

Case report

Acute stent thrombosis in a sirolimus eluting stent after wasp sting causing acute myocardial infarction: a case report

Martin Greif, Tilmann Pohl, Nico Oversohl, Christopher Reithmann, Gerhard Steinbeck and Alexander Becker*

Address: Department of Cardiology, Ludwig-Maximilians-University, Munich, Germany Marchioninistrasse 1581377 Munich, Germany

Email: MG - martin.greif@med.uni-muenchen.de; TP - tilmann.pohl@med.uni-muenchen.de; NO - nico.oversohl@med.uni-muenchen.de; CR - christopher.reithmann@med.uni-muenchen.de; GS - gerhard.steinbeck@med.uni-muenchen.de; AB* - alexander.becker@med.uni-muenchen.de

* Corresponding author

Received: 14 May 2009 Accepted: 27 July 2009 Published: 12 August 2009

Cases Journal 2009, 2:7800 doi: 10.4076/1757-1626-2-7800

This article is available from: <http://casesjournal.com/casesjournal/article/view/7800>

© 2009 Greif et al.; licensee Cases Network Ltd.

This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract

Introduction: Hymenoptera venoms contain thrombogenic substances that might be responsible for cardiovascular events independent of anaphylactic reactions.

Case presentation: We report a 55-year-old man who experienced an acute ST-elevation myocardial infarction after wasp sting. The patient presented without signs of anaphylaxis or shock. The coronary angiography showed an acute stent thrombosis of the right coronary artery. Percutaneous coronary intervention was performed immediately and this is an example for a cardiovascular complication associated with a hymenoptera sting, since the vasoactive, inflammatory, and thrombogenic substances of hymenoptera venoms potentially cause stent thrombosis and myocardial ischemia. To the best of our knowledge this is the first report of acute stent thrombosis in a sirolimus-eluting stent following hymenoptera sting.

Conclusion: Stent thrombosis is a possible complication after wasp sting induced by thrombogenic substances of the hymenoptera venom.

Introduction

Hymenoptera venoms contain thrombogenic substances that might be responsible for cardiovascular events, especially in patients with coronary artery disease. Independent of anaphylactic reactions phospholipase A₁ of the Hymenoptera venoms can induce thrombogenic reactions that might lead to acute arterial thrombosis.

Case presentation

A 55-year-old Caucasian German man with known ischemic heart disease presented in our emergency room, with an acute inferior myocardial ST-elevation infarction. Three years before stenoses of the proximal left anterior descending and the proximal right coronary artery were treated by PCI and implantation of two bare metal stents,

followed by PCI and implantation of a sirolimus-eluting stent (Cypher®) of an In-stent restenosis in the right coronary artery one year later. Afterwards no relevant cardiovascular events or hospitalizations occurred until the current admission. Clopidogrel (75 mg/d) was withdrawn 12 months after implantation of the sirolimus-eluting stent, while aspirin (100 mg/d) was continued. Known and treated cardiovascular risk factors were hypertension, hypercholesterolemia and diabetes mellitus.

As hobby beekeeper the patient was used to bee stings and never reported of any related symptoms, except local reactions. One hour before admission he was stung by 5 wasps. Just a few minutes after being stung he started complaining of chest pain and called an emergency physician. ECG revealed an acute myocardial infarction with inferior ST-segment-elevation (Figure 1), blood pressure was 140/85 mmHg, heart rate was 59/minute. The patient showed no signs of anaphylaxis or shock, neither any other complains. Aspirin (500 mg) and heparin (5000 IU) had been given by the emergency physician, chest pain improved after treatment with morphine.

Immediately after admission to the emergency room, coronary angiogram was performed showing a thrombosis in the two year old sirolimus-eluting stent of the right coronary artery occluding the vessel sub-totally. PCI was successfully performed with good angiographic result (Figure 2). Therapy with tirofiban (Aggrastat®, MSD, Germany) was started during intervention in a weight adjusted dose and had been continued for 24 hours. 600 mg clopidogrel were admitted following PCI, oral therapy was continued with 75 mg/day. Post interventional clinical course was uneventful, the patient recovered quickly, echocardiography showed left ventricular function within normal limits (fractional shortening 34%) without any regional wall-motion disturbances. The patient was discharged 6 days after myocardial infarction

on a regime of aspirin, clopidogrel, bisoprolol, ramipril and simvastatin.

