Case Report

CONGENITAL VISCERAL LEISHMANIASIS

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ABSTRACT

Visceral leishmaniasis (kala-azar) is caused by the protozoan parasite Leishmania donovani and transmitted by the bite of infected sand fly Phlebotomus argentipes. Only a few cases of congenital transmission have been reported. We are reporting a 15 days old baby with congenital visceral leishmaniasis and then the literature is reviewed.

KEY WORDS: Congenital Visceral Leishmaniasis.

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INTRODUCTION

Visceral leishmaniasis (kala-azar) is caused by the protozoan parasite Leishmania donovani and transmitted by the bite of infected sandfly Phlebotomus argentipes. Other possible routes of transmission are blood transfusion, sharing of needles in drug abusers, organ transplantation, laboratory infection and congenital transmission.[1]

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Visceral leishmaniasis was first described in 1824, in Jessore district of Bengal, what is now Bangladesh.[2] At the turn of the 20th century Visceral leishmaniasis was observed in many parts of India and attracted the attention of Charles Donovan- a British military medical officer. Early in 1903, Donovan in Madras and Leishman in London independently demonstrated the causative parasite in splenic tissue in autopsies from kala-azar patients infected in India.[3,4] Donovan performed a splenic aspirate in a young Indian patient in Madras later in 1903, demonstrating the parasite for the first time in a living patient.[5] Within a few months, Ronald Ross proposed the name *Leishmania donovani* for the newly discovered parasite.[6] In honor of Leishman, the disease associated with this parasite was called leishmaniasis, which is the most widely used terminology in western countries. The global estimates for the incidence and prevalence of kala-azar cases per year are 0.5 and 2.5 million, respectively.[7] More than 90 per cent of the world's VL cases are in Bangladesh, India, Nepal, Sudan and Brazil.[8] Although visceral leishmaniasis cases were reported from 34 of Bangladesh's 64 districts, more than 90 per cent of cases were reported

from just 10 districts namely Mymensingh, Pabna, Tangail, Jamalpur, Sirajganj, Gazipur, Natore, Naogaon, Manikganj, and Rajshahi.[9]

Although true incidences of visceral leishmaniasis in Bangladesh approaches nearly 40,000 to 45,000 per year,[9] no case of congenital visceral Leishmaniasis has still been reported from Bangladesh.

CASE REPORT

A 25 year old lady was admitted in the Department of Medicine with the complaints of fever for four months. The fever started while she was six months pregnant. It was low grade in nature, not associated with chills and rigor and did not follow any particular pattern. She gave birth to a male child 15 days ago. Because she was breastfeeding the child, she kept the baby with herself.

On examination, the lady had anemia and hepatosplenomegaly. Complete blood count showed leucopenia with relative lymphocytosis, ESR was 105. Immunochromatographic test for kala-azar was positive. Both the buffy coat and bone marrow aspirate were positive for LD body, thus confirming the diagnosis of visceral leishmaniasis. She was counseled about the diagnosis and treatment was started with sodium stibogluconate. While in hospital, she sought our advice regarding her baby, because she had noticed that her baby was also febrile since his birth. Out of curiosity, we have sent the blood of the child for immunochromatographic test for kala-azar. Surprisingly, the result came out to be positive. Then for microbiological confirmation, we had consulted the Department of Microbiology of Rajshahi Medical College and asked them to look for LD body in the buffy coat. Buffy coat examination demonstrated presence of LD bodies, thus giving the microbiological confirmation of visceral leishmaniasis in the baby. As the mother first became symptomatic in late pregnancy, and the baby was symptomatic since birth, the mode of transmission of Leishmania Donovani in the baby is likely to be transplacental. The baby was transferred to Department of Pediatrics for further management.

Sodium stibogluconate in a dose of 20 mg/kg was started. About 10 days after starting stibogluconate therapy, the baby became afebrile but developed breathlessness and abdominal swelling. Sodium stibogluconate was stopped and further investigation for evaluation of the situation was planned. But, before adequate evaluation, the mother had left the hospital along with the baby. By that time, mother's treatment was completed, and parasitological cure was demonstrated by negative LD body in bone marrow examination. The baby was not seen since then and his final outcome is also not known. Though this is not a success story, to the best of our knowledge, this is the first reported case of congenital kala-azar in Bangladesh.

