showed right bundle-branch block and ST segment elevation in the V1 to V4 leads. When he was admitted to the intensive care unit, he received acetylsalicylic acid and a coronary vasodilator, and streptokinase infusion 1,500,000 UI was started. After the end of the administration, performed over 60 minutes, electrocardiographic alterations appeared suggestive of coronary reperfusion, evolving without hemodynamic alterations. Eight hours after the end of thrombolytic infusion, he complained of knee pain. A physical examination demonstrated a slight increase in local temperature and erythema without radiologic alterations. He evolved with progressive worsening of the phlogistic signs and the appearance of edema suggestive of articular joint effusion. We performed arthrocentesis of both knees, and we obtained a frankly hemorrhagic synovial fluid, 100 mL from the right knee and 50 mL from the left knee. The analysis confirmed hemarthrosis and excluded the presence of crystals. A hematological evaluation removed the possibility of a coagulation disturbance, and the patient evolved with complete remission of the local signs and symptoms.

Discussion

As for as we know, the case presented here is the first report in the medical literature related to the occurrence of bilateral knee hemarthrosis after the use of streptokinase. The occurrence of acute hemarthrosis results in the appearance of intense local pain and inflammatory signs. When hemarthrosis is chronic and recurrent, as in hemophilia, it is common for it to evolve to joint deformities and permanent functional damage [3]. Hemarthrosis is normally associated with a history of previous trauma or joint instability; however, the nontraumatic causes, such as complications from oral anticoagulant use [4] and, less frequently, with the use of heparin, have been described [5]. The occurrence of hemarthrosis after the use of a thrombolytic is rare.

The first description of secondary hemarthrosis due to the use of streptokinase was reported in 1990 in 2 patients with acute coronary syndrome, treated with streptokinase, acetylsalicylic acid, and heparin [6]. The first patient reported right knee meniscus removal 35 years before, and an inflammatory process in the same joint 5 weeks before, with the subsequent appearance of hemarthrosis. The other patient experienced gangrenous pyoderma for 1 year associated with symmetric seronegative erosive polyarthritis. On admission, he presented with knee and elbow hemarthrosis. The therapeutic combination with streptokinase, acetylsalicylic acid, and heparin has been responsible by the majority

Faculdade de Medicina do ABC - Santo André, SP
Mailing address: Eduardo Pimenta - Rua das Camélias, 357 – 04048-060 São Paulo, SP – Brazil - E-mail: espimenta@hotmail.com

of cases reported, and in the present study we had the same complication using only acetylsalicylic acid and streptokinase, which in theory would have a lower chance of provoking hemorrhagic disturbances.

Another two cases reported by Sanders, demonstrated unilateral knee involvement[7]. In one of the patients, with a previous history of articular disease recombinant tissue-plasminogen activator (rt-PA) was used in association with heparin. The second patient with previous knee surgery performed 4 years earlier was medicated with streptokinase and acetylsalicylic acid. In 1993, a patient with an articular hemorrhagic event was described after simultaneous administration of streptokinase, rt-PA, heparin, and acetylsalicylic acid, according to the GUSTO protocol. This patient did not have a previous history of articular disease, and the knee hemarthrosis was unilateral [8].

Two other patients had hemarthrosis after the use of recombinant tissue-plasminogen activator and heparin and had previous involvement of the compromised joint. The first patient had a right knee sprain four days before the hemorrhagic event [9], and the second, described by Gallardo et al [10] presented right knee tendinitis.

Of all cases described, only one demonstrated biarticular involvement, which did not occur in symmetric joints.

The present report describes bilateral involvement of the knee directly related to the use of intravenous streptokinase in the acute phase of myocardial infarction. In almost all reports previously mentioned, the complication occurred due to the use of combined thrombolytic (streptokinase or recombinant tissue-plasminogen activator) with acetylsalicylic acid and heparin, and in all cases, the description is of unilateral involvement. The similarity of this case with others is that all patients had a history of joint problems, which probably predisposes bleeding with the use of anticoagulants or thrombolytic agents, such as streptokinase.

This is also the first report of a patient with gout joint disease that developed hemarthrosis after intravenous streptokinase. This factor is important because gout is a common disease in the population, and metabolic disturbances, such as gout or hyperuricemia, are risk factors for coronary events in the future. Gout antecedents, such as severe blood hypertension, gastric ulcers, and others, should be taken into account when streptokinase is used.

This patient was treated as recommended for other causes of hemarthrosis, such as joint puncture for symptom relief, performance of a synovial fluid analysis, and also evaluation of suspension of anticoagulant medication, and the use of antiinflammatory and analgesic medications. The patient evolved without secondary joint disease.

When acute arthritis occurs after acute myocardial infarction, a differential diagnosis must be performed that includes acute gouty arthritis and diseases due to calcium pyrophosphate deposits (pseudo gout), because these two diseases may be triggered by muscular necrosis occurring during acute myocardial infarction. In the present report, these possibilities were rated out due to the absence of crystals in the synovial fluid.

Although thrombolytic therapy is still very important to decrease myocardial damage, intensive care physicians must be aware of the possibility of a less common complication like hemarthrosis that seems to be more frequent in previously compromised joints. Correct and early diagnosis, associated with the risk-benefit of medication, enables simple and efficient intervention, relieving patient's discomfort, and reducing the risk of joint sequelae.

References

1. ISIS-2 Collaborative Group. Randomized trial of intravenous streptokinase, oral aspirin, both, or neither among 17187 cases of suspected acute myocardial infarction: ISIS-2. Lancet 1988; II: 349-60.
2. GUSTO. An international randomized trial comparing four thrombolytic strategies for acute myocardial infarction. N Engl J Med 1993; 329: 673-82.
3. York JR. Endocrine and hemoglobin arthropaties, hemophilia. In: Klippel JH, Dieppe PA, ed. Rheumatology. United Kingdom: Mosby-Doyma, 1994: section 7 21.1-21.4.
4. Andes WA, Edmunds JO. Hemarthrosis and warfarin: joint destruction with anticoagulation. Thromb Haemost 1983; 49: 187-9.
5. Salzman EW, Deykin D, Shapiro RM, Rosenberg R. Management of heparin therapy: controlled prospective trial. N Engl J Med 1975; 292: 1046-50.
6. Oldroyd KG, Hornung RS, Jones AM, Andrews NP, Dawes PT, Carson PHM. Spontaneous haemarthrosis following thrombolytic therapy for myocardial infarction. Postgrad Med J 1990; 66: 387-8.
7. Sanders PA. Haemarthrosis associated with thrombolytic therapy. Br J Rheumatol 1991; 30: 236-7.
8. Birnbaum Y,Stahl B, Rechavia E. Spontaneous hemarthrosis following thrombolytic therapy for acute myocardial infarction. Int J Cardiol 1993; 40: 289-90.
9. Finlayson J, Dunsmuir RA. Haemarthrosis following thrombolytic therapy for acute myocardial infarction. Injury 1997; 28: 146-7.
10. Gallardo A, Gallego JC, Enero J, Barambio M, Domínguez P, Aguilera M. Hemartrosis como complicación hemorrágica tras tratamiento trombolítico. Rev Esp Cardiol 2001; 54: 1230-2.