

Atypical Presentation of Visceral Leishmaniasis

Sir,

Kala Azar or visceral leishmaniasis [VL] presents as fever, pancytopenia and hypergammaglobulinaemia.^{1,2,3} Many presenting features overlap between VL and adrenal insufficiency [AI] viz. chronic diarrhea, weight loss, anorexia, skin hyperpigmentation and leucopenia. The presence of splenomegaly is characteristic of VL.¹ It may be absent in immunocompromised patients, who may present atypically.^{1,2} Absence of splenomegaly is rare in immunocompetent patients, though it may occur in the early stages. We report a patient who presented with transient AI, with no palpable spleen, who was subsequently documented as VL and successfully treated.

A 60 year old male hailing from Bihar presented with chronic diarrhoea of 4 months duration, anorexia, significant weight loss and intermittent fever. There was no h/o abdominal pain, alternating bowel habits, vomiting or bleeding per rectum. There was no h/o sternal tenderness, petechiae or purpurae. He had last visited Bihar one year ago. On examination, he was an ill-looking patient, febrile [38.5° C], thinly built, malnourished and had a uniformly dark pigmentation all over the body. On enquiry he mentioned that he had noticed the pigmentation a few months ago. Mucosal pigmentation was present in the oral cavity. He had mild pallor. There was no icterus, clubbing or lymphadenopathy. He was hypotensive, with a systolic BP of 80 mm of Hg. Orthostatic hypotension was present. The abdomen was soft and there was a 2cms firm, non-tender palpable liver, but no splenomegaly. Rest of the systemic examination was unremarkable. Investigations revealed Hb of 7.5 gms%, TLC of 1400/cmm, platelet count of 75,000/cmm and ESR of 92. Red cell indices showed a normochromic, normocytic anaemia. Serum sodium was 116 mEq/Lt and serum potassium 6.5 mEq/Lt. A repeat serum electrolytes also showed hyponatremia and hyperkalemia. Blood sugar was normal. Renal and liver function tests were normal except for serum albumin of 2.2 gms% and serum globulin of 5 gms% [A/G ratio=4.4]. Stool for occult blood was negative on three occasions. Blood cultures for bacteria and fungi were sterile. Abdominal sonography showed multiple gall bladder calculi. Chest radiograph was normal and HIV was non-reactive. In view of chronic diarrhoea, weight loss, orthostatic hypotension, mucosal pigmentation and hyponatremia, a diagnosis of primary adrenal insufficiency was suspected and hence an 8am Serum cortisol and ACTH were done. Serum cortisol was 12 microgm/dL which was relatively

low considering the presence of the stress of illness. ACTH was normal. Barium meal follow through was done to rule out intestinal tuberculosis, which was normal. To rule out malignancy, an upper and lower gastrointestinal scopy was done which was normal. ANA, dsDNA and antiphospholipid antibodies were negative. CT abdomen revealed many retroperitoneal lymph nodes and bilaterally small adrenals. Finally, a bone marrow examination was done to rule out haematopoietic malignancy or tuberculosis. This revealed a field full of LD [*Leishmania donovani*] bodies. The patient was treated with Amphotericin B in a dose of 0.5 mg/kg for 15 doses on alternate days. A lower than recommended dose was used since he was weak and malnourished and it was decided to increase the dose subsequently if he tolerated this dose, but he developed thrombophlebitis due to which the drug had to be temporarily discontinued and then restarted. Steroids in a tapering dose for adrenal insufficiency had already been given. He improved remarkably on treatment with disappearance of the skin and mucosal pigmentation. A repeat bone marrow examination showed few degenerated LD bodies. A repeat CT abdomen showed disappearance of all the lymph nodes. A repeat serum cortisol at the end of treatment was 11 microgm/dL. He was subsequently discharged and is doing well on follow up.

