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Case Report

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Malaria in A Single Kidney Donor: Case Report from Taranaki, New Zealand

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Introduction

Malaria is a parasitic infection caused by Plasmodium species spread by anopheles mosquitoes [1]. New Zealand (NZ) is Malaria free nation but not immune to imported Malaria. 34 cases of Malaria were reported to Ministry of Health (MOH) in 2018 [2]. Between 2008 and 20090, 36 cases of Malaria were reported in Auckland area [3]. This resembles closely with the study which found 340 notified cases of malaria over a period of 10 years [17]. Persistence of Malaria in NZ is alarming since incidence of severe malaria and associated case fatality rate is high in industrialized countries [4].

Renal impairment is independent predictors of mortality in all age groups affected by Malaria [5-8]. Plasmodium falciparum is responsible for most cases of severe malaria and Acute Kidney Injury (AKI) in endemic regions [9-13]. AKI has not been studied in non-endemic countries where incidence of imported Malaria is high. Moreover, no study has reported the renal consequences of acquiring Malaria in single donor kidney patient. Our case study provides an insight into renal status of an otherwise healthy young single kidney donor who acquired malaria after returning from a trip to South Africa.

Keywords: Malaria, Acute renal failure, chemoprophylaxis, Malaria related complications, AKI: Acute Kidney Injury, GN: Glomerulonephritis.

Case report

31 years Male of New Zealand European origin presented to General Practitioner's (GP's) office with fever, nausea, vomiting and malaise for 3 days. Patient recently returned from 16 days trip to Africa. He visited Dar el Saleem, Zanzibar, Kilwa Nariobi, Kasuma, Goya. He reported of feeling unwell with malaise and excessive sweating on the final day of the trip in Kenya. He also reported few insect bites. Patient did not take any drug prophylaxis but did use mosquito nets. Three days prior to presentation he had fever, malaise, myalgia, two episodes of vomiting, decreased appetite, reduced oral fluids intake, passing dark cola coloured urine. He denied any sick contacts. He denied any seizures, epistaxis, hematuria, rectal bleeding or collapse. He did not have any shortness of breath, chest pain, abdominal pain, palpitations or diarrhea. No headache, neck stiffness. He did not visit any game parks. No tick bites/ horsefly bites. Past medical and surgical history was significant for him being kidney donor within the last year of presentation. GP's office exam findings were-temp 39.1*C, no signs of meningism, dry mucus membranes, soft non-tender abdomen and clear chest. Urgent bloods were done for tropical disease screen and swabs for viral illness including flu.

He was referred to Emergency Department from the GP's office when the preliminary blood results came back positive for malaria. On presentation to ED, patient looked unwell with fever of 38.5*C, RR 16, HR 71, BP-123/64, GCS 15, Spo2 100% on room air. On examination patient was alert oriented, responding to questions appropriately. No kusmaul's ventilation. There was no neurological deficit. ENT exam was normal other than dry mucus membranes. No jaundice or pallor was noted. Chest was clear with no increased work of breathing, no crepitations or wheeze. Cardiac exam was normal with good peripheral pulses and normal JVP. Abdomen was soft non-tender, no hepatosplenomegaly. No skin rashes. Since the patient arrived with confirm diagnosis of Malaria, he received Quinine and Doxycyline in the ED. ECG was within normal limits (concern for development of prolong QT on anti-malarial treatment). He also received supportive treatment with paracetamol and intravenous fluids. Given single kidney, febrile episodes and dehydration- patient was

admitted for monitoring of renal function, antibiotics and IV hydration. Artesunate (first line of drug) (Drug of Choice-DOC) was not available at the site of treatment, so the treatment was continued Quinine and Doxycycline which was switched to first line drug- Riamet (artemether + Lumefantrine) as per protocol in the ward. Patient continued to spike fever post admission at least once every twenty-four hours for four days. Aggressive Intravenous hydration was initiated and continued. Routine bloods were checked daily to monitor renal function. Chronology of laboratory results is presented in tables 1 and 2.

