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Renal involvement in patients with visceral leishmaniasis

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Abstract
Visceral leishmaniasis (kala-azar) is a chronic and potentially fatal parasitic disease. It has been determined that renal involvement can lead to glomerular and tubular dysfunction.
This study aimed to assess renal function in Sudanese patients with visceral leishmaniasis by measuring plasma urea and creatinine levels
Methods: A case control study was conducted at Gadarif Teaching Hospital in Eastern Sudan. Thirty patients with confirmed visceral leishmaniasis and twenty healthy matched controls were enrolled. Plasma levels of urea and creatinine were measured for all participants at enrollment, for the patient group measurements were repeated after treatment with sodium stibogluconate.
Results: Plasma creatinine and urea levels were significantly higher (p<0.001) in untreated visceral leishmaniasis patients (1.38±.44 mg/dl and 28.09±8.80 mg/dl respectively). Compared to healthy controls (0.90±0.26 mg/dl and 21.86±5.43 mg/dl).Following treatment these levels decreased significantly (p<0.001) to 87±35 mg/dl (creatinine) and 15.5±.82 mg/dl (urea). Post treatment creatinine levels were not significantly different from controls (p<0.05) while urea levels were significantly lower (p<0.05) but remained within the normal range.
Conclusion: Kala-azar causes significant but reversible renal impairment, as evidenced by elevated urea and creatinine levels that normalize with successful treatment. Monitoring renal function is crucial in the management of VL.

Keywords: Sudan, Kala-azar, visceral leishmaniasis, renal impairment, urea, creatinine, sodium stibogluconate

Introduction
Leishmaniasis is a group of illnesses brought on by protozoan parasites belonging to the Leishmania genus, transmitted by the bite of infected female phlebotomine sandflies. Visceral leishmaniasis (VL), also known as kala-azar, is the most severe form of the disease and is fatal if left untreated. [1] It is caused primarily by *Leishmania donovani* and *L. infantum*, Over 90% of all VL occurrences globally occur in six countries Ethiopia, Brazil, Bangladesh, Sudan, South Sudan, and India [2]. VL is still a major public health issue in Sudan, especially in the savannah regions of the eastern and central parts of the country [3]. The parasitization of reticuloendothelial cells is a characteristic of the illness, resulting in typical symptoms like fever, pancytopenia, hepatosplenomegaly, and weight loss [4]. In addition to these systemic impacts, renal involvement is a frequent and dangerous side effect that raises the overall morbidity and mortality rate of VL [5]. The development of nephropathy related to kala-azar is complex, involving the accumulation of immune complexes in the glomeruli, widespread activation of B-cells, and a series of inflammatory responses [6, 7]. This can lead to glomerulonephritis and renal tubular dysfunction, which may manifest clinically as proteinuria, hematuria, and in more severe instances, either acute or chronic renal failure [5, 8]. Additionally, the nephrotoxic effects of primary anti-leishmanial medications, like pentavalent antimonials, may worsen kidney damage [9].
Although renal dysfunction in VL has been documented in various studies [8, 10], data specific to the Sudanese population are limited. Therefore, this study aimed to evaluate renal function by measuring plasma urea and creatinine levels in Sudanese patients with visceral leishmaniasis before and after treatment with sodium stibogluconate.

2. Materials and Methods

2.1 Study Design and Subjects

A case-control study was conducted at the Kala-azar ward of Gadarif Teaching Hospital in Eastern Sudan from March to May 2004. The case group consisted of thirty patients with parasitologically confirmed VL. A control group of twenty healthy individuals, matched for age and sex, was recruited from the community. Informed consent was obtained from all participants prior to their inclusion in the study.

2.2 Diagnosis of Visceral Leishmaniasis

Diagnosis was confirmed by the microscopic identification of Leishmania donovani amastigotes in Giemsa-stained smears prepared from lymph node or bone marrow aspirates, examined under an oil immersion lens [11].

