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Splenic Rupture and Massive Hemoperitoneum Due to Coagulopathy after *Atheris* Viper Snakebite

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Abstract: Coagulopathy with defibrination is one of symptoms accompanying snakebite envenoming, where life-threatening complications such as massive bleeding and organ hematomas formation can occur. Here, we report a case of hemocoagulation failure due to bite by African Great Lakes bush viper *Atheris nitschei* with impossibility of specific treatment for absence of antivenom and its life-threatening complication: very rare and unexpected atraumatic splenic rupture with massive hemoperitoneum and necessity of urgent splenectomy.

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Introduction

One of the snakebite envenoming manifestation is hemocoagulation failure: venom-induced consumption coagulopathy (VICC), most commonly with defibrination and subsequently possible bleeding and organ hematoma formations. This type of envenoming belongs to main venom's effects of snake's family *Viperidae* (not-European vipers, pit-vipers, rattlesnakes et al.). Besides of components affecting coagulation directly, in venom are present hemorrhagings and some other destructive enzymes that damage vascular endothelium and subendothelial tissue and are involved in the state of coagulation and the vascular system integrity during envenoming. The only effective final treatment for VICC is administration of appropriate antivenom.

In the case of African bush vipers *Atheris*, whose venom causes VICC, antivenom is not produced. Hence, in these cases, treatment is limited to mostly ineffective and not recommended fibrinogen (FBG) or fresh frozen plasma (FFP) substitution during several days of slow gradual spontaneous resolution of VICC, and eventual solution of emerging complications. Hemoperitoneum as a consequence of VICC is a relatively rare complication. Non-traumatic splenic rupture and bleeding after snakebite has been described so far in three cases, of which only in one case initially intact spleen with massive hemoperitoneum and splenectomy necessity, has occurred (Kang et al., 2014; Lal et al., 2014; Lee and Sung, 2019).

Case report

A 44-year-old man, an amateur snake breeder, was bitten into the 2nd finger of his right hand by the African Great Lakes bush viper *Atheris nitschei* (Figure 1). In about

Figure 1 – Great Lakes bush viper Atheris nitschei *(photo Jiroušek).*

an hour, he went to a specialized medical facility, the Toxinology Center of the General University Hospital in Prague, where he worked as a biochemist.

Affected hand was swollen, no clinical systemic symptoms were present. Anamnestically, he was observed for chronic glomerulonephritis.

In the first hemocoagulation examination 1 hour after the bite, only D-dimer 3,781 μg/l was recorded as pathological. Then, 5 hours after the bite, the laboratory finding already showed the development of VICC: INR (international normalized ratio) 3.8, APTT (activated partial thromboplastin time) 62.0 s, FBG < 0.1 g/l, D-dimer > 6,400 μg/l, finding of hemolysis. Next examination, 8 hours after the bite, already showed fully developed afibrinogenemia with INR > 10, APTT > 180 s, TT (thrombin time) > 180 s, D-dimer > 6,500 μg/l and hemolysis. Number of PLT (platelets) 214–216×10^9/l and antithrombin activities 65–70% did not deviate significantly from normal values, which corresponds with the common course of VICC. Leukocytosis (14–20×10^9/l) usually accompanies majority of snake envenoming.

As antivenom against *Atheris* viper venom is not produced, 6 TU (transfusion units) FFP were administered when FBG < 0.1 g/l. The effect was slight and transient, only in PT (prothrombin time) and TT (INR 3.8–2.5; TT > 180–63.4 s). Further examination showed again afibrinogenemia with immeasurable coagulation times. On the lower part of the right arm a hematoma formed. Due to the described positivity of the effect (Robinson et al., 2004) of formerly produced antivenom Near Middle East Antivenom Behringwerke (*Cerastes*, *Echis*, *Vipera* vipers), 3 vials of EchiTAb Clodomiro Picado (among others against *Echis* vipers) were administered as a test, but without any effect.

Laboratory findings of severe VICC with afibrinogenemia and unmeasurable coagulation persisted for 5 days, as well as hemolysis in some samples. At the same time, oliguric acute renal failure develops in grade III. according to KDIGO (Kidney Disease: Improving Global Outcomes) based on acute tubular necrosis in the field of already premorbid chronic glomerulonephritis with a creatinine peak of 395 μmol/l. During hospitalization, renal function gradually repaired without the need of renal replacement therapy, renal biopsy was not indicated.

From day 6, when coagulation values first appeared in the measurable range (FBG 0.14 g/l, INR 1.97, APTT 42.2 s, TT 27.1 s, D-dimer 5,024 μg/l), the patient was allowed to get out of bed for bathroom, contrary to previous strict sleep mode. Same day during defecation, weakness and vertigo suddenly developed. Diffuse palpable abdominal pain and hypotension (mean arterial pressure 55–60 mm Hg) were present, heart rate was stable, did not exceed 80/min. Urgent massive volume replacement therapy had not enough effective response; lower norepinephrine support (up to 0.12 μg/kg/min) was added. In laboratory findings Hb (hemoglobin) decline (97–74 g/l) and further progression of leukocytosis (11–16×10^9/l) after previous normalization were present. Bed side ultrasonography examination showed free fluid in the abdominal cavity, and immediate abdomen computer tomography

an obvious rupture of the spleen with massive hemoperitoneum. Norepinephrine support had to be increased temporarily up to 1 μg/kg/min.