IV artesunate was received from Napier and Auckland and was started on day three; patient received it for two days. Patient became afebrile after 24 hours of initiating the above medicine and stayed afebrile till discharge. His clinical condition improved with improved fluid status and lab results. IV artesunate was switched back to Riamet on the day of discharge. Patient was discharged home with TDHB stock of Riamet.

Labs	**05-04-2017**	**06-10-2017**	**15-02-2018**	**04-07-2018**	**05-07-2018**	**06-07-2018**	**07-07-2018**	**08-07-2018**	**09-07-2018**	**07-08-2018**	**08-10-2018**	**13-03-2019**
Sodium				**133**	**132**	**133**	**134**	136	137		142	
Potassium				3.8	3.8	3.8	3.8	3.7	3.7		4.2	
Creatinine umol/L	**168**	**159**	**161 1.82 mg/dl**	**194 2.19 mg/dl**	**170**	**147**	**145**	**146**	**139**	**155**	**158**	160
Est GFR	**46**	**49**	**48**	**38**	**45**	**54**	**55**	**54**	**57**	**51**	**49**	48
CRP				**128**	**192**	**213**	**200**	**146**	**89**			
Bilirubin				**34**		**28**						11

Parameters	04-07-18 16:30	05-07-18 09:55	06-07-18 05:50	06-07-18 08:50	07-07-18 08:30	08-07-18 08:45	09-07-18 08:43	08-10-18 15:40	13-03-19
Haemoglobin	*146	***118**	***127**	***122**	***114**	***127**	***121**	*151	150
PCV	*0.42	***0.34**	***0.37**	***0.35**	***0.33**	***0.38**	***0.36**	*0.45	0.44
MCV	*82	*81	*83	*82	*83	*82	*82	*85	84
MCH	*28	*28	*28	*28	*28	*28	*28	*29	29
Platelet Count	**44**	**30**	**34**	**36**	**57**	**88**	**98**	193	199
WBC	***3.4**	***3.1**	***3.9**	***3.9**	*4.6	*4.8	*4.2	*6.0	5.0
Neutrophil	*2.5	*2.3	*2.8	*2.9	*2.6	*2.2	***1.7**	*3.0	2.5
Lymphocyte	***0.42**	***0.42**	***0.7**	***0.46**	*1.4	*1.9	*1.7	*2.4	1.9
Monocyte	*0.46	*0.33	*0.33	*0.43	*0.41	*0.43	*0.5	*0.38	0.4
Eosinophil	*0.01	*0.04	*0.07	*0.05	*0.12	*0.23	*0.23	*0.20	0.2
Basophil	*0.01	*0.01	*0.01	*0.01	*0.02	*0.03	*0.04	*0.07	0.1

Discussion

Malaria remains one of the serious and sometimes fatal illnesses in spite of chemoprophylaxis and effective treatment. Malaria immune countries still are at risk of acquiring and spreading imported malaria to its non-immune population where case fatality rates can be very high [1,4].

Renal involvement in Malaria

Malaria can cause impairment in glomeruli, tubules and interstitium. Microcirculation obstruction, endothelial activation, cytokines release has been implicated in pathophysiology behind renal involvement in Malaria [5]. Renal involvement in Malaria has been extensively studied in endemic regions and in patients with normal renal functions. 58% of adults with severe malaria had AKI as defined by Kidney Disease: Improving Global Outcomes (KDIGO), 40% of these patients died, leading to 71% overall mortality [5, 25].

Chronology of laboratory results is shown in Table1. Before infection with malaria our patient's kidney functions at baseline were abnormal for his age as expected [27]. There are few studies which states reverse [28]-renal function

should either stay same or actually improve post kidney donation-? due to hyper glomerular blood flow. Patients' eGFR pre-donation was 85 with creatinine of 102 umol/L and creatinine clearance of 19 [8-18]. Post donation eGFR is reported to be 49 and creatinine is 168 umol/L (3 months post donation).