2.3 Blood Sample Collection and Analysis

Three milliliters of venous blood were drawn from the antecubital vein of each participant into heparinized vacutainers. For patients, samples were collected at the time of diagnosis (pre-treatment) and after completion of therapy (post-treatment). Plasma was separated by centrifugation and stored at -20 °C until analysis. Plasma urea concentration was determined using an enzymatic colorimetric method based on the Berthelot reaction.Plasma creatinine was measured using the alkaline picrate (Jaffe) method, as described by Monica (1992) [12].

2.4 Treatment

Patients were treated with sodium stibogluconate (Pentostam®) at a standard dose of 20 mg/kg body weight daily.

2.5 Statistical Analysis

Data are presented as mean ± standard deviation (SD). Statistical comparisons between groups were performed using Student's t-test. A p-value of < 0.05 was considered statistically significant. All analyses were performed using SPSS software

3. Results

The results of the plasma creatinine and urea analyses for the different study groups are summarized below

Table 1: Plasma creatinine and urea levels in untreated kala-azar patients and healthy controls.

Parameters	Untreated Kalaazar patients	Control	P-Value
Creatinine (mg/dl)	1.38 ±0.44	0.90 ±0.26	$p<0.001$
Urea (mg, dl)	28.09± 8.80	21.86±5.43	$p<0.001$

As shown in Table 1, the mean plasma levels of both creatinine and urea were significantly elevated in patients with untreated VL compared to the healthy control group ($p<0.001$ for both).

Table 2 demonstrates that following treatment with sodium stibogluconate, there was a highly significant reduction in the mean plasma levels of both creatinine and urea compared to pre-treatment levels ($p<0.001$).

Table 2: Plasma creatinine and urea levels in kala-azar patients before and after treatment.

Parameters	Pretreatment	Post treatment	P-Value
Creatinine (mg/dl)	1.38 ±.44a	0.87±0.35b	$p<0.001$
Urea (mg/dl)	28.09±8.80a	15.5±0.82b	$p<0.001$

Table 3: Comparison of plasma creatinine and urea levels in treated kala-azar patients and healthy controls.

Parameters	POS treatment	Control	P-Value
Creatinine (mg/dl)	0.87±0.35	0.90±0.26	($p<0.05$)
Urea (mg, dl)	15.5±0.82	21.86±5.4	($p<0.05$)

As shown in Table 3, after treatment, the mean plasma creatinine level in patients was not significantly different from that of the healthy controls ($p<0.05$). The mean plasma urea level in treated patients was significantly lower than in controls ($p<0.05$) but well within the normal physiological range.

4. Discussion

This study demonstrates a significant impairment of renal function in Sudanese patients with active visceral leishmaniasis, as indicated by elevated plasma levels of urea and creatinine. The findings are consistent with the well-documented phenomenon of kala-azar-associated nephropathy [5, 8].

The significant elevation of urea and creatinine in untreated patients (Table 1) aligns with the results of previous studies [10, 13]. The pathogenesis is largely attributed to immune complex deposition in the glomeruli, leading to glomerulonephritis, and potential direct tubular damage, which disrupts the normal renal filtration and excretion processes [6, 7]. The activation of T-cells and inflammatory cytokines further contributes to this renal injury [7].

The most compelling finding of this study is the significant reversal of renal impairment following successful chemotherapy. The post-treatment levels of creatinine normalized completely, showing no significant difference from the healthy control group (Table 3). This suggests that the glomerular filtration rate, which creatinine is a marker for, was restored to normal after the parasitic load and associated inflammatory responses were eliminated. The significantly lower urea level in treated patients, while still normal, could be related to improved nutritional status or liver function post-recovery, as urea synthesis is dependent on hepatic function.

The successful reversal of renal dysfunction with sodium stibogluconate treatment, as shown in Table 2, is a key clinical insight. It indicates that the renal damage in kala-azar, at least in this cohort, was primarily functional and related to the active disease state rather than permanent structural damage. This finding is supported by other researchers who found normal renal function after treatment [14, 15]. However, it is important to note that certain anti-leishmanial drugs, including antimonials, can be nephrotoxic [9]. The fact that renal function improved despite

this potential toxicity underscores the dominant role of the infection itself in causing renal impairment in our patient group.