Discussion

Myocardial infarction following hymenoptera stings (e.g. bees, wasps and hornets) is rare but there are some cases reported in the literature [1-5]. However to the best of our knowledge this is the first report of acute stent thrombosis in a drug-eluting stent caused by a wasp sting leading to myocardial infarction.

Despite from local cutaneous manifestations or generalized anaphylaxis, hymenopterans stings can cause a variety of unusual reactions. There have been reports of neurological reactions such as myasthenia gravis, peripheral neuritis and Guillain-Barré syndrome. Diffuse alveolar hemorrhage, acute renal failure, rhabdomyolysis, thrombocytopenic purpura and vasculitis are other pathologies presumed to be related to insect stings [6].

Myocardial infarction after bites of hymenopterans is mostly reported to be preceded by an allergic reaction, sometimes with angiographic evidence of undamaged coronary arteries [4]. Pathophysiological determinant seems to be related to the chemical composition of hymenopterans venom, basically containing vasoactive, inflammatory and thrombogenic substances (e.g. histamine, serotonin, dopamine, leukotrienes, thromboxanes and bradykinin) and potentially allergenic proteins (e.g. phospholipases, hyaluronidases, acid phosphatases, and mellitin) able to create vasospasm and/or coronary thrombosis [7,8]. Histamine and Serotonin are stimulating endogenous secretion of adrenalin and noradrenalin. The vasoactive mediators (e.g. histamine, serotonin, dopamine, noradrenaline) can lead also to myocardial ischemia either by hypotension or by increased myocardial oxygen demand due to direct inotropic or chronotropic effects, especially in patients with known coronary artery disease [2]. Many mediators influence

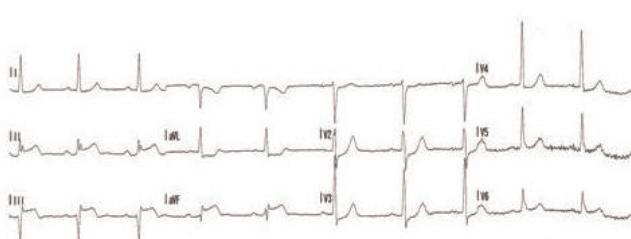


Figure 1

Figure 1. ECG registered by the emergency physician with ST-segment elevation in the inferior leads.

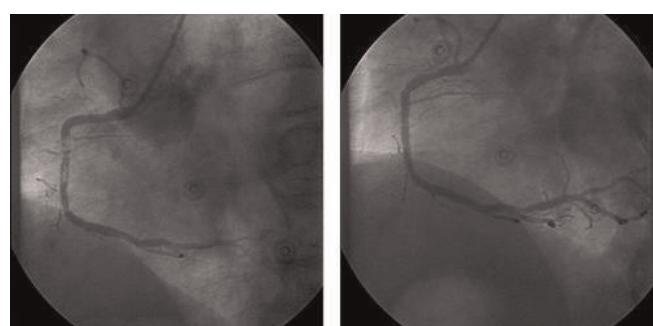


Figure 2. Acute stent thrombosis of the right coronary artery before (**Left**) and after PCI (**Right**).

potentially significant actions on coagulation, favouring platelet aggregation and thrombosis [9]. Yang *et al.* recently described a protein, magnifin, which was purified from the venom of a wasp *Vespa magnifica* (Smith), containing phospholipase-like activity and inducing platelet aggregation and thrombosis *in vivo* [10]. Chen *et al.* reported a case of thrombosis of the descending aorta and cerebral infarction after massive wasp stings [11].

In the cases reported, the causal relationship between hymenoptera sting and myocardial ischemia was sometimes not very clear. Some patients had signs of anaphylactic shock and therefore were treated with adrenaline. On one hand hypotension caused by anaphylaxis can lead to myocardial hypoperfusion, on the other hand adrenaline itself has thrombogenic effects and is able to provoke vasoconstriction [4], contributing to aggravation of myocardial ischemia. Korantzopoulos *et al.* reported of an acute ST-elevation myocardial infarction in the context of an anaphylactic reaction caused by a European hornet sting. In presence of ST-elevations at presentation the patient was not treated with adrenaline. In this patient exogen adrenalin did not contribute to the development of the myocardial infarction, but hypotension due the anaphylactic shock may have contributed to the development or the worsening of myocardial infarction [1].