VL is a chronic infectious disease caused by *Leishmania donovani* [LD] and characterized by irregular fever, hepatosplenomegaly, weight loss, pancytopenia and hypergammaglobulinaemia.^{1,2} There is infiltration of the reticuloendothelial system with amastigotes, which gives rise to the clinical and biochemical features.^{1,2} Splenomegaly is an important feature of the clinical presentation due to the hyperplasia of the reticuloendothelial cells that are filled with parasites.^{1,3} It is reported to be present in 100% patients in one series.² It may be absent in immunocompromised patients such as HIV positive patients, renal transplant recipients, haematological malignancies and patients on long term steroids.^{1,2} Rarely it may be absent in acute cases, or in the early stages of the disease.¹ Our patient had no splenomegaly even though he was immunocompetent. An unusual feature in our patient was the presence of primary adrenal insufficiency. Because serum cortisol concentrations range between 20 and 120 µg/dL during stress in patients with normal adrenal function, a plasma cortisol concentration of less than 20 µg/dL favours a diagnosis of primary adrenal insufficiency along with supporting clinical and biochemical features.⁴ The definition of stress is vague, but conservative criteria include fever above 38° C [100° F], surgical procedures or injuries and gastroenteritis with associated vomiting

and diarrhea.⁴ Adrenal insufficiency is a differential diagnosis of VL, but the absence of a palpable spleen favoured the diagnosis of AI rather than VL in our patient. Eventually, a bone marrow examination done to look for the cause of AI, revealed the presence of LD bodies. One odd feature in the diagnosis of primary AI was the presence of a normal ACTH. The possible reasons for this could be 1) A technical error in performing the test. 2) Initiation of treatment with steroids before performing the test. 3) Presence of secondary AI, but there were no neurological or ophthalmological symptoms and no other hormonal axes was involved. Cosyntropin test could not be done due to the non-availability of the reagent. However, tests for AI rely primarily on measurements of cortisol and its metabolites rather than on measurements of ACTH.³ His skin pigmentation may have been because of kala azar, but mucosal pigmentation is not known in kala azar and was most probably due to AI. VL affects a number of organs, and parasites have also been identified in the heart muscle, the adrenal glands and the parotid glands.¹ In HIV positive patients, CMV regularly involves adrenal glands and *M. avium-intracellulare*, *Cryptococcus* and Kaposi's sarcoma involvement of adrenals have been reported.³ Hence in patients who are co-infected with HIV and LD, the presence of adrenal insufficiency would not be surprising. But our patient was HIV negative and there was no evidence of

tuberculosis [lymph nodes disappeared on treatment with Amphotericin B]. Autoimmune disorders too were ruled out. Antiphospholipid syndrome is another recently discovered cause of AI which may be associated with VL, especially in the setting of catastrophic antiphospholipid syndrome [CAPS], where the infection triggers the catastrophe.^{5,6} But these antibodies were absent in our patient. There are case reports of VL presenting as adrenal cystic masses from which LD bodies have been isolated.⁷ An internet search did not reveal any report of VL presenting as AI. It is difficult to say whether AI was a part of VL or VL precipitated acute AI in a case of chronic AI. It is well known that primary AI is associated with a number of chronic infectious diseases such as tuberculosis, cryptococcosis, etc.⁴ We feel that since VL and AI were co-existing, they were most probably causally related.

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Fig. 1 : The skin pigmentation in kala azar



Fig. 2 : Presence of mucosal pigmentation s/o Addison's disease

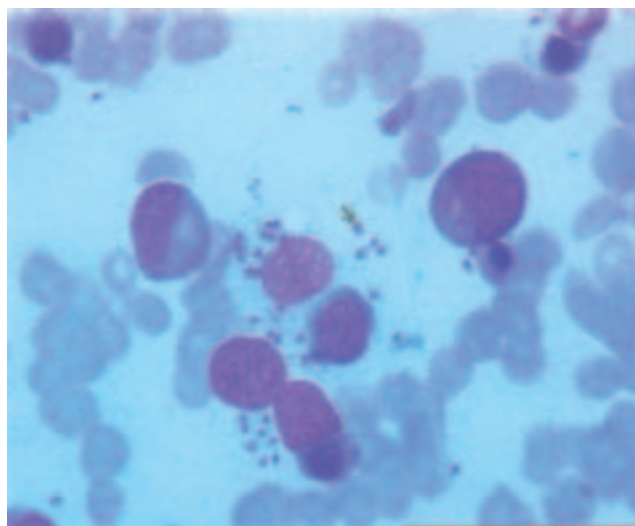


Fig. 3 : Bone marrow showing field full of LD bodies

publish this article. We also thank the Endocrinology Dept for their assistance.

