

Hydrocarbon's Chronic Kidney Disease

Pierre Eric Gandzali-Ngabe^{1*}, Richard Loumingou², Hamadoun Yattara³, Donatien Moukassa¹

¹HGELBO Internal Medicine Service, Oyo, The Republic of the Congo

²Department of Nephrology CHU of Brazzaville, The Republic of the Congo

³Department of Nephrology and Hemodialysis of Point G, Bamako, Mali

Email: *ngabe_eric@yahoo.fr

How to cite this paper: Gandzali-Ngabe, P.E., Loumingou, R., Yattara, H. and Moukassa, D. (2020) Hydrocarbon's Chronic Kidney Disease. *Open Journal of Nephrology*, 10, 18-22.

<https://doi.org/10.4236/ojneph.2020.101003>

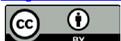
Received: December 25, 2019

Accepted: February 7, 2020

Published: February 10, 2020

Copyright © 2020 by author(s) and Scientific Research Publishing Inc. This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).

<http://creativecommons.org/licenses/by/4.0/>



Open Access

Abstract

Hydrocarbons are dangerous for health, and American Society of Nephrology has already described renal involvement by Hydrocarbon. The Republic of Congo produces oil but there is no study among these working with hydrocarbon to evaluate their renal status. The patient of 39 years working for 5 years as carrier oil, with high exposure to oil or more than 2 hours per day. He has no history of herbal medicine, no edema, no diabetes, no high blood pressure and he was not suffering from infectious diseases and there was no concept of kidney disease in the family. He was hospitalized for febrile generalized tonic convulsions with impaired renal function. After patient stabilization, clinical and biological examinations allowed us to conclude the chronic glomerulonephritis syndrome in five stages of chronic kidney disease. As creatinine at the entrance was 195 mg/l, clearance was 3 ml/min 1.72 m² by CKD-EPI. The entrance to the BUN was 3.57 g/l. The albumin to creatinine ratio of urinary of 300 mg/g (ACR) is a glomerular disease. HIV serology test was negative. The abdominal-pelvic ultrasound revealed dedifferentiated kidneys of normal size. The patient received 7 hemodialysis sessions with femoral right catheter with heparin. The evolution was good and the patient was enrolled in chronic hemodialysis. This interference could be regarded as an occupational disease and we wish early detection.

Keywords

Hydrocarbon, Chronic Kidney Disease, The Republic of the Congo

1. Introduction

The Republic of the Congo is the sixth oil producing country in Africa south of SAHARA [1]. As a result, exposure to inhalation of hydrocarbons by air pollution is reported by populations, although exposure to hydrocarbons is a factor harmful to health [2]. A meta-analysis published in 2000 of 14 case-control stu-

dies devoted to chronic glomerulopathies does not show a causal role (initiator) of organic solvents but suggests an acceleration of the evolution towards the IRT when the pathology is installed: thus, the occupational exposure to solvents appears likely to worsen the nephropathy of hypertensives and diabetics [3]. Exposure to organic (hydrocarbons) solvents has been shown to double the risk of IgA glomerulonephritis and quadruple the risk of extra membranous glomerulonephritis (GEM) [4] [5] [6]. Thus we report a case of chronic glomerulonephritis after exposure to hydrocarbons. This patient from Pointe Noire city is an isolated case in Congolese nephrology practice, although several cases have been reported in the literature [2].

2. Observation

He is a 39-year-old patient working for 5 years as a hydrocarbon transporter, having a high exposure to hydrocarbons, more than 2 hours per week. He had no history of herbal medicine, no edema, no diabetes, no high blood pressure, and he had no infectious diseases and there was no notion of kidney disease in the family.

He was hospitalized for generalized apyretic convulsions with impaired renal function.

On clinical examination after stabilization of the patient with diazepam 10 mg half ampoule intramuscularly, we have:

Discreet edema of the lower limbs keeping the cup with puffiness of the face hyposthenuria, with the urine strip (US):

Proteinuria and hematuria were positive at one to one cross (+), a PH at 6. On the other hand the urinary cylinders, glycosuria, leukocyturia and nitrites were negative.

Blood pressure at 170/110 mmhg or hypertension. The respiratory rate is 20 cycles per minute which is normal.

In biology we have:

The albuminuria to creatininuria ratio at 300 mg/g (ACR) is glomerular involvement. The creatinine level at entry was 195 mg/l, *i.e.* a clearance of 3 ml/Min/1.72 m² according to CKD-EPI.

