INTRODUCTION
Snakebite is an occupational hazard mainly in the rural communities. About 410 of approximately 3000 species of snakes that exist worldwide are considered venomous.[1] Globally, about 1.2–5.5 million snakebites occur worldwide with at least 421,000 envenomation. It may account for as high as 94,000 deaths yearly.[2] The burden of snakebite is highest in Southeast Asia, South and Central America, and Sub-Saharan Africa.[2] The incidence of snakebite injury is 497/100,000 population with a case fatality rate of 12.2% in Northeast Nigeria.[3]

The spectrum of presentation may vary from mild local symptoms to rapid envenomation leading to sudden death. Acute kidney injury (AKI) and tissue necrosis are possible complications of viperine snakebite.[3,4] The incidence of AKI in hospitalized patients with snakebite in Nigeria was reported to be between 1% and 10% and it is associated with increased mortality.[4,6]

We report a case of 73-year-old rural male farmer who had digital gangrene and AKI following a vipersnakebite. He was managed for left digital gangrene and AKI, but did not have complete renal recovery.

CASE REPORT
The patient is a 73-year-old male farmer who resides in a rural community in Ondo State, Southwest Nigeria. He was referred to our facility on account of deranged electrolyte, urea, and creatinine 12 days following viperine snakebite. There was delay before presenting for medical care. He had amputation of the gangrenous digit and was managed with haemodialysis. He had incomplete renal recovery with an estimated glomerular filtration rate of 23 ml/min/1.73m2 4 months after discharge. Delay in seeking appropriate medical care in patients with snakebite injury may be associated with significant morbidity.

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Worsening of the symptoms made him present 24 hours later at a secondary healthcare facility where he was treated with polyvalent antivenom and had a left forearm fasciotomy on account of compartment syndrome. There was no bleeding from any orifice. He was later noticed to be passing dark-colored (cola) urine with reduced urinary output. He was not a previously diagnosed hypertensive or diabetic.

At presentation in our facility, he was conscious but confused. His pulse rate was 84 beats/min and blood pressure was 170/110 mmHg. The left index finger was also found to be gangrenous [Figure 1]. Examination of other systems was essentially normal. Urinalysis showed proteinuria (4+), hematuria (2+), and urobilinogen (2+). A diagnosis of left digital gangrene and AKI following snakebite injury was made.

**Investigations**

Urine microscopy showed numerous isomorphic red blood cells and granular casts, packed cell volume of 24%, total white cell count of 4,900 cells/mm³ with normal differential count, platelet count of 221,000 cells/mm³, serum creatinine of 1.243.9 µmol/L, urea of 58.8 mmol/L, sodium of 135.5 mmol/L, chloride of 110.8 mmol/L, potassium of 7.3 mmol/L, and bicarbonate of 9 mmol/L. The prothrombin time, activated thromboplastin time, and renal scan were normal. Liver function test was essentially normal except the total serum bilirubin that was slightly elevated (51 µmol/L).

He was transfused with blood and commenced on antibiotics and antihypertensives. The patient had amputation of the gangrenous left index finger. He had six sessions of hemodialysis before he was discharged. He is presently being followed in outpatient clinic and his last estimated glomerular filtration rate (GFR) was 23 ml/min/1.73 m² 4 months after discharge.

**DISCUSSION**

Snakebite is an occupational hazard faced by farmers, farm laborers, and hunters in tropical countries as seen in this index patient. This patient was bitten by a snake which belongs to the viperidae family. The venom of this snake has been found to contain a prothrombin-activating procoagulant, hemorrhagic, and cytolytic fractions which could cause hemorrhage, shock, local reactions, and necrosis.[4]

The kidneys are well vascularized; hence, they are vulnerable to snake venom injury that may lead to AKI.[7] The spectrum of renal injury following snakebite may include acute tubular necrosis, acute cortical necrosis, acute interstitial nephritis, mesangiolysis, glomerulonephritis, and vasculitis.[7-9] The predictors of AKI in patients with snakebite are old age, the presence of proteinuria, hypotension, evidence of hemolysis, anemia, dark-colored urine, late presentation, and late antivenom treatment.[10,11] All these factors were present in this patient except hypotension.

There are various pathogenetic mechanisms that may lead to AKI in patients with snakebite such as hypotension, disseminated coagulopathy, cytokine-mediated injury, pigment nephropathy, and direct nephrotoxicity by the snake venom.[7,12] This patient had cola-colored urine which may be due to hemolysis or rhabdomyolysis. Markers of hemolysis and rhabdomyolysis were not done due to financial constraint. However, the presence of elevated serum bilirubin, urobilinogen in urine, and low hematocrit may be suggestive of hemolysis. He had hematuria which was composed of isomorphic red cells on microscopy. This may be possibly explained by acute tubular necrosis which is common in AKI following snakebite[9] and not coagulopathy which was not evident at the time of referral to us.

This patient had gangrene of left index digit which was the site of the snakebite. The gangrenous digit might have resulted from delayed treatment with antivenom as well as the compartment syndrome of the affected limb although he had fasciotomy. Toxins of viperinae are known to be necrotic which may cause tissue necrosis and gangrene.[4]
Hemodialysis as a mode of renal replacement modality used in the management of this patient has been reported to be associated with better outcome compared to peritoneal dialysis. There are two possibilities in this patient based on his clinical course and estimated GFR of 23 ml/min/1.73 m² 4 months after discharge. The first possibility is that he could have had acute on chronic kidney disease precipitated by the snakebite injury while the second is that he had incomplete renal recovery following AKI caused by snakebite injury which has progressed to CKD. Long-term follow-up of patients who had snakebite induced AKI showed that a significant proportion had features of persistent renal damage. This patient may require renal replacement therapy later, which he may not be able to afford due to his low socioeconomic status.

This patient survived possibly because some factors that are significant predictors of mortality in snakebite such as respiratory failure, shock, and bleeding tendencies were absent in him even though he had AKI.

CONCLUSION
Delay in seeking appropriate medical care in patients with snakebite injury may be associated with significant morbidity as seen in this patient.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES