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From Bite to Bleed-out: Managing Vasculotoxic Snakebite Induced Catastrophic Complications in Resource Constrained Settings

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Abstract

Snake bite envenomation is a serious but under-reported disease in sub-Saharan Africa, especially among the rural population who frequently experience catastrophic complications. The management of these complications can be challenging for the attending physician in resource constrained settings. Herein, is a case of a 27-year-old farmer referred to our hospital with two weeks history of snakebite on his left thumb. Three days after the bite, he presented to a rural hospital with spontaneous bleeding from the gum and at the site of a previous traumatic foot ulcer which resulted in a transfusion of seven pints of blood but no anti-snake venom therapy. Nine days after spontaneous gum bleeding, he developed progressive abdominal swelling and passage of melena at the local hospital which necessitated his referral.

At the presentation, his bedside whole blood clotting time was more than 20 minutes. Abdominal paracentesis yielded non-clotted hemorrhagic aspirate, pack cell volume was 18% and abdominopelvic ultrasound revealed moderate to severe intra-abdominal hemorrhage. Coagulopathy was not reversed despite therapy with the only available 200 mls of polyvalent anti-snake venom and five days later, he received one vial (10mls) of EchiTab-plus, the only available specific anti-snake venom procured far away at an outrageous cost. Besides, he got three pints of blood, prophylactic antibiotics, close monitoring, and reassurance. He archived the reversal of coagulopathy with a favorable clinical outcome. In sub-Saharan Africa, without, access to appropriate anti-snake venom in our health facilities, severe complications may result in rapid death.

Keywords

Vasculotoxic snake-bite; Catastrophic complications; Coagulopathy; Intra-abdominal hemorrhage; Gastrointestinal bleeding; EchiTab-plus

Abbreviations

- **MOD:** Multiple organ dysfunction
- **WBCT:** Whole blood clotting time
- **FDP:** Fibrin degradation products
- **ED:** Emergency department
- **PCV:** Pack cell volume
- **GI:** Gastrointestinal bleeding

Introduction

Snakebite envenoming in sub-Saharan Africa is a neglected and poverty-related disease with significant loss of productivity, health care cost, catastrophic complications, morbidity and mortality [1-5]. Venomous snakebite appears to be a double tragedy in sub-Saharan Africa because it is related to poverty and it occurs in a region with extreme poverty and limited resources, making the prognosis dismal. The main factors contributing to poor clinical outcomes in snakebite envenomation in sub-Saharan Africa are delayed hospital presentation, unavailability and high cost of anti-snake venom, health care costs, poor health care and storage facilities, counterfeit anti-snake venom, poverty, illiteracy, limited health care access in remote areas of the region and poor public awareness (were most affected patient always resort to trade-medicine before presentation to hospital) and the false public believes in snake charmers [1,4-6]. Snakebite envenoming in sub-Saharan Africa is mainly a rural problem and most people at risk are subsistent farmers, herdsmen, hunters, snake charmers, and plantation workers with most bites occurring before the rainy season and during heavy rain [1,4,5]. In view of the aforementioned observations, management of snake bite envenomation in sub-Saharan Africa is challenging.

Clinico-pathologically, snakebite envenoming is categorized into three on the basis of the body system involved into myotoxic, neurotoxic, and vasculotoxic (hematoxic). The vasculotoxic snakebite envenoming in sub-Saharan Africa is commonly caused by the Viperidae family of snakes which include *Echis ocellatus* [2,4,5]. The venom is a complex secretion that contains high molecular weight enzymes ranging with molecular weight ranging between 13-150 Kilo Daltons (KDa) which form 80-90 percent of the viper's

venom, polypeptide toxins with a molecular weight of 5-10kDa, and low molecular weight compounds with a molecular weight of less than 1.5kDa [5]. The enzymes include serine proteases (procoagulants of viper venoms, zinc metalloproteinases) and hemorrhagins, while *Echis* venom contains activators of factor X and prothrombin hemorrhagins, a prothrombin-activating procoagulant, phospholipase A2, and hyaluronidase [4,5,7,8]. In vasculotoxic snakebite envenoming, incoagulable blood and hemorrhage arise from venom anticoagulants, consumptive coagulopathy, thrombocytopenia, platelets dysfunction, primary fibrinolysis, vascular wall damage by hemorrhagins and rupture or necrosis of intra-abdominal organ such as the intestine, liver, and spleen [4,5,7-9,10]. All these combines to cause life-threatening hemorrhage, whereas biogenic amines and phospholipase A2 induce local swelling [4,5].