Entrance blood urea was 3.57 g/l; Calcemia at 63 mg/l; AgHbs: negative; Anti HCV AC: negative; syphilitic serology was negative; the cholesterol/HDL ratio at 4.2 with standards < 5; the LDL-Cholesterol/HDL Cholesterol ratio at 2.9 is normal with standard < 3.5; the HbA1 at 4.8% is normal with standard between 4% to 6%; fasting blood sugar was 1.01 g/l.

And the hemogram showed: Hb at 7.7 g/dl, VGM = 91 (Normal) and CCMH = 29.1 (normal) or Normochromic Normocytic anemia

HIV retroviral serology has been negative.

The abdominopelvic ultrasound revealed normal size, dedifferentiated kidneys.

The patient received 7 sessions of hemodialysis with a right femoral catheter with heparin.

The evolution is marked afterwards by the disappearance of edema and a satisfactory blood pressure balance and a drop in urea to 1 g/l and the creatinine level to 23 mg/l, *i.e.* a clearance of 40 ml/Min/1.73 m² according to CKD-EPI. The patient was enrolled in chronic hemodialysis.

3. Discussion

A meta-analysis published in 2000 of 14 case-control studies devoted to chronic glomerulopathies does not show a causal role for solvents but suggests an acceleration of the evolution towards IRCT when the pathology is installed: thus, the solvents appear susceptible to aggravate the nephropathy of hypertensives and diabetics [3].

In a French case-control study published in 2007, the risk of progression from Berger disease to the IRCT is doubled in the event of high exposure to solvents [4]. Exposure to organic solvents affects the functioning of the kidneys, this has been a hypothesis since 2007 issued by the American Society of Nephrology [2] [3].

In fact, the GN-PROGRESS study described that exposure to organic solvents gave chronic glomerulonephritis and accelerated the onset of end-stage renal disease. Exposure to organic (hydrocarbons) solvents has been shown to double the risk of IgA glomerulonephritis and quadruple the risk of extra membranous glomerulonephritis (GEM) [4] [5] [6].

In our case, the patient was exposed to hydrocarbons for 5 years with a high exposure either more than 2 hours per day or a high exposure and he was immunocompetent. What is found in the GN PROGRESS study.

The Renal Biopsy Puncture (PBR) was not done in our case so histologically we do not know the lesion; however the lesions of GEM, HSF and IgA have been described by several authors [4]-[14]. Hence the importance of establishing the histology of lesions of chronic renal hydrocarbon disease in our environment.

The patient has chronic glomerulonephritis with tubulointerstitial involvement due to the presence of hyposthenuria. This is identical to the observation of Yaqoob M *et al.* [7] who, after post exposure to hydrocarbons, found chronic glomerulonephritis with tubular involvement. This could be explained by the deposits of intra-tubular hydrocarbons. FANCONI syndromes have even been described in the event of exposure to hydrocarbons [15] [16] [17] [18].

4. Conclusion

Congo Brazzaville is an oil producing country; we have many people exposed to hydrocarbons. Apart from this case, we must do early screenings among personnel exposed to hydrocarbons. This study should consist of performing urine test strips, 24-hour proteinuria or albuminuria and creatininemia to have the Albumin Creatinine Ratio, Red Cell-Leukocyte Minutes flow rate, creatininemia and blood urea. Renal or even abdominopelvic ultrasound and Renal Biopsy Puncture are not systematic in the detection and monitoring of renal diseases in

subjects exposed to hydrocarbons. But the biopsy is important to establish what histological lesions are found. We must work with specialists in occupational medicine because this injury could be considered as an occupational disease.

Informed Consent

Patient consent has been obtained.

Thanks

Professor Malick Faye
Doctor Noé Motoula
Professor Saraladevi Naicker
Professor Boucar Diouf

Conflicts of Interest

The authors state that there is no conflict of interest.