Urgent revision of the abdominal cavity was indicated immediately, FBG 2 g, tranexamic acid 1 g were administered. Perioperatively found hemoperitoneum 3.5–4 l without blood clot formation and spleen rupture. Splenectomy performed. Another 2 g FBG, 6 TU RBC (red blood cells), 3 TU FFP, tranexamic acid 1 g were administered concurrently. During operation, the hemodynamic parameters gradually normalized, catecholamine support was reduced.

Spleen: 135×85×45 mm, in one edge with laceration of the capsule and parenchyma in the range of approximately 65×35×30 mm, in the adjacent approximately two thirds of the parenchyma dispersed multiple minor hemorrhages. Spleen capsule slightly dull. Microscopically, massive confluent fresh hemorrhages were found in the parenchyma. Rupture area without significant inflammatory reaction. Apart from hemorrhage, the structure of the parenchyma is usual with dominated congestion of the red pulp.

After the operation, the circulation parameters were normalized, catecholamine support was discontinued and Hb values corrected by transfusions to values of 90–100 g/l.

The further course was complicated on day 8 by bronchopneumonia, for which he was treated with antibiotics. Gradually occurring reactive thrombocytosis (308–1,230×10^9/l) as a result of splenectomy and infection was secured by acetylsalicylic acid 100 mg/day.

The patient was discharged in good health on day 17 after snakebite and admission.

Discussion

No specific antivenom is produced for the treatment of envenoming caused by *Atheris* vipers. The reasons are probably low incidence and mild course with low amount of bites complications in African territories of occurrence. The situation is similar in the case of snake's breeds. Literature description of envenoming is rare (Robinson et al., 2004; Top et al., 2006; Hatten et al., 2013). In 20-year-period, 5 cases of envenoming after *Atheris* bite were hospitalized and successfully treated without antivenom at the author's workplace (Valenta et al., 2014). Robinson et al. (2004) published a successful treatment using currently unproduced Antivenom Near Middle East Behrinwerke intended, among other, against the venom of the distantly related viper *Echis*. In our described case, however, a similar therapy with another antivenom against the African viper *Echis* proved ineffective.

FBG and FFP substitution with no antivenom treatment is not recommended. Present, not neutralized venom causes rapid destruction of FBG. The result is only an increase in fibrin degradation products, especially D-dimers. However, this procedure can be indicated in the case of the occurrence of serious bleeding, or its imminence during long-term afibrinogenemia (White, 2005; Berling and Isbister, 2015).

Renal injury accompanies a numbers of snakebite envenomations, especially with hemotoxic and myotoxic components in venom. Its origin is multifactorial, a participation has among others e.g. coagulation failure in VICC, hypotension, vascular infliction by hemorrhagins involvement, hemolysis with hemoglobinuria and others (Vikrant et al., 2017).

Although organ hematomas and spontaneous hemoperitoneum are not frequent complications of VICC, they may play a serious role in morbidity and lethality after snakebites in some localities of snake's occurrence (Berling and Isbister, 2015). Tchaou et al. (2016) reported in Benin (mostly saw-scaled vipers *Echis*) internal bleeding in 56% envenomed cases, in this number 22% of hematomas with hemoperitoneum and 12% hemoperitoneum cases. On other hand in Brazil Amazon, systemic bleeding was observed during hospitalization only in 15.3% lanceheads *Bothrops* snakebite patients with VICC, no hemoperitoneum included (Oliveira et al., 2019). Hemoperitoneum accompanying VICC is described rather sporadically (Rathod et al., 2003; Ahn et al., 2007; Diallo et al., 2019). Splenic rupture is then very rare complication (Kang et al., 2014; Lal et al., 2014).

Spleen tissue injury and its subsequent rupture in described case was contributed by more influences. Activated red pulp was probably congested, among others, even due to the number of hemolyzed erythrocytes. Also, the endothelial and subendothelial vascular injury caused by venoms hemorrhagines and other enzymes can be expected (Kang et al., 2014; Berling and Isbister, 2015). This, together with ongoing plasma coagulation system failure during VICC, caused numerous and confluent hemorrhages found in the parenchyma. Although the spleen has not been significantly enlarged, its more fragile structure could be expected. Probably, even with the participation of the abdominal pressure during defecation, there became a laceration of the parenchyma at the site of multiple hemorrhages, followed by rupture. Massive abdominal hemorrhage with incipient hemorrhagic shock requested urgent splenectomy.

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