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Rickettsia (Spotted Fever Group) Infection with Multiorgan Dysfunction

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Abstract In recent times Rickettsial disease has been an important cause of re-emerging infection all over the world. On most occasion they are difficult to diagnose as their presention resembles that of viral, bacterial or protozoal diseases. This may lead to unnecessary investigation and treatment and may lead to considerable morbidity, irreversible damage to vital organ and finally death if not treated earlier. However the positive aspect of this condition lies in the fact that they can easily be diagnosed by cheap, inexpensive blood test (Weil Felix test) and can be treated with less expensive oral drugs like doxycycline if high degree of suspicion is maintained. Here I present a case of rickettisia infection caused by spotted fever group who came with fever, rash, multiorgan dysfunction diagnosed outside as viral fever but was successfully treated in our icu with complete recovery.

Keywords: *fever, jaundice, rash, ARDS, AKI, hepatitis--Rickettsia (spotted fever group) infection*

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1. Introduction

The rickettsia group of organism are obligate flagellated intracellular gram negative coccobacilli most of which are transmitted by tick, mite, flea, louse. Rickettsia belong to six genera(Rickettsia, Orienta, Ehrlichia, Anaplasma, Neorickettsia, Coxiella. [1,2] The spotted fever group comprise of Rocky mountain spotted fever(caused by rickettsia rickettsi, prevalent in America, mexico, Canada), tick borne spotted fever(caused by rickettsia Conorri , rickettsia Africae, rickettsiae japonica), flea borne spotted fever(caused by R.felis). Rickettsia conori is the cause of Ticktyphus in India and Mediterranean region. [2,3,4,5]. The organism proliferate in small blood vessels of skin, , lungs, brain, kidneys, heart, skeletal muscle causing vasculitis responsible for skin rash, microvascular leakage, edema, tissue hypoperfusion and end-organ ischemic injury. Formation of thrombi can lead to tissue infarction and hemorrhagic necrosis.

Inflammation and vascular leakage leads to interstitial pneumonitis, noncardiogenic pulmonary edema, cerebral edema and meningoencephalitis. Infection of endothelial cells also induce procoagulant activity that promotes coagulation factor consumption, platelet adhesion and leucocyte emigration and may result in clinical syndrome similar to disseminated intravascular coagulation [3].

Following a incubation period ranging from 2-28days, disease is heralded by sudden onset of highgrade fever with chills and rigors, malaise headache, dry cough, body pains, conjunctival congestion and vomiting. A maculopapular rash develops after 3-5days which starts in the extremities and spreads centripetally to involve other parts of body. [6]. The disease can be complicated by multiorgan involvement in the form of pneumonitis, Ards, hepatomegaly, deranged LFT, renal failure, myocarditis, thrombocytopenia, meningoencephalitis, DIC. [7]. If not treated early the mortality rate is as high as 30-35% [3].

2. Case Report

24 year old male was admitted in downtown hospital with history of high grade fever associated with chill, headache, yellowish discoloration of urine, rash in abdomen and chest of 3weeks duration. This was followed by distention of abdomen, decrease urine output and swelling of feet and difficulty in breathing. Patient did not have history of cough, joint pain, burning micturition, pain abdomen, diabetes, hypertension, asthma, blood transfusion, illicit drug abuse or herbal medicine intake. Initially he was admitted in two different hospitals and treated with several antibiotics, antimalarials but his condition detoriated further and then shifted to our hospital

On arrival

Patient toxic, febrile(101*F), pulse-120/minute, B.P-120/70mmhg, JVP not raised, RR-36breaths/minute, Pallor-negative, Icterus-positive, Cyanosis-negative, Edema both feet-Present, Conjunctival congestion-present, SP02 of 86% in room air and 96% with 4litres oxygen by face mask.

Chest auscultation-Bilateral crepitations base of lung. CVS-normal heart sound without any murmur.

Perabdomen-Liver palpable(2cm below the costal margin)

Maculopapular rash of skin over neck, upper chest, abdomen present

Patient was immediately shifted to ICu and was put on oxygen, IVfluids, 3sample of blood culture sent. Routine investigation of blood revealed –TC -18500(P-82, L-16, M-2, E-0), Platelet count(70, 000), Serum creatinine (5.16mg/dl), Na-115.1mg/dl, K+3.08mg/dl. LFT-Serum Bilirubin (2.36mg/dl(D-2.3), I(0.06)), Total protein (4.83mg/dl), serum albumin (2.04mg/dl), SGPT(169), SGOT(399), Salkphosphate (531). PT INR(1.7). Sammonia-50mg/dl. CRP-39.

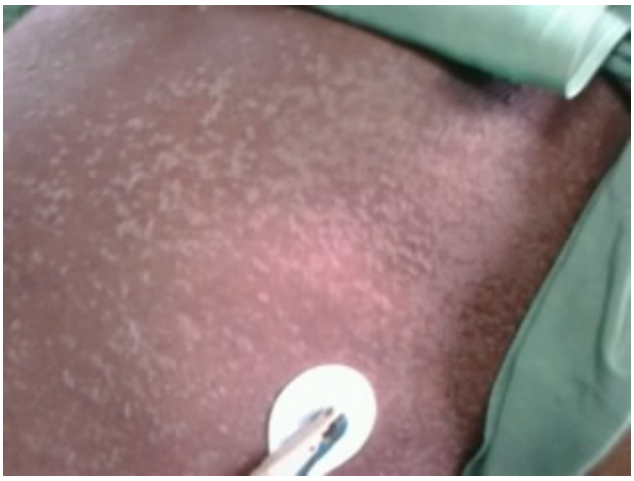

Figure 1.