References

- [1] Carcillo, S., Leigh, D. and Villafuerte, M. (2007) Catch-Up Growth, Habits, Oil Depletion, and Fiscal Policy: Lessons from the Republic of Congo. IMF Working Paper No. 07/80. <https://doi.org/10.5089/9781451866445.001>
- [2] Jacob, S., Hery, M., Protois, J.C., *et al.* (2007) Effect of Organic Solvent Exposure on Chronic Kidney Disease Progression: The GN-Progress Cohort Study. *Journal of the American Society of Nephrology*, **18**, 274-281. <https://doi.org/10.1681/ASN.2006060652>
- [3] Ravnskov, U. (2000) Hydrocarbons May Worsen Renal Function in Glomerulonephritis: A Meta-Analysis of the Case-Control Studies. *American Journal of Industrial Medicine*, **37**, 599-606. [https://doi.org/10.1002/\(SICI\)1097-0274\(200006\)37:6<599::AID-AJIM4>3.0.CO;2-X](https://doi.org/10.1002/(SICI)1097-0274(200006)37:6<599::AID-AJIM4>3.0.CO;2-X)
- [4] Testud, F. (2010) Maladie de Berger et peinture automobile. *Archives des maladies professionnelles et de l'environnement*, **71**, 946-947. <https://doi.org/10.1016/j.admp.2010.10.007>
- [5] Jacob, S., Hery, M., Protois, J.C., *et al.* (2007) New Insight into Solvent-Related End Stage Renal Disease: Occupations, Products and Types of Solvents at Risk. *Occupational and Environmental Medicine*, **64**, 843-848. <https://doi.org/10.1136/oem.2007.032482>
- [6] Couchoud, C., Stengel, B., Landajs, P., *et al.* (2006) The Renal Epidemiology and Information Network (REIN): A New Registry for End-Stage Renal Disease in France. *Nephrology Dialysis Transplantation*, **21**, 411-418. <https://doi.org/10.1093/ndt/gfi198>
- [7] Yaqoob, M., Stevenson, A., Mason, H., *et al.* (1993) Hydrocarbon Exposure and Tubular Damage: Additional Factors in the Progression of Renal Failure in Primary Glomerulonephritis. *Quarterly Journal of Medicine*, **86**, 661-667. <https://doi.org/10.1093/qjmed/86.10.661>
- [8] Van Der Laan, G. (1980) Chronic Glomerulonephritis and Organic Solvents. A Case-Control Study. *International Archives of Occupational and Environmental*

- Health*, **47**, 1-8. <https://doi.org/10.1007/BF00378323>
- [9] Bell, G.M., Gordon, A.C., Lee Doig, P., *et al.* (1985) Proliferative Glomerulonephritis and Exposure to Organic Solvents. *Nephron*, **40**, 161-165. <https://doi.org/10.1159/000183476>
- [10] Harrison, D.J., Thomson, D. and MacDonald, M.K. (1986) Membranous Glomerulonephritis. *Journal of Clinical Pathology*, **39**, 167-171. <https://doi.org/10.1136/jcp.39.2.167>
- [11] Harrington, J.M., Whitby, H., Gray, C.N., *et al.* (1989) Renal Disease and Occupational Exposure to Organic Solvents: A Case Referent Approach. *The British Journal of Industrial Medicine*, **46**, 643-650. <https://doi.org/10.1136/oem.46.9.643>
- [12] Porro, A., Lomonte, C., Coratelli, P., *et al.* (1992) Chronic Glomerulonephritis and Exposure to Solvents: A Case-Referent Study. *The British Journal of Industrial Medicine*, **49**, 738-742. <https://doi.org/10.1136/oem.49.10.738>
- [13] Stengel, B., Cénee, S., Limasset, J.C., *et al.* (1995) Organic Solvent Exposure May Increase the Risk of Glomerular Nephropathies with Chronic Renal Failure. *International Journal of Epidemiology*, **24**, 427-434. <https://doi.org/10.1093/ije/24.2.427>
- [14] Mediouni, Z., Potherat, G., Barrere, X., *et al.* (2011) Renal Failure and Occupational Exposure to Organic Solvents: What Work-Up Should Be Performed? *Archives of Environmental & Occupational Health*, **66**, 51-53. <https://doi.org/10.1080/19338244.2010.506501>
- [15] Carlisle, E.J., Donnelly, S.M., Vasuvattakul, S., *et al.* (1991) Glue-Sniffing and Distal Renal Tubular Acidosis: Sticking to the Facts. *Journal of the American Society of Nephrology*, **1**, 1019-1027.
- [16] Moss, A.H., Gabow, P.A., Kaehny, W.D., *et al.* (1980) Fanconi's Syndrome and Distal Renal Tubular Acidosis after Glue Sniffing. *Annals of Internal Medicine*, **92**, 69-70. <https://doi.org/10.7326/0003-4819-92-1-69>
- [17] Patel, R. and Benjamin, J. (1986) Renal Disease Associated with Toluene Inhalation. *Journal of Toxicology: Clinical Toxicology*, **24**, 213-223. <https://doi.org/10.3109/15563658608990459>
- [18] Streicher, H.Z., Gabow, P.A., Moss, A.H., *et al.* (1981) Syndromes of Toluene Sniffing in Adults. *Annals of Internal Medicine*, **94**, 758-762. <https://doi.org/10.7326/0003-4819-94-6-758>

Abbreviations

BU: Urinary strip.

ACR: Albumin Creatinin Ratio.

HbA1C: Glycated Hemoglobin.

HLM: Red blood cell-minute leukocyte throughput.

PBR: Renal Biopsy Puncture.

CKD-EPI: Chronic Kidney Disease Epidemiology.