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REFERENCES

1. Jeffrey D Chulay. Leishmaniasis. In : Strickland GT ed. Hunter's Tropical Medicine. 7th ed. Philadelphia: WB Saunders, 1991;638-55.
2. Bryceson ADM. Leishmaniasis. In: Manson ed. Manson's Tropical Diseases. 20th ed. WB Saunders, 1996;1213-45.
3. Williams GH, Dluhy RG. Diseases of Adrenal Cortex. In : Fauci AS ed. Harrison's Principles of Internal Medicine. 14th ed. New York: McGraw Hill, 1998;2035-56.
4. D Lynn Loriaux . Adrenocortical Insufficiency. In : KL Becker ed. Principles and Practice of Endocrinology and Metabolism. 2nd ed. Philadelphia: JB Lippincott Co. 1995;682-86.
5. Oelker's W. Adrenal insufficiency. *N Engl J* 1996;335:1206-12.
6. Asherson RA. <http://www.rheuma21st.com>.
7. Brandonisio O, Fumarola L *et al*. Unusual presentation of leishmaniasis as an adrenal cystic mass. *Eur J Clin Microbiol Infect Dis* 2002;21:682-3.

Surat 1994 Plague – That Never Was!

Sir,

I have read with great interest the review article : "Plague : A decade since the 1994 outbreaks in India" by Angela Clem and S. Galwankar in the May 2005 issue.¹ It is an excellent review of an age-old disease especially in the new context of bioterrorism. The authors rightly conclude that "with prompt diagnosis and treatment the morbidity and mortality due to plague can be greatly reduced, and future out-breaks like those that occurred in Surat and Beed in 1994 can be prevented". Going through the 39 references cited in the review article it is clear that the authors have not taken cognizance of a sizable body of published expert opinion which challenged the conclusion that the 1994 outbreak in Surat and Beed was indeed plague.

Since the last three decades I have regular academic and scientific contacts with the physician community of Surat. I had inspected the Surat Medical College and Hospital on behalf of the Medical Council of India. During the period of September – October – November 1994 I was in frequent telephonic contact with eminent physicians in Surat who did not agree with the hypothesis that there was an epidemic of "Primary Pneumonic Plague" in Surat. I suggested that they postulate and test alternative hypotheses. In view of the newspaper reports of recent flooding (August 1994) in parts of Surat prior to the epidemic, and people wading through muddy water, I suggested two possibilities – leptospirosis and melioidosis and suggested testing samples from the wet soil and stagnant waters for the

organism *Burkholderia pseudomallei*, which gives bipolar staining and "safety pin appearance" similar to *Y. Pestis*. On their invitation I made a visit to Surat to get a first-hand feel of the entire scenario. Although I had no official mandate for any such investigation, the doctors of the Surat Medical College & Hospital extended to me all the courtesies and gave me full access to the case records, chest X-rays and laboratory data of the admitted patients. I discussed with the physician who made the first diagnosis of "Primary Pneumonic Plague", about his thinking about the problem.

Before I visited Surat I had studied the literature of pneumonic plague in India. In September 1983, a disease simulating pneumonic plague hit Tangu village in Himachal Pradesh located at a height of 10,000 feet above mean sea level. After handling a wild rat, the index case developed a primary pulmonary infection and transmitted it to relatives and friends leading to secondary cases. This well-defined human to human transmission involved 22 cases and 17 deaths. The outbreak lasted for three weeks and abruptly stopped after the suspected cases and contacts were effectively treated and control measures instituted (Govt. of India's Report on Sylvatic Plague surveillance during 1984-1992 : NICD Plague surveillance Unit-Bangalore). Since the affected areas were quite inaccessible, samples for laboratory analysis could not be collected properly and the only positive evidence was from smears made from lung tissues of a case at autopsy, which showed presence of organisms morphologically resembling *Y. Pestis*.