After admission patient's creatinine raised to 191umol/L (baseline at 161) (Patient developed AKI on day one (more than 0.3 mg/dl increase in creatinine as defined by KDIGO's AKI criteria) and eGFR dropped to 38 from 49 (two months prior to admission). Also, since patient was severely dehydration it is unclear if the acute renal injury was due to dehydration or direct impact of malaria on kidneys. Quick recovery following aggressive hydration in next 24 hours indicated towards the latter as etiology. Acute renal failure complicates falciparum malaria more so in nonimmune (25-30%) than in immune populations (1-4.8%).

Hyponatremia

Patient also had hyponatremia of 133 mmol/L (normal range: 135-145mmol/L). 55-67% of patients with P falciparum develop hyponatremia [23,26].

Thrombocytopenia/cytology

Another important finding was thrombocytopenia. Since there was no evidence of bleeding from any site nor significant reduction in hemoglobin levels coagulation profile was not checked in this patient. Several studies have studied thrombocytopenia in Malaria [19-22]. Raised inflammatory marker CRP up to 200s on day three of admission is consistent with febrile events. White cell count on the other hand was low ranging from 3.1 to 3.9.

Jaundice

Our patient had increased bilirubin at 30umol/L (normal range: 9-20umol/L). One study reported that over 75% of patients with ARF malaria develop jaundice. It is described as biphasic, initially due to hemolysis and later due to cholestasis [24].

This patient was followed by Renal Physician at Taranaki Base Hospital as per protocol and showed steady slow improvement in his clinical status however a new low renal function status.

Global burden

WHO estimates that 228 million cases of malaria occurred worldwide in 2018 (confidence interval: 206–258 million) and about 405 000 people died from the disease, mostly children under 5 years of age in sub-Saharan Africa [14].

One observational study conducted in Auckland to assess magnitude of Malaria reported no NZ tourist travelers infected with Malaria. New entrant or the NZ residents with family ties in the endemic region are at risk of acquiring malaria[3].Quinine and Mefloquine were the most common anti-malarials prescribed in NZ during 1993-98 [17]. Change in type of plasmodium affecting NZ. In 1980-90's it was P Vivax [15,16] but recent studies have shown that the incidence of P falciparum is more than P Vivax [3].

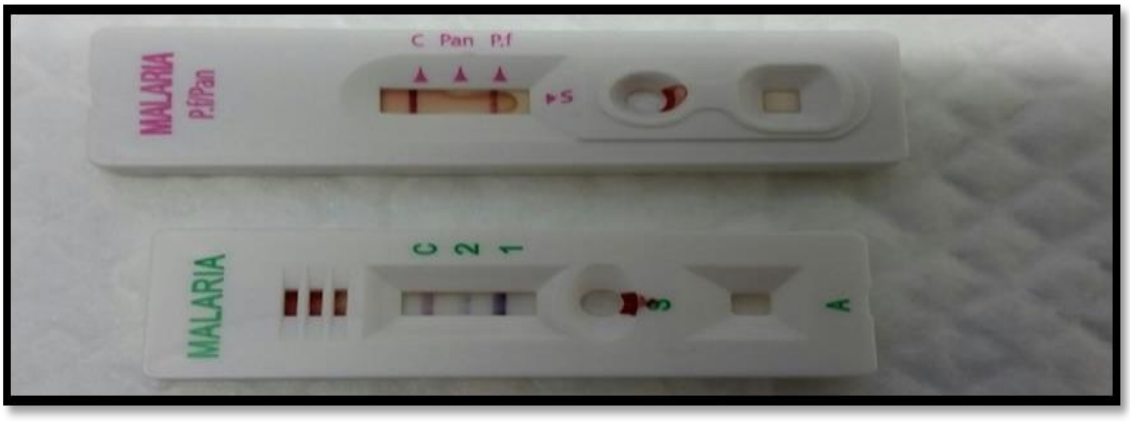
Conclusion

This report would like to propose the possible outcome of uncomplicated P Falciparum Malaria in a non-immune person with single kidney residing in a non-endemic area.