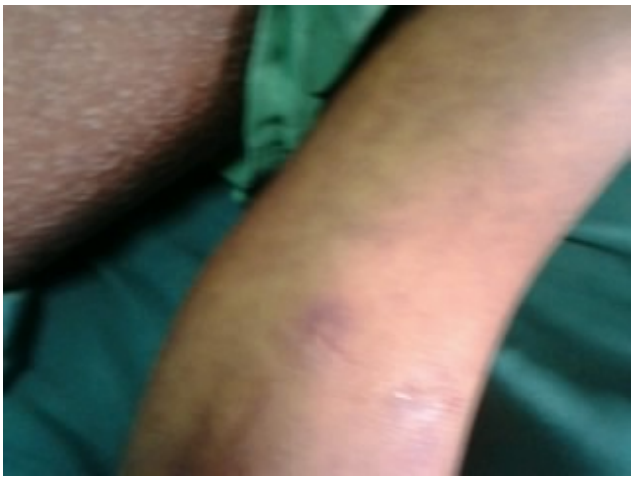

Figure 2.

ECG-sinus tachycardia. Echocardiography –Normal. Xray chest-Bilateral infiltrates in both lung fields. Peripheral smear for malaria parasite negative, widal test negative, leptospira antibody negative. Hepatitis A, B, C, E-negative, Dengue and VDRL serology-negative.HIV 1 and 2 negative. Urine R/E-Normal Patient was started on meropenum injection(500mg I.V.B.D), teicoplanin injection (200mg I.V. OD). Vital signs, urine output, respiration, oxygen saturation were frequently monitored. Nephrology, medicine consultation taken.This management was continued for next 24hrs. However on the next day patient had high fever, tachycardia, tachypnea, restlessness. Total WBC count rose to 20, 000/cumm. ABG –PO2(49.9mmhg), PCO2(32.3mmhg), PH-7.320, Hco3-16mmol with Fi02 of 40%. Po2/Fio2-124. Patient was put on mechanical ventilator(volume control). Xray chest revealed ARDS with bilateral opacities in lung. Tracheal cultures were sent which came to be sterile. Ventilator settings were set according to Ards network protocol. Strict input /output charting was done keeping in slight negative balance.

As the general condition of the patient was not improving, again a thorough history from his collegues, and review of old medical records was done. History from one of his collegues revealed that he had a travel history to jungles and hilly regions as regards his posting(CRPF personal) two weeks prior to his fever. Admission in two other hospital revealed he was empirically treated with antimalarial and broad spectrum antibiotics but without any improvement. This made us think of a alternate diagnosis and possibility of rickettsial infection which explained all his symptoms and clinical finding was considered. Weil Felix agglutination test for proteus (OX19, 2, K) was immediately sent and empirically Tablet Doxycycline (100mg) twice daily was started. Over next 48 hrs his fever subsided, urine output improved, rash diminished, ABG improved and was slowly weaned from ventilator and extubated on 4thday. Blood culture was sterile, however his weil felix test was highly positive for spotted fever group rickettsia(Proteus Antigen OX19, OX2)(1:320). The patient was kept in ICU for 2 more days where he underwent physiotherapy and then shifted to ward continued on tablet doxycycline for further 14days and discharged after 3days.

3. Discussion

The patient presented with history of fever, rash, jaundice initially and despite being treated with antibiotics and antimalarials he did not improve. When he arrived in our icu he already had acute kidney injury, Ards, high total count, low platelet, conjunctival congestion and prompt support in the form of ventilator and broad spectrum intravenous antibiotics was instituted. Even after 24hrs as his condition did not improve history and old medical records was again reviewed it led us to think of a alternate diagnosis which ultimately led to his recovery.

Diagnosis of rickettsial infection in such cases can be difficult as a host of other medical condition like malaria, typhoid, dengue, sepsis, leptospira, liver disease can all produce the same picture. In Icu setting as support of vital organs and resuscitation takes more priority when such patients presents with multiorgan dysfunction, diagnosis is overlooked or missed. Hence a high degree of suspicion should be maintained especially when general condition does not improve or detoriates.

Specific diagnosis is made by weil felix test, immunoflourescent assay, Elisa and polymerase chain reaction. Weil felix test is heterophile antibody test against agglutinins to Proteus vulgaris ox 2, ox 19 and ox k. . It is inexpensive and easy to perform. A four-fold rise in agglutinin titer in paired sera or single titer of more than equal to 1:320 is considered diagnostic for infection with these organism.

4. Conclusion

A high degree of clinical suspicion, coupled with good history of patient illness including travel and clinical

examination, along with appropriate investigation, aggressive organ support, are hallmarks of early detection and treatment of rickettsial infection in order to prevent fatal complication. Rickettsial infection should always be considered as a differential diagnosis in patients with unknown fever particular if associated with rash and multiorgan failure.

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