Clinically, the initial manifestation of vasculotoxic snakebite envenoming is local pain and progressive swelling, followed by coagulopathy which presents as continuous bleeding (>20 minutes) from the fangs puncture wound, venipuncture sites, and previously partially healed wounds. These are the first clinical evidence of coagulopathy followed by spontaneous systemic hemorrhage which manifests as gingival bleeding, most often detected in the gingival sulci, epistaxis, and hematuria (asymptomatic or total) detected a few hours after the bite [4,5]. Other forms of spontaneous bleeding are subconjunctival hemorrhage, intracranial hemorrhage in the form of intracerebral or subarachnoid hemorrhage (manifesting with restlessness, irritability, loss of consciousness, meningism, cerebral thrombosis, hemiplegia or hemiparesis), bleeding into the floor of the mouth and tympanic membrane, gastrointestinal bleeding presenting as (melena or hematochezia), ecchymoses, petechiae, discoid/follicular hemorrhage and the anterior pituitary hemorrhage (which mimic Sheehan's syndrome) [4,5,7,8,11].

A rare and life-threatening intra-abdominal hemorrhage in the form of hemo-retro-peritoneum, hemoperitoneum, or both, and gastrointestinal bleeding from vasculotoxic snakebite has been reported in the literature [2,3,9,10]. Other rare complications associated with vasculotoxic snakebite envenoming include hemoptysis, endocrinopathies such as stress-induced hyperglycemia, hypoglycemia and Sheehan's syndrome, shock, cardiotoxicity, multiple organ dysfunction (MOD), acute kidney injury and severe anemia [4,5,7,8,11]. In spite of the aforementioned complications associated with vasculotoxic snakebite envenoming, the clinical severity is determined by the type of snake, the nature, quantity, and degree of toxicity of the snake venom administered, the location of the wound, timing of first-aids and therapies provided, and underlying medical comorbidities [12,13].

We present a case of vasculotoxic snakebite-induced catastrophic complications; coagulopathy, intra-abdominal hemorrhage, gastrointestinal bleeding, and anemia in a subsistent farmer with financial constraints, who have multiple risk factors for snakebite, indices of clinical severity, and predictors of mortality. Despite these and management challenges, the clinical outcome was favorable.

Case report

A 27-year-old subsistent farmer presented to our emergency department (ED) with 2 weeks history of snake bite to his left thumb while clearing the farm. The snake was identified as a carpet viper (*Echis ocellatus*) by the patient and coworkers. Immediately after the bite, severe pain and fang marks with

minimal hemorrhage occurred at the site, which he overlooked without seeking for trado-medicine or medical attention. However, over time, progressive swelling that involved two-thirds of his left forearm was noted. Three days after the bite, he developed spontaneous gum bleeding at the site of a prior traumatic left foot ulcer. Nine days after spontaneous gum bleeding, the patient started passing melena and developed progressive abdominal swelling with associated mild lower abdominal pain. However, no hematuria, hematochezia, petechial hemorrhages, ecchymosis, and differential limb weakness. No history of chronic liver disease, bleeding disorders, diabetes mellitus, acid peptic disease, and chronic kidney disease. On account of the severe gum bleeding, he presented to a nearby general hospital where he was admitted for about 11 days and was transfused 7 pints of blood, but no anti-snake venom therapy prior to referral to our hospital because of unavailability.

General physical examination was unremarkable except for tachypnea, pallor, dehydration, significant peripheral (left axillary) lymphadenopathy, and the site of the bite in the dorsolateral aspect of the left thumb (Figure 1). Cardiovascular examination revealed a pulse rate of 92/minute, Blood pressure of 140/80 mmHg, and heart sounds were loud S1 and S2 only. Respiratory rate was 28 breath/minute with normal breath sound. The abdomen was grossly distended with a girth of 116 cm (Figure 2), moved with respiration, no areas of tenderness and organomegaly difficult to appreciate, and bowel sound was reduced. Diagnostic abdominal paracentesis yielded hemorrhagic non-clotted aspirate (Figure 3) and per rectal examining finger was stained with a black stool. Neurological examination was unremarkable. Bedside whole blood clotting time (WBCT) was more than 20 minutes and random blood sugar was 6.5 mmol/L.

