Table 2 Clinical progression of edema and bleeding for viper envenomations in Benin

	Edema	Bleeding
Stage 1	Does not extend beyond wrist/ankle	Persistent atraumatic bleeding from the bite wound >1 h
Stage 2	Does not extend beyond major joints (elbow/knee)	Bleeding from old cuts and wounds elsewhere on patient
Stage 3	Extends beyond major joints	Spontaneous bleeding from healthy mucosa (i.e. gingiva)
Stage 4	Reaches but does not extend beyond multiaxial joints (shoulder/hip)	Externalization of internal bleeding (hematemesis, melena, etc.)
Stage 5	Extensive edema beyond multiaxial joints	Cerebral, meningeal, intra-abdominal, or retroperitoneal hemorrhage; critical hemorrhagic shock

or *N. katiensis*, one dry bite of unknown origin and one peculiar dry bite or very mild envenomation from an unidentified rear-fanged colubrid or small elapid. With the exception of the two suspected dry bites, all patients were treated with 1–6 vials of Antivipmyn® Africa; there were no deaths and all patients recovered without major sequelae. There were 17 patients (74%) who showed clinical signs of abnormal local or systemic bleeding and abnormal WBCT (Table 3) meeting diagnostic criteria for the hemorrhagic syndrome.

The 17 patients with hemorrhagic envenomations included two *B. arietans* envenomations and 15 *E. ocellatus* envenomations. Most (11/17, 65%) showed anemia (hematocrit ≤35%) and nearly half of these patients (7/17, 41%) arrived in critical condition with a late-stage hemorrhagic syndrome (stages 4 or 5), unstable vital signs, major complications, and a high likelihood of imminent death unless urgent interventions were taken. The findings of the initial clinical assessment at H_0 (abnormal local or systemic bleeding, anemia, blistering, necrosis, edema, etc.) supported the results of the initial diagnostic WBCT in all of our patients, and there were no false positive results at H_0. The remaining 6/23 patients without coagulopathy or bleeding are detailed below (Table 4) and included four clinically significant envenomations that received antivenom treatment and two suspected dry bites that were managed symptomatically.

Discordance in WBCT results at 20 and 30 min was observed in 82% (14/17) of patients with bleeding disorders who received both tests, and are detailed in Table 3. The variation we observed between WBCT results at 20 versus 30 min-reading times was substantial, with an average time to resolution of coagulopathy following antivenom treatment of 6.7 h by WBCT20 and 13.1 h by WBCT30. Discrepancies were noted in at least one pair of serial WBCT readings and were identified specifically in four circumstances: upon initial assessment at H_0 (Group 1, $n = 3$); upon initial normalization of hemostasis following antivenom therapy (Group 2, $n = 7$), upon secondary resumption of coagulopathy (Group 3a, $n = 3$); and upon restoration of hemostasis after a secondary resumption of coagulopathy (Group 3b, $n = 2$). There were three additional cases in which inconsistency was noted between results of the WBCT and the clinical assessment of the patient (Group 4). In these cases, both the WBCT20 and WBCT30 indicated restoration of hemostasis despite the presence of ongoing external and internal bleeding, which had markedly improved with antivenom therapy but persisted solely at the site of the gingival sulci for an additional 3 to 6 h before complete cessation of bleeding was observed. Discrepancy between results of WBCT readings was observed in only one of these three patients (case no. 5): WBCT20 was restored at H_3, WBCT30 at H_6, and bleeding resolved at H_{12}. Tests were repeated using a new set of tubes in all three cases to confirm the peculiar results. Finally, in the remaining 3/17 patients (case no. 6, 8, and 18) normal coagulation was restored permanently and completely by H_3 following a single dose of antivenom at H_0.

Differences noted during initial assessment (Group 1) presented as variations in severity of coagulopathy (one gradation of WBCT scoring on the scale detailed in methods) rather than entirely contradictory results; two patients (case no. 4 and 15) exhibited a coagulopathy that improved by one grade between 20 and 30 min and the third patient (case no. 17) had a friable clot at 20 min that was completely incoagulable by 30. In six of seven cases, the two tests did not normalize simultaneously, WBCT20 was corrected earlier than WBCT30. Resumption of coagulopathy was observed in a total of seven patients with *E. ocellatus* envenomations and five of them exhibited discrepancies in WBCT results (Groups 3a and 3b; Table 5).

In two patients (cases no. 1 and 12, Fig. 3) there was a transient resumption of WBCT30 in the first 24 h that was inconsistent with the clinical picture of improvement observed at the time of the test, and there was no resumption of WBCT20 in either case. In the other five patients, a resumption of coagulopathy coincided with a resumption of envenomation signs and symptoms (gingival bleeding, internal bleeding, pain, edema, etc.) and therefore fit the diagnosis of a recurrent envenomation. In one of these patients (case no. 10), WBCT30 detected the recurrence of envenomation 21 h before it was detected by WBCT20 and coincided with the earliest symptoms of a renewed subarachnoid hemorrhage.