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Simultaneous Involvement Of Cns And Pns In Multiple Co-Infection Of Arthropod Born Febrile Illness Patient : A Rare Case Presentation

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I. Introduction

Plamodium falciparum,plasmodium vivax are protozoan disease spread by female anopheles mosquito bite which constitutes most important of parasitic disease of humans. Scrub typhus caused by orientia tsutsugamushi which spreads due to bite of trombiculid mite is also a common cause of acute febrile illness in many parts of Asia including Indian sub continent.Chikungunya spreads by the bite of Aedes mosquito is quite a less common cause of patient presenting with acute febrile illness. Presentation and laboratory features of above diseases are common which includes high grade fever, low platelet count and multi organ system including cardiovascular, haematological, neurological manifestation involvement ultimately leading to shock, bleeding tendencies ,delirium, seizure, peripheral neuropathy etc. Coinfection of all four illness along with involvement of central nervous system and peripheral nervous system in the form of neuropathy is extremely rare. Here we report a case which had involvement of both CNS and PNS inform of demyelinating and axonal neuropathy with co-infection of plasmodium vivax,plasmodium falciparum,scrub typhus and chickengunea the above described illness.

II. Case Report

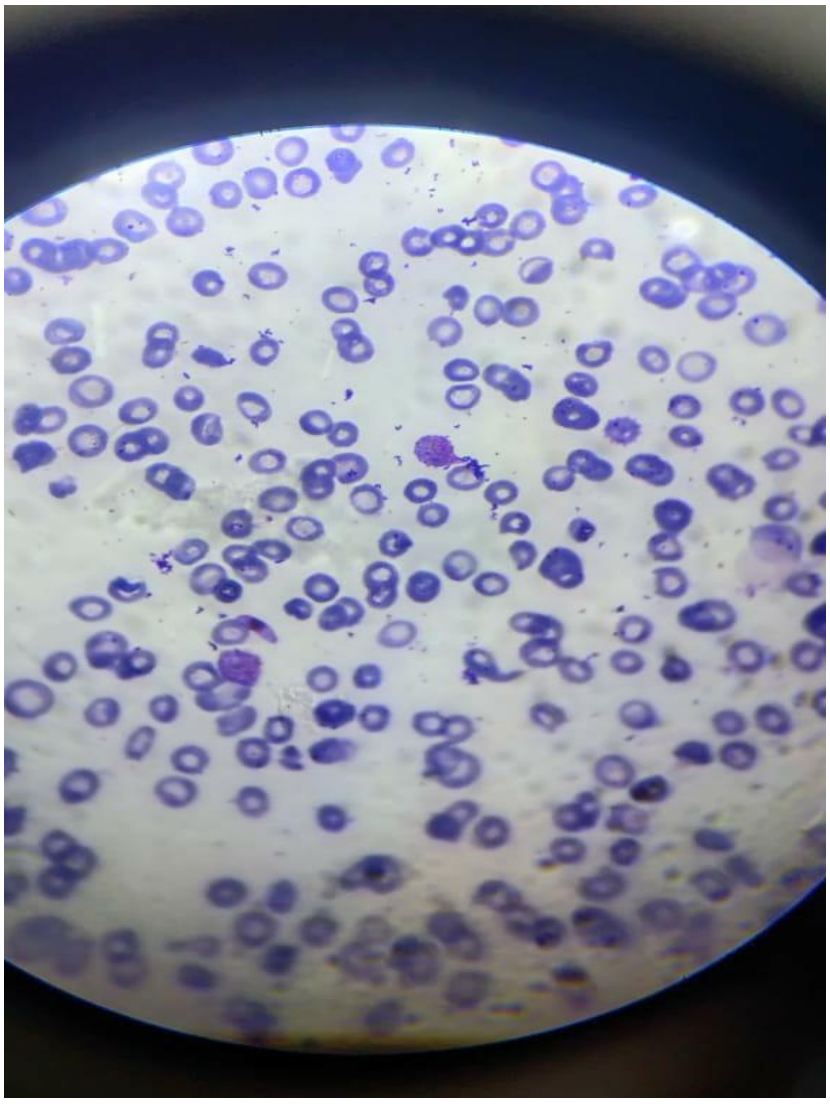
An elderly female aged 50 year came with chief complaints of high grade fever with chills and rigor for 8 days and joint pain for last 2 to 3 days, altered sensorium for last 2 day. Fever was intermittent in nature with multiple spikes in a day. Patient also complained of generalized body ache with multiple painful joints without selective joint pattern. Followed by sudden onset altered sensorium for last 2 days with bowel and bladder incontinence. No complains of burning micturation, abdominal pain ,diarrhea, vomiting, cough.No history of head trauma, fall, abnormal body movement . No past history of chronic medical illness or previous hospitalization was present .No history of chronic medications and no history of any recent vaccination.Her general physical examination revealed altered conscious in stuprous state and disoriented to time, place, person. Her Blood pressure was 130/80 mm of Hg, pulse rate 102 bpm, respiratory rate 22 p/min, temperature 102 degree Fahrenheit on admission. Glasgow coma scale had score of $E_1V_1M_3$ .Pupil bilateral mid size sluggish reactive to light. Bilateral pitting edema was present in both lower limbs. No icterus, cyanosis, lymphadenopathy, rash or eschar mark was present. Wrist,knee and ankle joint tenderness was there. Respiratory system examination revealed bilateral basal fine crepitations. In Central Nervous System examination higher mental function cant be examined ,cranial nerve examination revealed II including fundus,III,IV,VI as normal and rest could not be examined. Sensory system, motor system could not be examined due to stuperous state.Bilateral plantar reflex were extensor . Rest of Superficial reflex were absent. Deep tendon reflex were +2 in all four limbs.Spine did not revealed any abnormality. Gait cannot be assessed. Signs of meningeal irritation were absent