Figure 1: Dorsolateral surface of the left thumb showing the site of bite.

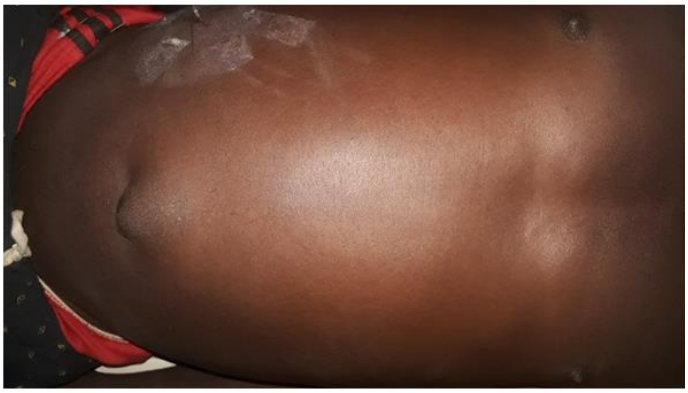

Figure 2: Showing grossly distended abdomen due to intra-abdominal hemorrhage.

Figure 3: Hemorrhagic non clotted aspirate yielded from Abdominal paracentesis.

Diagnosis of venomous snake bite with systemic envenomation: coagulopathy; intra-abdominal hemorrhage; gastrointestinal (GI) bleeding probably upper GI bleeding, and anemia was entertained. Blood samples were taken for investigations that included blood grouping and cross-matched, and the result is shown in Table 1

Table 1: Results of blood investigation.

Investigation	Result	Reference
PCV	18	37 – 45%
WBC	13.6	4-11 X 10^3/dL
Platelets	195	150 – 400 X 103/µL
BUN	2.7	2.5 – 6.5 mmol/L
Serum creatinine	0.8	0.7 – 1.4 mg/dL
Serum sodium	129	135 -149 mmol/L

Serum potassium	4.1	3.5 – 5.2 mmol/L
Aspartate transaminase	33	Upto 12 U/L
Alanine transaminase	91	Upto 12 U/L
Total serum bilirubin	2.49	Upto 1.2 mg/dl
Direct bilirubin	0.14	Upto 0.25 mg/dl
Total serum protein	6.2	6.0 – 8.0 g/dl
Serum albumin	3.9	3.5 – 5.0 g/dl
Urinalysis	Protein 1+	

Abdomino-pelvic ultrasound scan revealed moderate to severe intra-abdominal collection in the entire abdominal quadrant, presumably due to intra-abdominal hemorrhage. The liver, gall bladder, kidneys, and pancreas were normal in size and echo pattern. The bowels loops are normal in caliber, wall thickness, and peristalsis. No free intraperitoneal fluid collection or mass lesion is demonstrated. The urinary bladder was distended with clear urine, showing a regular outline and normal wall thickness. The sonographic conclusion is suggestive of intra-abdominal hemorrhage.

At the presentation to the ED, anti-snake venom was commenced, and 50 mls of intravenous polyvalent anti-snake venom (COMBIPACK) in 200 mls of normal saline was administered over 2 hours after a negative test dose. Repeated doses were given which totaled 200 mls in 24 hours, procured at an exorbitant price. The six-hourly bedside whole blood clotting time (WBCT) in the first 24 hours and beyond was more than 20 minutes, despite receiving 200 mls of polyvalent anti-snake venom. Five days after therapy with COMBIPACK, one vial (10mls) of EchiTab-plus, the only available specific anti-snake venom sourced from far away at an outrageous cost was administered. He had resolution of coagulopathy as evidenced by WBCT which became less than 20 minutes, cessation of spontaneous gum bleeding, passage of melena, and regression of abdominal girth. He had three pints of fresh whole blood transfused, sourced with difficulty. Other adjunctive therapies instituted included prophylactic antibiotic therapy with ceftriaxone and doxycycline (in view of the intra-abdominal blood clot, a potential culture media for bacteria as well bacteria from snakebite). Fresh frozen plasma and cryoprecipitate were not available in our center. Screening chest X-ray and echocardiography were done for possible hemothorax, and hemopericardium, which were essentially normal. ECG was also normal. Abdominal CT scan showed moderate intraperitoneal collection presumably hemoperitoneum, mild splenomegaly, and thickened gastric wall (Figures 4 and 5).