Coming back to Surat, the first sputum culture for *Y. Pestis* was reported on 25th September 1994, six days after the original wave of deaths was reported. But unlike the Himachal epidemic, there was not a single instance of person-to-person spread in Surat – all were single cases from different locations. I asked myself : what alternative diagnosis could I offer for this acute onset illness with cough, blood-stained sputum and death within a short period of time ? I could think of ARDS (Adult respiratory distress syndrome) – a descriptive term applied to many acute diffuse infiltrative lung lesions of diverse etiologies, including sepsis and infection : viral, bacterial, fungal, leptospiral, parasitic (plasmodium falciparum) etc. The records showed that some patients had falciparum malaria, some had jaundice.

Judith B. Tysmans – Plague in India 1994 – conditions, containment and goals stated the following : "It is highly probable that workers from Maharashtra traveled to Surat after they had become infected. Living in crowded conditions without medical care or money to pay for it, untreated bubonic plague infections progressed systematically to plague pneumonia and so invariably commenced the rapid person-to-person spread of pneumonic plague".² Scientists must have the courage to accept their pet hypothesis being shattered by the

actual facts which are quite contrary to their speculations.

The 18th conference of the Indian Association of Medical Microbiologists in Pune on 12th November 1994 declared that there was not a single convincing evidence of plague either in Surat or Beed. An expert committee appointed by the Gujarat Government in 1995 under the chairmanship of Dr. NR Mehta, retired professor of Community Medicine in Surat Medical College, concluded that the majority of evidence does not support the diagnosis of Pneumonic plague in Surat. The Expert Committee of the Directorate of Health Services Maharashtra investigating plague epidemic in Beed concluded that the outbreak of plague-like illness was not bubonic plague.

Prof. NS Deodhar (former Director All India Institute of Hygiene of Public Health, Calcutta), Dr. VJ Yemul (Former Director of Haffkine Institute Mumbai) and Dr. Kalyan Banerjee (Director National Institute of Virology Pune) presented a paper at the 3rd National Conference of the Indian Association of Epidemiologists in New Delhi 7th & 8th February 1995 on "Plague that never was". An elaborate paper co-authored by all the three of them appeared in the US Journal of Public Health Policy in 1998 which concluded that the 1994 Surat epidemic was not plague.³ An editorial by VH Talib, SK Khurana and SK Verma entitled "Plague in India 1994 : Was it Really Plague?" appeared in the Indian Journal of Pathology and Microbiology.⁴ Renu Bharadwaj et al reported an outbreak of plague-like illness caused by *Pseudomonas Pseudomallei* in Maharashtra⁵ and suggested *Burkholderia pseudomallei* as a cause of the Indian plague-like illness.⁶

It is worth noting the comments made by expert reviewers of the 1998 paper published by NS Deodhar et al. Abram S. Benenson, editor, Control of Communicable Diseases in man, American Public Health Association, opined that the paper clearly indicates that what was going on in Surat was not plague, certainly not pneumonic plague. There was no rat fall, flea incidences were low, and there were single cases per household and no secondary cases, as would be expected in primary pneumonic plague. Causal organisms were not demonstrated even in "fatal pneumonic plague" cases. None were isolated from material sent to Haffkine Institute. A few plague-like organisms recovered from old specimens were non-pathogenic for mice. The diagnosis of plague was based on serological results using techniques and test kits which were questionable. No four-fold or greater rise in antibody titre was demonstrated in any patient.

Dr. Chin of CDC Atlanta, another reviewer stated that he had participated in an epidemic investigation of plague in Central Java in 1968. The diagnosis was easily made by direct smears of bubo aspirates, confirmed by culture. This evidence was absent from the "bubonic

plague" in Beed in 1994 hence "I heartily agree with the authors' conclusions that there was no plague in India in 1994".

The Technical Advisory Committee (TAC) headed by Prof. Ramalingaswamy, based on PCR studies concluded that the Surat epidemic was plague. The National Institute of Virology Pune also showed PCR bands from DNA extracted from autopsy specimens of two patients from Surat, but they declined to brand it as plague bacillus in view of the discordance with epidemiological, bacteriological and serological evidence. TAC's own findings were that the meticulously isolated plague bacilli were non-pathogenic to mice.