In our patient no signs of anaphylaxis or shock were present. First symptoms of chest pain occurred a few minutes after the wasp stings. Immediately afterwards the patient called the emergency physician. Initial ECG revealed ST-elevations in leads II, III and aVF, while blood pressure was within normal range (140/85 mmHg). Considering the short time interval between the wasp attack and the beginning of chest pain, it is reasonable to assume, that the wasp venom caused acute stent thrombosis followed by myocardial infarction.

It is notable that our patient as beekeeper was used to bee stings and has been stung by bees many times without any symptoms except for local reactions. As bee and wasp venoms are highly complicated mixtures of pharmacologically or biochemically active agents, it remains unclear which components of the wasp venom differing from bee venom induced the acute stent thrombosis. Interestingly wasp venom contains more phospholipase A₁ as phospholipase A₂ in contrast to bee venom [12]. This and the finding of Yang *et al.* that phospholipase A₁ isolated from wasp venom inducing platelet aggregation and thrombosis *in vivo* [10], could be crucial for the distinct reaction of our patient to different hymenoptera venoms.

Conclusion

Stent thrombosis is a possible complication after wasp sting induced by thrombogenic substances as for example phospholipase A₁ of the hymenoptera venom.

Abbreviations

ECG, electrocardiogram; PCI, percutaneous coronary intervention.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review from the journal's Editor in Chief.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

MG was responsible for manuscript. TP, NO, CR, and GS were responsible for the diagnostics and treatment of the patient. AB was responsible for manuscript review.

References

- Korantzopoulos P, Kountouris E, Voukelatou M, Charaktsis I, Dimitroula V, Siogas K: **Acute myocardial infarction after a European hornet sting-a case report.** *Angiology* 2006, **57**: 383-386.
- Jones E, Joy M: **Acute myocardial infarction after a wasp sting.** *Br Heart J* 1988, **59**:506-508.
- Ceyhan C, Ercan E, Tekten T, Kirilmaz B, Onder R: **Myocardial infarction following a bee sting.** *Int J Cardiol* 2001, **80**:251-253.
- Wagdi P, Mehan VK, Burgi H, Salzmann C: **Acute myocardial infarction after wasp stings in a patient with normal coronary arteries.** *Am Heart J* 1994, **128**:820-823.
- Levine HD: **Acute myocardial infarction following wasp sting. Report of two cases and critical survey of the literature.** *Am Heart J* 1976, **91**:365-374.
- Reisman RE: **Unusual reactions to insect stings.** *Curr Opin Allergy Clin Immunol* 2005, **5**:355-358.
- Habermann E: **Bee and wasp venoms.** *Science* 1972, **177**:314-322.
- Wallace JF: **Disorders caused by venoms, bites and stings.** In *Harrison's principles of internal medicine. Volume 2.* 12th edition. Edited by Wilson JD, Braunwald E, Isselbacher KJ, Petersdorf RG, Martin JB, Fauci AS, Root RK. New York: McGraw-Hill; 1991:2187-2194.
- Handin RI, Loscalzo J: **Hemostasis, thrombosis, fibrinolysis, and cardiovascular disease.** In *Heart disease: A textbook of cardiovascular medicine. Volume 2.* 4th edition. Edited by Braunwald E. Philadelphia: WB Saunders; 1992:1767-1789.
- Yang H, Xu X, Ma D, Zhang K, Lai R: **A phospholipase A1 platelet activator from the wasp venom of Vespa magnifica (Smith).** *Toxicon* 2007, **51**:289-296.
- Chen DM, Lee PT, Chou KJ, Fang HC, Chung HM, Chen DM, Chang LK: **Descending aortic thrombosis and cerebral infarction after massive wasp stings.** *Am J Med* 2004, **116**:567-569.
- Binder M, Fierlbeck G, King T, Valent P, Buhring HJ: **Individual hymenoptera venom compounds induce upregulation of the basophil activation marker ectonucleotide pyrophosphatase/phosphodiesterase 3 (CD203c) in sensitized patients.** *Int Arch Allergy Immunol* 2002, **129**:160-168.