DISCUSSION

Congenital visceral leishmaniasis was described first in 1926 by Low and Cooke.[10] Since 1926, only 11 case reports of congenitally acquired VL have been published. Our patient is the 12th case. Most cases have been observed after the mother had VL during pregnancy. Meinecke et al first reported congenital transmission from an asymptomatic mother to her child.[11] The last case was reported in 2006 in a German infant, who never had been to a VL-endemic area. This report also suspected congenitally transmitted Leishmania donovani from asymptomatic mother to her child.[12] During pregnancy, a shift from cell mediated to humoral immunity has been described in mice as well as in humans.[13] Therefore, females may have a higher susceptibility to leishmaniasis during pregnancy, as has been shown recently in mice.[14] This may also suggest that pregnancy can trigger reactivation of leishmaniasis. The course of the disease seems to be identical in congenital transmitted and otherwise acquired kala-azar. Most of the children developed the disease in the first year of life. However, in congenital cases the route of transmission remains unclear. Infection may be transmitted during labor, because of exchange of blood from mother to child, or it may be transplacental transmission. But, a case report from Sudan showed that,

no parasites were found in the organs of an aborted fetus of 5 months' gestational age that was born to a 30-year old infected mother while the placenta showed large numbers of amastigotes.[15] This area of uncertainty requires further probing.

Reducing overall Kala-azar burden in the community is probably the best way to prevent congenital Kala-azar. Fortunately, government of Bangladesh has taken the task of elimination of Kala-azar quite seriously. In 2005 a Memorandum of Understanding was signed by the Health Ministers of Bangladesh, India and Nepal to eliminate kala-azar from the Indian sub-continent by 2015. The target is to reduce kala-azar to less than one per 10,000 people in endemic areas. Active case detection, proper management, and vector control are the key strategies for kala-azar elimination program.

CONCLUSION

In an endemic country like Bangladesh, congenital visceral leishmaniasis is probably more prevalent than known. A very high index of suspicion is required to diagnose congenital visceral leishmaniasis, as both symptomatic and asymptomatic mother can be a source of congenital transmission.

REFERENCES

1. Guerin PJ, Olliaro P, Sundar S, Boelaert M, Croft SL, Desjeux P, et al. Visceral leishmaniasis: current status of control, diagnosis, and treatment, and a proposed research and development agenda. Lancet Infect Dis 2002;2:494-501.
2. Sengupta PC. History of kala-azar in India. Indian Med Gaz 1947;82:281-6.
3. Leishman W. On the possibility of the occurrence of trypanosomiasis in India. Br Med J 1903;1:1252-4.
4. Donovan C. The etiology of heterogeneous fevers in India. Br Med J 1903;2:1401.
5. Donovan C. On the possibility of the occurrence of trypanosomiasis in India. Br Med J 1903;2:79.
6. Ross R. Further notes on Leishman's bodies. Br Med J 1903;2:1401.
7. Life in the twenty-first century: a vision for all. World Health Report 1998. Geneva: World Health Organization; 1998 p. 45.
8. Desjeux P. Human leishmaniasis: Epidemiology and public health aspects. World Health Statist Q 1992;45:267-75.
9. Caryn Bern, Rajib Chowdhury. The epidemiology of visceral leishmaniasis in Bangladesh: prospects for improved control. Indian J Med Res 2006;123:275-288.
10. Low GC, Cooke WE. A congenial infection of kala-azar. Lancet 1926;II:1209–1211.
11. Meinecke CK, Schottelius J, Oskam L, Fleischer B. Congenital transmission of visceral leishmaniasis (kala-azar) from an asymptomatic mother to her child. Pediatrics 1999;104:e65.
12. Boehme C, Hain U, Novosel A, Eichenlaub S, Fleischmann E, Loscher T. Congenital visceral leishmaniasis [letter]. Emerg Infect Dis 2006;12(2):359-360.
13. Wegmann TG, Lin H, Guilbert LJ, Mosmann TR. Bidirectional cytokine interactions in the maternal-fetal relationship: is successful pregnancy a TH2 phenomenon? Immunol Today 1993;14:353–356.
14. Krishnan L, Guilbert LJ, Russel AS, Wegmann TG, Mosmann TR, Belosevic M. Pregnancy impairs resistance of C57BL/6 mice to Leishmania major infection and causes decreased antigen-specific IFN-g responses and increased production of T helper 2 cytokines. J Immunol 1996;156:644–652.
15. El-Toum IA, Zijlstra EE, Ali MS. Congenital kala-azar and leishmaniasis in the placenta. Am J Trop Med Hyg 1992;46:57–62.