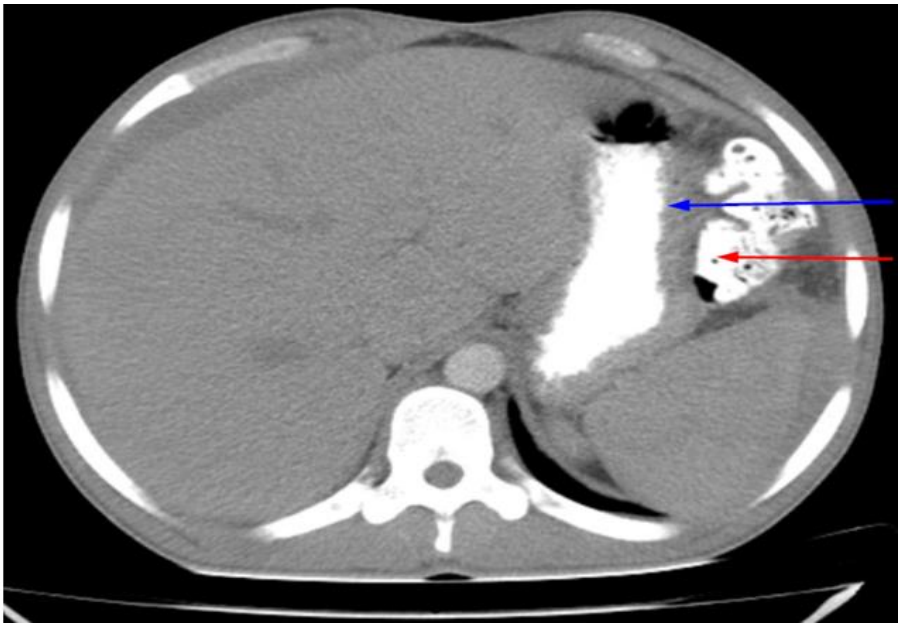

Figure 4: Axial post contrast abdominal CT image of the patient showing hemoperitoneum (red arrow) and thickened gastric wall (blue arrow).

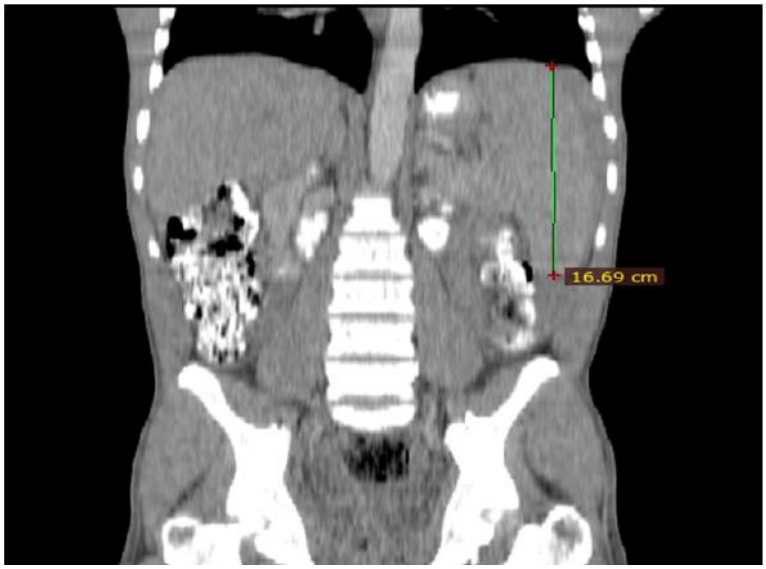

Figure 5: Reformatted post contrast coronal abdominal CT image of the patient showing an enlarged spleen.

All the requested investigations were fully funded by the hospital management, while medications and blood transfusion were partly funded by hospital management because of patient financial constraints. Our index case achieved remarkable improvement as evidenced by the normalization of the WBCT, cessation of gum and gastrointestinal bleeding, and regression of abdominal distention. The patient was discharged home after 9 days of hospital stay with post-transfusion PCV of 31% and was given a 2-week follow-up appointment. Follow-up abdominopelvic ultrasound scan showed resolution of intra-abdominal

hemorrhage, with an abdominal girth of 84 cm and normal WBCT with satisfactory clinical status and outcome.