A limitation of this study is the lack of urinalysis data (e.g., proteinuria, hematuria) to correlate with the biochemical findings. Future studies would benefit from a more comprehensive renal workup, including urinary parameters and more sensitive biomarkers of glomerular and tubular injury.

5. Conclusion

This study concludes that visceral leishmaniasis (kala-azar) causes significant but largely reversible renal impairment in Sudanese patients, as evidenced by elevated plasma urea and creatinine levels. The successful treatment with sodium stibogluconate effectively normalizes these parameters, highlighting the importance of timely diagnosis and treatment. Monitoring renal function using simple biochemical tests like urea and creatinine is crucial in the management of kala-azar to assess disease severity and response to therapy.

6. Author Contributions

Z.E.N: Conceptualization, Data duration, Formal analysis, Investigation. M.E.E: Writing-original draft, Writing-review & editing, Methodology, Project administration. A.I: Resources, Validation. K.E.A.S: Supervision, Resources. All authors have read and agreed to the published version of the manuscript.

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Conflict of Interest

Not available

Financial Support

Not available

References

1. Magill AJ. Leishmania species: Visceral (kala-azar), cutaneous, and mucosal leishmaniasis. In: Mandell GL, Bennett JE, Dolin R, editors. Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. 7th Ed. London: Churchill Livingstone; 2009, p. 3463-80.
2. Alvar J, Vélez ID, Bern C, *et al*. Leishmaniasis worldwide and global estimates of its incidence. PLoS One. 2012;7(5):e35671.
3. El-Safi SH, Bucheton B, Kheir MM, Musa HA, El-Obaid M, Hammad A, *et al*. Epidemiology of visceral leishmaniasis in Atbara River area, eastern Sudan: the outbreak of Barbar El Fugara village (1996-1997). Microbes Infect. 2002;4(14):1439-47.
4. Mandell GL, Bennett JE, Dolin R. Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. 7th ed. Philadelphia (PA): Churchill Livingstone Elsevier; 2010.
5. Clementi A, Battaglia G, Floris M, Castellino P, Ronco C, Cruz DN. Renal involvement in leishmaniasis: A review of the literature. NDT Plus. 2011;4(3):147-52.
6. Costa FA, Prianti MG, Silva TC, Silva SM, Guerra JL, Goto H. T cells, adhesion molecules and modulation of apoptosis in visceral leishmaniasis glomerulonephritis. BMC Infect Dis. 2010;10:112.
7. Prianti MG, Yokoo M, Saldanha LCB, Costa FAL, Goto H. Leishmania (Leishmania) chagasi-infected mice as a model for the study of glomerular lesions in visceral leishmaniasis. Braz J Med Biol Res. 2007;40(6):819-23.
8. Dutra M, Martinelli R, Carvalho DEM, *et al*. Renal involvement in visceral leishmaniasis. Am J Kidney Dis. 1985;6(1):22-7.
9. Schwarz A, Perez-Canto A. Nephrotoxicity of antiinfective drugs. Int J Clin Pharmacol Ther. 1998;36(3):164-7.
10. Mustafa EM. Changes in selected biochemical parameters of renal function in Sudanese patients with kala-azar [MD Thesis]. Khartoum: University of Khartoum; 1998.
11. Manson P, Bell DR. Manson's Tropical Diseases. 19th ed. London: Baillière Tindall; 1987.
12. Monica C. A Textbook of Medical Laboratory Technology. 1st Ed. New Delhi: Jaypee Brothers; 1992.
13. Salgado Filho N, Ferreira TM, Costa JM. Involvement of the renal function in patients with visceral leishmaniasis (kala-azar). Rev Soc Bras Med Trop. 2003;36(2):217-221.
14. Balsan M, Fenech F. Acute renal failure in visceral leishmaniasis treated with sodium stibogluconate. Trans R Soc Trop Med Hyg. 1992;86(5):515-6.
15. Lima Verde EM, Lima Verde FAA, Lima Verde FA, *et al*. Evaluation of renal function in human visceral leishmaniasis (kala-azar): A prospective study on 50 patients from Brazil. J Nephrol. 2007;20(4):432-438.

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