trunk which spread to the palms and soles over the next few days. On day 5, she developed congestion of conjunctiva and of the oral cavity and on day 7 she had left focal seizures. She was admitted at a local hospital where her investigations revealed a Hb of 7.0 g/dL, a normal leukocyte count, and platelet count of 80,000/cc. Her CSF examination was not done. She received antibiotics and anti convulsants for a week. However since her fever did not resolve she was referred to us. On examination she was irritable, had a generalized maculopapular, petechial rash all over the body, including palms and soles. She had edema over the dorsum of hands and feet but no organomegaly, lymphadenopathy or meningeal signs. Her total leukocyte count was 29,800 per cu mm with polymorphs of 84%, platelet count was 4.5 lakhs, ESR was 60, and CRP was elevated. Weil Felix test was positive in a titre of 1: 160 for Proteus Ag Ox 19 and 1: 80 for Proteus Ag Ox K. She was started on oral doxycycline. She became afebrile in 48 hours.

We used the clinical scoring system described by Rathi, *et al*. [1] and she had a score of 17 which is considered to have a specificity of 100%. We could not do the Elisa for IgM antibodies to Rickettsia because of financial constraints.

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Visceral Leishmaniasis (Kala-azar) without Splenomegaly

We are reporting an unusual case of visceral leishmaniasis (VL) in a 7-year-old male, presenting without splenomegaly

A 7-year-old male child presented with fever often associated with rigor and chills and loss of weight and appetite for last one month. There were no other significant localizing symptoms. A general physical examination revealed significant pallor. Systemic examination, including absence of organomegaly on abdominal evaluation, was non-contributory. Hematological parameters revealed pancytopenia with hemoglobin 4 g/dL, total leukocyte count of 2100 (N-22%,L-74%,E-2%,M-2%,B-0%), ESR in first hr 86mm, platelet count of 38000 and peripheral smear showing a leucoerythroblastic picture. Hypregama-globulinemia with albumin globulin ratio of 0.3 was seen. Liver function test on admission was normal. Peripheral smear for microfilaria was negative. Human immunodeficiency virus (HIV), Hepatitis B (HBsAg), hepatitis C (HCV) and dengue serology were negative. Chest *X*-ray was normal and Mantoux test was negative. Urine culture showed no growth. Ultrasound of whole abdomen was a normal. RK-39 was positive. Bone marrow examination was done, which revealed Leishmania Donovani (LD) bodies.

A final diagnosis of kala- azar was made. Absence of splenomegaly was outstanding finding in our patient. Patient was started on amphotericin B and other supportive therapy including blood and platelet transfusions. The patient improved and was discharged after giving full course of amphotericin B.

VL comprises a broad range of manifestations of infection. Infection remains asymptomatic or subclinical in many cases or can follow an acute or chronic course. The clinical symptoms are characterized by prolonged and irregular fever often associated with rigor and chills, splenomegaly, lymphadenopathy, hepatomegaly, pancytopenia, progressive anemia, weight loss and hypergamma-globulinemia (mainly IgG from polyclonal B cell activation) with hypoalbuminemia [1]. A presumptive provisional clinical diagnosis is made on the basis of presenting clinical features and history of living in an area endemic for VL. Leishmanial infection does not lead to clinical disease in all cases; asymptomatic and subclinical forms are frequent which has been demonstrated in various epidemiological surveys [2,3].

In endemic areas; infected subjects may or may not develop classic signs and symptoms. Capacity to produce IL-2 and interferon-gamma (IFN-γ) is associated with asymptomatic or subclinical self-healing infection. In contrast, individuals whose lymphocytes do not proliferate and, thus, do not produce IFN-γ when stimulated by *Leishmania* antigen, will develop acute VL that progresses to classical disease [4]. The subclinical form of VL shows nonspecific clinical manifestation, characterized by, fever, hepatomegaly, and hypergamma-globulinemia, increased ESR, without splenomegaly and leucopenia, leading to difficulties in diagnosis [5]. The

occurrence of splenomegaly and leucopenia distinguishes the acute form from subclinical form [5].

The main intention of reporting this case is to raise the awareness of possibility of kala-azar in absence of splenomegaly. Instead of relying solely on the classical clinical features of visceral leishmaniasis (pyrexia with splenomegaly), simple laboratory findings like pancytopenia, altered albumin/globulin ratio and a positive aldehyde and RK- 39 dipstick tests can help make an early diagnosis even in atypical cases, thereby reducing the mortality of visceral leishmaniasis.

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Hemothorax Following Snakebite

We read with interest the publication on hemothhorax following *Echis carinatus* snake bite [1]. Clinical manifestations of *Echis* bite envenomation are acute external bleeding (gum bleeds, hematemesis) or internal (serous cavities, peri-nephric, retroperitoneal, intra cranial and hematoma in muscles). Echis venom is a rich source of procoagulant which convert prothrombin to thrombin resulting in fibrin deposition which is later fibrinolysed resulting in hypofibrinogenemia and thrombocytopenia and thus disseminated intravascular coagulation. Acute uncontrolled bleeding due to DIC is corrected by blood and blood products rather than heavy doses of ASV [1]. In a viper bite, initial hypotension and shock is attributed to various actions of venom such as increased vascular permeability or leaking syndrome, or by direct action on vascular smooth muscle [2].

There is no additional advantage of giving high dose ASV [5]. ASV neutralizes the free circulating venom, and is unnecessary once venom is attached to receptor site on red cells, platelets, smooth muscles and endothelium. In such a situation, one has to counter the after-effects of venom, such as bleeding and DIC by blood and blood products [1]. Scientists working on venom should prepare monovalent antivenom and antigen detection kit to know the species of snake bitten and exact amount of circulating snake antigen level and dose of ASV needed to neutralize the same [2].

The authors may also want to keep regular follow up of this child for possibility of development of hypopituitarism. Hypopituitarism is reported with Russell's viper bite but not due to saw-scaled viper; howerver, in the present case authors are not sure of the species [1,2].

E carinatus is found all over India. The amount of its venom and its toxicity varies according to geographical regions. The venom of *Echis* from Jammu causes severe coagulation defects and renal failure, which is uncommon in Maharashtra [3]. Antivenom producers in India should be encouraged to prepare anti snake venom (ASV) from snakes caught from relevant areas of country [4].

Snake bite is a major public health problem in India. Unfortunately, public health authorities have given little attention to this time limit and life threatening medical emergency, relegating snake bite envenoming to the category of a major neglected disease of 21st century.

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