Discussion

Vasculotoxic snakebite-induced coagulopathy, intra-abdominal and gastrointestinal bleeding are all life-threatening medical emergencies that require access to well-equipped hospitals, prompt identification, timely administration of anti-snake venom, and adjunctive therapies to avert dismal prognosis. Few examples of this clinical presentation have been documented in the literature, and all of them originated in the tropical and subtropical regions of the world [2,3,9,10], with half of these cases reported in resource-constrained regions of the world [2,3]. Our index case is a young man with identifiable risk factors for snakebite that include young age, subsistent farmer, rural dwelling, male gender, and subtropical location [1,4,5,14]. These risk factors for snakebites were equally observed in the case reported by Yakubu et al [2].

Medically important snakes causing life-threatening coagulopathy and hemorrhages in sub-Saharan Africa are two Viperoid species: Saw-scaled (*Echis ocellatus*) and Russell's vipers [2,4,5]. The viperoid species responsible for the bite in our patient is *Echis ocellatus*. This specie of snake is widely found in Nigeria and sub-Saharan Africa causing rare but catastrophic complications as earlier reported by Yakubu et al [2]. Similar complications have been associated with other viperoid species including Russell viper as reported by Rothold et al [3], and pit viper as reported by Kang et al and Ahn et al [9,10]. The bite in our patient occurred on the left hand while clearing farmland during the day in the dry season which is in contrast to the case reported by Yakubu et al [2]. The indices of clinical severity of snakebite envenoming in our patient at presentation included viperoid species, vasculotoxity, probably the nature and quantity of the snake venom injected, site of the bite (hand) and delay in seeking first-aids in the first 72 hours and specific therapy after 2 weeks [12,13].

Gum bleeding was the earliest systemic manifestation of carpet viper-induced coagulopathy in our patient, similar to the case reported by Yakubu et al [2]. On admission, we also considered the possibility of consumptive coagulopathy in our patient. In view of the persistently prolonged whole blood clotting time (WBCT), exceeding 20 minutes, and the potential signs of fatal hemorrhages. However, the lack of serum assays for fibrin degradation products (FDP) and fibrinogen in our center prevented this diagnosis from being made. Another possible explanation of gum bleeding in our patient is venom-induced thrombocytopenia or platelet dysfunction. However, our patient had an adequate platelet count and no other clinical features suggestive of thrombocytopenia or platelet dysfunction, such as purpura, petechiae hemorrhages, ecchymosis, and a history of easy bruisability. Therefore, platelet disorders are unlikely to be the cause of gum and intra-abdominal bleeding in our patient. Even-though, test for platelet dysfunction is not available in our hospital.

The rare but catastrophic complications of vasculotoxic envenoming, such as severe intra-abdominal hemorrhage (manifesting as progressive abdominal swelling), and gastrointestinal bleeding (manifesting as melena), as seen in our index case, are similar to that reported by Yakubu et al [2]. These complications can lead to severe anemia, shock, multiple organ dysfunction (MOD), and ultimately, death. The

pathophysiologic mechanisms of intra-abdominal hemorrhage and gastrointestinal bleeding are numerous, and this includes venom-induced consumptive coagulopathy, hemorrhagin-induced direct mesenteric vascular endothelial injury, and phospholipase A2-induced vascular endothelial injury [15]. Rupture or necrosis of intra-abdominal organs including the intestine, liver, and the spleen are most important causes of intra-abdominal bleeding in vasculotoxic envenoming [3,10,16]. The site of intra-abdominal hemorrhage from vasculotoxic snakebite in our patient may be established by celiac angiography or computed tomography angiographic scan which the former is not available in our center. Clinically, intra-abdominal hemorrhage from vasculotoxic snakebite presents as hemo-retroperitoneum or hemoperitoneum, or both as reported in the literature [3,10,15,16]. In this regard, patient with snakebite envenoming with coagulopathy in sub-Saharan Africa should undergo close monitoring of abdominal girth. Emergency and follow-up abdominopelvic ultrasound are recommended, along with screening chest X-rays and echocardiography, when possible, to access for hemothorax and hemopericardium, as advised by Tchaou et al [17].