After the publication of Prof. Ramalingaswamy's report I personally met him and suggested the urgent need for a national debate on this enigmatic subject since the TAC itself admits that there were many gaps in their knowledge of Indian plague outbreak of 1994. Prof. Ramalingaswamy agreed with my suggestion but somehow the debate never took place while he was alive, or later on till today. Dr. Satnam Singh, a former programme Director in WHO Delhi Office told the PTI in December 1999 that the same disease which occurred in 1994 continues to occur sporadically in Surat following periodic flooding of the Tapi river, which should be probed further". But evidently no one is now interested in pursuing the matter, till another catastrophe hits!

Current Science in 1996 a special section : The Plague Epidemic of 1994 has printed eight separate papers authored by Prof. V Ramalingaswamy and some of the members of the TAC which give data identifying *Y. pestis* (pp 789-790)⁷ and PCR of *pl* gene (p. 796). Critics of the TAC conclusions especially virologists and microbiologists will have to reconcile with this data. The JAPI review article of May 2005 should be a trigger to initiate a national debate, where protagonists and antagonists come face-to-face and resolve issues.

The endeavour of science is the search of truth. In Medicine there are "contemporary truths" which may change as new evidence comes forth. The American Public Health Association, in the 16th edition of its Manual on Control of Communicable Diseases had mentioned that "an outbreak of primary pneumonic plague occurred in the city of Surat in 1994. But in the 17th edition, this reference was deleted. The trouble with sticking a label such as "primary pneumonic plague" on the Surat Epidemic of 1994 is two-fold : it closes the door for further probing with an unbiased mind, and it sidelines many important issues such as characterization of *Y. Pestis* like organisms, possible mutants, and the specificity of PCR based conclusions, which should be seriously addressed by microbiologists and molecular biologists. In Medicine we are all life-long learners.

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REFERENCES

1. Angela Clem, Galwankar S. "Plague : A decade since the 1994 outbreaks in India". *JAPI* 2005;53:457-64.
2. Judith B. Tysmans – Plague in India 1994 – conditions, containment and goals. *JAPI* 2005; ref. No. 33.
3. Deodhar NS, Yemul VJ, Banerjee K. "Plague that never was". *US Journal of Public Health Policy* 1998;19:184-99.
4. Talib VH, Khurana SK, Verma SK. "Plague in India 1994 : Was it Really Plague?". *Indian J Pathology and Microbiology* 1995;38:131-32.
5. Bharadwaj R, Kagal A, Deshpandey SK, Joshi SA, Khare PM, Junnarkar AR, Phadke MA. Outbreak of plague-like illness caused by *Pseudomonas pseudomallei* in Maharashtra, India. *Lancet* 1994; 344:1574.
6. Bharadwaj R, Kagal A, Deshpandey SK, Joshi SA, Khare PM, Junnarkar AR, Phadke MA. *Burkholderia pseudomallei* and Indian plague-like illness. *Lancet* 1995;346:975.
7. Ramalingaswamy V. The Plague Epidemic of 1994. *Current Science* 1996;71:781-806.

Reply from the Author

Calling for Increasing Strategies to Promote Exchange of Knowledge

Sir,

We welcome Prof. Lele's views about the controversies surrounding the Plague outbreaks in Surat and Beed in 1994. There is no debate to the point that Plague as an Epidemic in 1994 India was controversial. However I am providing a few more references from International Information Documents which suggest that the International Community still goes on to claim in favor of the fact that Beed and Surat outbreaks were Plague and not anything else.