Complete blood count revealed (Hb-5.4g/dl),TLC 7000 cells/mm^3 (N=64%,L=30%,M=4%,B=1%) and thrombocytopenia(52,000cells/mm^3),PBF revealed normocytic normochromic anaemia ,ESR =21mm/hr..Renal functions revealed elevation of urea (88.5mg/dl) with normal creatinine(1.27mg/dl).There was mild derangement of liver function tests with elevation of total bilirubin(2.1mg/dl) and direct bilirubin(0.868)with mild elevation of liver enzymes(SGOT 113mg/dl,SGPT 35mg/dl,alkaline phosphate 138mg/dl). Thick and thin blood smears were prepared which showed ring forms and gametocytes of plasmodium falciparum and trophozoites ,schizonts and gametocytes of plasmodium vivax , IgM ELISA test positive for scrub typhus and also for chickengunea and negative IgM ELISA test for dengue by. She was treated with broad spectrum antibiotics,inj.artesunate(2.4mg/kg stat iv followed by 2.4 mg/kg at 12,24 h and then daily od for 4 day,cap doxycycline 100mg bd ,tablet Paracetamol, capsule Omeprazole and intravenous fluids. For further evaluation

of the poor GCS and slow reactive pupils NCCT brain and fundus carried out which showed no significant abnormality. As the possibility of meningoencephalitis was still a concern CSF evaluation was done which showed total cells of 30 with 60% neutrophils and rest of lymphocytes with normal glucose, protein and no organisms on staining. Chest X ray PA view no abnormality detected,ECG showed sinus tachycardia and USG abdomen revealed splenomegaly, mild ascitis.

After 4 days her GCS improved ($E_4V_5M_6$) and she started following verbal commands, taking per oral feeding. Then we found during examination weakness in all four limbs.Weakness was more in lower limb than upper limb and more distal then proximal.Grading include upper limb 3/5 and lower limb1/5.DTR were diminished in upeer limb and absent in lower limb. Bilateral plantar reflex were mute .Other superficial reflex were absent.Sensory examination revealed loss of fine touch,pain ,vibration and temperature till above ankles in both lower limbs and till wrist joint in upper limbs.Spine examination was normal. Gait cant be assesd.No meningeal sign of irritation was present .Rest systemic examination which included CVS,GIT,Respiratory system revealed no abnormality.ENMG study of both sensory and motor nerve of both upper and lower limbshowed mixed demyelinating and axonal neuropathy and radiculopathy consistent with a diagnosis of Guillain-Barre syndrome (GBS).

She was continued with the same treatment with daily physiotherapy and there was gradual increase in power and sensations with complete recovery by day13.She was discharged after day 13 with primaquine tablet for 14days for vivax radical cure(G-6 PD levels normal) in afebrile condition and with routine investigation's returning to normal limits and advised for follow-up after one week.



Ring forms and gametocytes of plasmodium falciparum.
Trophozoites, schizont and gametocytes of plasmodium vivax.

III. Discussion

Malaria, Dengue and scrub typhus infection are the most frequently listed presumptive diagnoses in patients with acute febrile illness. Chikungunya also presents with similar acute febrile illness with arthralgia. No case study has been published on co infection of these three vector borne disease malaria, scrub typhus and chikungunya fever as per literature. Coinfection should always be considered in endemic areas. A high degree of suspicion for coinfection must be present in patient presenting with febrile illness in tropics especially post monsoon season with multisystem involvement[1]. Atypical neurologic manifestation in malaria are cerebral malaria, psychiatric manifestation, cerebellar ataxia ,post malarial neurologic syndrome ,isolated hemiparesis, peripheral neuropathy. Peripheral neuropathy in form of Gullian barre syndrome in vivax and falciparum has been previously reported in rarely few cases.[2] Even plasmodium vivax mono-infection has the potential to cause severe manifestation including cerebral malaria and multiorgan dysfunction[3]. We could locate 12 cases of GBS following malarial illness from literature.8 of them following falciparum infection and 4 of them following vivax .Of this 4 cases out of 8 having GBS due to falciparum malaria developed severe paralysis and respiratory failure[4]. Spectrum of neurologic presentation with scrub infection involve both CNS and PNS. Meningitis and meningoencephalitis remain most common CNS manifestations.In PNS being mononeuritis multiplexa, brachial plexus neuropathy, polyneuropathy, myelitis, GBS.GBS in scrub is rare and remains under reported manifestation[5]. GBS is a rare neurologic complication which may occur after subsidance of fever and constitutional symptoms by several neurotropic viruses[6]. Various neurologic complication associated with chickengunya have been described such as meningoencephalitis, myelitis, GBS, encephalitis ,myelopathy, peripheral neuropathy, myeloneuropathy and myelopathy[7].