The predictors of mortality in our patient were bleeding tendencies [12], delayed presentation to the hospital most likely due to illiteracy, low disease awareness, and poverty along with failed prompt referral and delayed administration of specific therapy [13]. In spite of the aforementioned predictors of mortality, besides financial constraints and treatment challenges, the clinical outcome was favorable. Therefore, this case scenario can serve as a valuable learning experience for healthcare workers in sub-Saharan Africa. It highlights the importance of identifying and predicting clinical outcomes in high-risk cases of snakebite envenoming's. This information can be used to facilitate immediate referrals to well-equipped health centers with expertise in snakebite management and access to appropriate anti-snake venom, thereby averting potentially dismal prognoses.

The major therapeutic challenge encountered in this case is the apparent ineffectiveness of the administered 200 mls COMBIPACK anti-snake venom, albeit at a high cost. Similarly, this ineffectiveness of COMBIPACK in reversing coagulopathy was also noted previously in the literature [18,19]. Other possible reasons for the ineffectiveness of this polyvalent anti-snake venom include intraspecies variation in snake venom composition and antigenicity [20], lack of evidence for optimal dosing schedule [21], poor storage, fake or expired anti-snake venom [20,21]. In view of these, the use of this anti-snake venom (COMBIPACK) to treat Carpet viper envenoming may not be cost-effective in our region.

The most interesting observation in our patient management the efficacy of a single vial of EchiTab-plus in reversing coagulopathy and improving clinical status [22]. Without this intervention, the clinical outcome could have been fatal. The efficacy of EchiTab-plus in reversing carpet viper-induced coagulopathy was also noted by Yakubu et al [2]. Therefore, EchiTab-plus may be the anti-snake venom of choice, and currently, EchiTab-plus therapy is the only cost-effective and available therapeutic option to reverse carpet viper-induced coagulopathy and intra-abdominal hemorrhage in our patient. Although celiac angiography-guided splenic artery embolization (SAE), hepatic artery embolization (HAE) [3], and splenectomy [10] have been as alternative treatment interventions for intra-abdominal hemorrhage in the literature, these these endovascular interventions are not available in our region.

Conclusion

To avert fatal clinical outcomes in a patient with vasculotoxic snakebite-induced catastrophic complications in Sub-Saharan Africa, there is a need for community health awareness, as well as advocacy to the community leaders and the traditional healers on the need for prompt reporting of cases of snakebite. Furthermore, attending physicians in sub-Saharan Africa should anticipate challenges in the management of vasculotoxic snakebite-induced catastrophic complications, besides the use of potent anti-snake venom such as the EchiTab plus. Therefore, our health workers in peripheral health centers should be reoriented to indentify indices of clinical severity and predictors of mortality in any patient with snakebite envenomation. They should immediately refer such patients to centers with expertise in snakebite management and access to specific anti-snake venom. Ideally, this anti-venom should be administered free of charges, as the clinical outcome can be fatal without it.

Learning points

Finally, the learning points in this case report are as follows: Carpet viper (*Echis ocellatus)* induced catastrophic complications are rare but associated with management challenges. Timely administration of potent anti-snake venom such as EchiTab-plus may be necessary to avert dismal prognosis; In sub-Saharan Africa, patients with vasculotoxic snakebite-induced catastrophic complications usually present with multiple risk factors for snakebite, indices of clinical severity and predictors of mortality; Patient with vasculotoxic snakebite envenoming should have screening imaging for internal bleeding that include abdominopelvic ultrasound, chest X-ray and echocardiography; To avert fatal clinical outcome from snakebite envenoming in sub-Saharan Africa, there is need for community health education and awareness programs about snakebite.

Data availability

The data of this case study are available with the corresponding author on reasonable request

Conflicts of interest

None declared.

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Consent to participate

The authors certify that they have obtained patient consent forms. In the form, the patient has given his consent for his clinical information and images to be reported in a journal. The patient understands that his name and initials will not be published in a journal.

Authors Contributions

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2. Administrative support: Hayatu U, Faruk B, Akintomide AA, Aminu A; Ibrahim AA;
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4. Collection and assembly of data: Hayatu U, Akintomide AA, Ibrahim AA, Shamsuddeen AA, Faruk KU, Aminu A, Faruk B;
5. Data analysis and interpretation: Hayatu U, Akintomide AA, Shamsuddeen AA, Faruk KU, Aminu A, Faruk B Ibrahim AA;
6. Manuscript writing: All the authors
7. Final approval of manuscript: All the authors.

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