Various studies in our review of literature did point out to the controversy when we were writing the History Component of Plague in our article titled "Plague- A Decade since the 1994 outbreaks in India". In 1997 a study published by Kumar and Colleagues concluded that presence of *Y pestis* antibodies in *Rattus rattus* collected from Beed and Surat were indicative of bubonic plague outbreak in Beed and the pneumonic plague outbreak in Surat during 1994.¹ What really caught our eyes was the Current World Health Organization Documents on South East Asia report that the 1994 epidemics in Surat and Beed were Plague.² Additionally in their Communicable Diseases Surveillance Report on Plague quotes that 1994 was a Plague outbreak in India.³ Also a 2002 Document from WHO states that 1994 and 2002 outbreaks in India were plague.⁴ Currently Plague is considered a Category One Biological Weapon. The Weekly Epidemiological Records Reports from WHO in 2003 stated that 1994 outbreaks in Beed and Surat in India were Plague.⁵

As we see there is a lot debate as regards the fact that whether the 1994 outbreaks in India were Plague. What really is more interesting is that the international Community still reports till 2003 that the 1994 outbreaks in India were Plague. I feel that it will help our colleagues around the Global if they know that there are definite Documents and reports from India that suggest that 1994 Epidemics in India were not Plague. This correspondence has driven home another point apart from the debate regarding diagnosis that we have to escalate the exchange of knowledge between India and the Global Community.

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REFERENCES

1. Kumar K, Jamil-Ur-Rahman et al. Entomological and rodent surveillance in plague-suspected areas during September 1994 and thereafter. *Jpn J Med Sci Biol* 1997;50:97-111
2. The Fifth Decade , on World Health Organization – South east Asia Website Reviewed on 21st May 2005. <http://w3.whosea.org/aboutsearo/88-97-8.htm>
3. Communicable Diseases Reports page on World Health Organization Website on 21st May 2005. <http://www.who.int/csr/disease/plague/impact/en/>
4. Plague Surveillance and Outbreak Response Document from World Health Organization in 2002. http://whqlibdoc.who.int/searo/2002/SEA_PLAGUE_20.pdf
5. Human Plague in 2002 and 2003 in the Weekly Epidemiological Records from World Health Organization. [http://whqlibdoc.who.int/wer/WHO_WER_2004/79_301-08\(no33\).pdf](http://whqlibdoc.who.int/wer/WHO_WER_2004/79_301-08(no33).pdf)

QTc Interval in Idiopathic Epilepsy

Sir,

Long QT syndrome (LQTS) is an important preventable cause of sudden cardiac death in the young, and often presents as seizures.^{1,2} We decided to screen young patients with seizure disorder followed at the epilepsy clinic at KEM Hospital, Mumbai. The inclusion criteria were (i) age < 40years, (ii) inadequately controlled seizures, (iii) treatment with two or more anti-epileptic drugs for at least three months and (iv) no simultaneous use of medication known to affect the corrected QT interval (QTc). The 12 lead electrocardiogram (ECG) of each of these patients was recorded and QTc was evaluated using Bazett's and Fredreicia's corrections.

One hundred and seven patients were studied, 89 were male. The age of the study population was 29 ± 10 years (range 12 – 40 years). The QTc was 403 ± 26 ms. The QTc among males was 396 ± 23 ms and among females was 407 ± 20 ms. No patient had a long QTc (> 450ms in males and > 470ms in females).

Seizures in long QT syndrome states are secondary to torsades de pointes, which require specific therapy and

not anti-epileptic treatment. Channelopathies are found to be responsible for both long QT syndrome and primary seizure disorder.³ The simultaneous presence of both in a patient can be an independent association necessitating definitive therapy for long QTc besides anti-epileptics for optimum seizure control. However, we screened a large population but did not find any patient with epileptic disorder having an undiagnosed long QT syndrome. Nevertheless, this is in the setting of a tertiary care hospital with an experienced neurology faculty in charge of the epilepsy clinic. This may not be generalizable to all settings and we still feel that an ECG should be performed as part of the workup of seizure disorder.

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REFERENCES

1. Pacia SV, Devinsky O, Luciano DJ, Vazquez B. The prolonged QT syndrome presenting as epilepsy: A report of two cases and literature review. *Neurology* 1994;44:1408-10.
2. Bell MR, Kozak RJ. Seizure and the long QT syndrome. *Ann Emerg Med* 1996;28:556-60.
3. Bate L, Gardiner M. Genetics of inherited epilepsies. *Epileptic Disorders* 1999;1:7.

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