Cerebral malaria has been a known complication in falciparum malaria owing to its property of cytoadherence, agglutination and rosette formation by RBCs in capillaries and there by affecting microcirculation[8]. Peripheral nervous system involvement being extremely rare has been seen in all these illness but there was no literature stating its incidence in each of them. Pathogenesis of peripheral neuropathy in these illness is still unknown and likely to be immunogenic. Other mechanisms being obstruction of vaso nervorum by parasitic emboli, release of neurotoxins, immune mediated capillary damage and release of free radicals and tumor necrosis factor.[4] Different infections can lead to peripheral neuropathy which differ in spectrum of autoantibodies and clinical symptomatic presentation.This may be of some significance for early prognosis and future possibility for choosing therapeutic options.[9]

In our patient clinical picture had developed after an attack of falciparum, vivax , scrub typhus and chickengunya. Since patient had no other preceding infection that could be associated with peripheral neuropathy or drug usage that could be linked to GBS,mixed infection thought to be the triggering factor for GBS .Hence there is distinct need for physicians and health workers at all level of care in india to be aware of clinical features, available diagnostic tests and their interpretation and therapy for these infections[10].

IV. Conclusion

A presentation of acute febrile illness with deranged laboratory values and multi system organ involvement should raise suspicion for coinfections in patients presenting from endemic areas.Febrile illness presenting with multiple overlapping clinical symptoms should be screened for coinfections and thorough clinical examinations throughout the stay of patient and workup accordingly will help in initiating appropriate treatment and reducing mortality and morbidity associated with them.

References

[1]. Kamath V, Ganguly S, Avinash B L. A comparative study of concurrent infections of rickettsial infection, Malaria, Typhoid, and Chikungunya with Dengue. APIK J Int Med 2019;7:120-6

[2]. Gangula RS, Stanley W, Vandanapu A, Prabhu MM. Guillain-Barre Syndrome with Falciparum Malaria and Scrub Typhus Mixed Infection-An Unusual Combination. *J Clin Diagn Res.* 2017;11(9):OD10-OD11. doi:10.7860/JCDR/2017/28390.10629

[3]. Ghosh S, Das SK, Sharma A. Uncommon neurological manifestations of a common tropical vector borne disease. Trop Parasitol 2015;5:61-3

[4]. Chakravarty A, Ghosh B, Bhattacharyya R, Sengupta S, Mukherjee S. Acute inflammatory demyelinating polyneuropathy following plasmodium vivax malaria. Neurol India 2004;52:130-1

[5]. Phillips A, Aggarwal GR, Mittal V, Singh G. Central and peripheral nervous system involvement in a patient with scrub infection. Ann Indian Acad Neurol 2018;21:318-21

[6]. Agarwal, A., Vibha, D., Srivastava, A.K. *et al*. Guillain-Barre syndrome complicating chikungunya virus infection. *J. Neurovirol.* **23,** 504–507 (2017). https://doi.org/10.1007/s13365-017-0516-1

[7]. Mahto SK, Gupta PK, Singh A, Meena RC. Atypical Neurological Manifestations of Chikungunya Fever: Two Case Reports. *Indian J Crit Care Med*. 2018;22(4):306-308. doi:10.4103/ijccm.IJCCM_459_17

[8]. J.Larry Jameson,Dennis.L.Kasper,Dan .L.Longo,Anthony.S.Fauci,Stephen.L.Hauser,Joseph Loscaizo;Harrison principal of internal medicine 20th edition;Chapter 219 ;Malaria ;page number 1577-1585

[9]. 4.G S Tanwar, P C Khatri, G S Sengar, A Kochar, S K Kochar, S Middha, G Tanwar, N Khatri, D Pakalapati, S Garg, A Das & D K Kochar (2011) Clinical profiles of 13 children with *Plasmodium vivax* cerebral malaria, Annals of Tropical Paediatrics, 31:4, 351-356, DOI: 10.1179/1465328111Y.0000000040

[10]. Sambor grygorczuk,Jaama zajkowska,Maciej kondrusik;Gullain barre syndrome and its association with infectious factors.Neurology neurochir pol 2005;230-236.
[11]. Rahi M, Gupte M D, Bhargava A, Varghese GM, Arora R. DHR-ICMR Guidelines for diagnosis & management of Rickettsial diseases in India. Indian J Med Res 2015;141:417-22

XXXX, et. al. "Simultaneous Involvement Of Cns And Pns In Multiple Co-Infection Of Arthropod Born Febrile Illness Patient : A Rare Case Presentation." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 19(12), 2020, pp. 15-18.