Highlights of our report

1. Case report on patient with single kidney acquiring malaria is first of its kind.
2. Renal complications (AKI, GN etc) directly due to malaria are well known4[4,5, 6]. But renal complications in a patient with single kidney have never been reported in literature.
3. Renal status 1-year post Malaria.

Our patient had six out of the ten complications described in literature which were directly related to falciparum malaria. All these complications are well studied in malaria endemic regions in otherwise healthy patient with normal kidney function. Our case report is different as we describe systemic and renal complications associated with imported malaria in a single kidney donor patient.

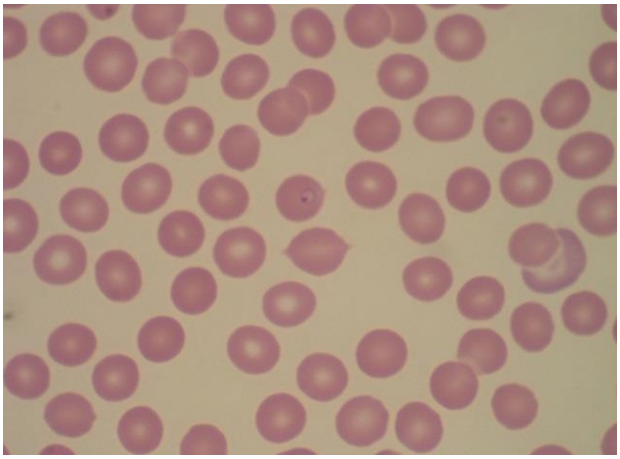


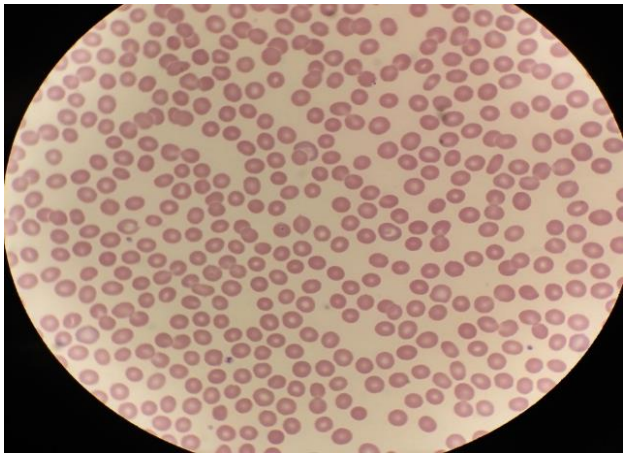
Lesson learned is immune patients with one kidney are at increased risk of multiple complications of Malaria in non-endemic countries such as New Zealand.

The patient was quickly diagnosed and treated based on the travel history. However delayed diagnosis, incomplete history etc. could mislead or delay the diagnosis. As mentioned earlier the case fatality rates are very high in industrialized countries and hence prompt diagnosis and treatment is essential.

Another important aspect is chemoprophylaxis. Several studies have shown effectiveness of appropriate chemoprophylaxis in prevention of tropical disease. However, we fail to follow the guidelines and put ourselves at risk of infection. Chemoprophylaxis is of outmost important in travelers to endemic areas.

A distant Malaria complication is another concern 18]. Close differential diagnosis of P falciparum is Plasmodium knowlesi. It is important to bear in mind that if the rapid screening test is negative it still could be P. knowlesi as there is no screening test available for this fifth type of Malarial protozoa [29].





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Conflicts of interest
There are no conflicts of interest.

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