LEISHMANIASIS

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Leishmaniasis is caused by a flagellate parasite belonging to the genera leishmania, of which there are 4 main species. The extent of the invasion and the disease produced, varies according to the species involved, and the host resistance. The transmission is by the bite of sandfly belonging to the phlebotomus genus.[1] Although most sandflies are much alike in general appearance,. there are great biologic differences, which result in different epidemiological situations in the world. [2,3] Leishmania organism was discovered by Cunningham in 1885 in the tissues of Dehli boil in Calcutta who gave the name Sporozoa Frumulosa. In 1903, James Homer Wright demonstrated the organism in cutaneous leishmaniasis and named it Heleosma in1904, but it was Luke in 1906 who gave the name Leishmania tropica. [4,5] The taxonomy of leishmania, is moving into a new phase, morphological differences among species and strains are minor even at the electron microscopic level, and are therefore not useful in definition of taxa. Intrinsic physiological, metabolic, antigenic and immunogenic characters of various strains are therefore being considered as the possible basis of molecular and immunological taxonomy rather than a clinical or morphological one. [6] Two important methods of diagnosis in Leislunaniasis are direct microscopic examination of tissue juice aspirated from a Leishmanial lesion, and culture of such tissue juice when parasites are too scarce to be found microscopically. The immunodiagnostic methods in current use include humoral (detection of antibody) and cellular methods (Leishmanin skin testing). Fife[7] has evaluated the test systems now available in the immunodiagnosis of parasitic disease. As immune status of the population is a determining factor therefore serology on a large scale should be undertaken to determine the immune status of the population. Methods such as collection of fmgerprick blood samples on filter paper have been devised to make it possible to process large number of samples. [8,9] As antibody titers of leishmaniasis becomes zero on cure so the presence of antileishmanial antibody in the blood is interpreted as a sign of the continuing infection in the host. [10,11] The natural IgM antibodies in the sera of rodents and lago morphs agglutinate and lyse leishmanial promastigotes but not amastigotes. [12] In nature, this tends to destroy most of the parasites inoculated by sandfly bite. Extensive destruction has been observed within hours of syringe inoculation in hamsters receiving L. tropica promastigote. Human serum also has a direct effect on promastigotes, causing immobilization and some times lysis[13]. As far as the immunology of leishmaniasis is concerned there is a change in the "classical" view exemplified by Adler[14,17] to the view dominated by cell mediated immunity exemvlified by the studies of Garnham and Humphre[8,18-20] and others. It is becoming clear that leishmaniasis are prototypical of a group of chronic diseases caused by intracellular organisms in which cell mediated reactions are paramount[8,9,20]. The basic susceptibility to leishmaniasis is thought to be racial as the disease is present in different races and populations. In other parts of the world people develop a successfill response and over come the disease. There are reports available that the disease was first described by El Razi in Iraq around the year 1500 A.D. However in the latter part of the last century, the disease was recognized by Altounyan in Aleppo Syria and by Borovsky in 1898 in Tash-kent, U.S.S.R. [21] Records are not available as to when the disease was first reported in Pakistan, however it wasin 1935 that a severe outbreak of the disease occurred in Quetta. No definite endemic area has yet been defined[22] however the disease is reported along the entirewestern border extending north into N.W.F.P. down to South West Baluchistan and Northern area of Sind Baluchistan territory. In Punjab, the epidemics have been reported in Multan, Lahore and Dera Ghazi Khan[22,23]. The areas of Baluchistan which show the highest incidence of this disease are Gambaz Maiwand, Kehan, Loherktak,

Tali Tangi, Spin Tangi, Dera Bugti, Sibi. Surrounding areas of. Kohlu, Loralai, Fort Sandeman, Khuzdar and Lasbella. In Sind the diseasà has been reported from Dadu, Larkana and Jacobabad districts. Cutaneous Leishmaniasis also occurs in Northern areas[23], Multan, Quetta, Lasbella and Lahore. The status ofLeishmaniasis in Pakistan has been changing with years. In. 1960 Khaplu valley was the hot bed of disease. In 1964 due to control measures taken these villages had no visceral cases of leishmaniasis. In 1974 in Kharmang valley a new foci was discovered. In 1975, 2 cases were seen in the village of Parkuta. In 1979 the whole of Baluchistan was surveyed and no active case of Kalazar was revealed, but after the 1935 earthquake there was a severe outbreak of cutaneous leishmaniasis in Quetta. Incidence of the disease in Baluchistan is extensive. In 1974 the army personnel who were posted there contracted the disease. In 1971 another outbreak occurred in non immune personnel in Uthal area of Lasbella. In Multan there was an epidemic in 1971-72. One explanation offered for these epidemics is the building up of a nonimmune population in between the epidemics, for the immune response appears to be of utmost importance in resistance to the disease. It takes 15-18 years for the epidemics to recur. In Pakistan the important reservoir for urban areas is the dog and for rural areas, wild rodents e.g. Rhombomysopimus, Meriones humianae, M. lybicus, M. Persicus, M. crassus and other species are reported[21]. To determine the species of sandfly in Pakistan an entomological survey was undertaken in Baluchistan an endemic area in Pakistan. Three species were found i.e. P. sergentii 63.7% and P. papatasii 30.5%, P. sergentomya squamipleuris 5.8%; P. sergentii was found to be the most important vector in causation of disease. [21] In this issue two cases of visceral leishmaniasis have been reported from Karachi especially one case that never left Karachi[24] so the source of infection and method of transmission have yet to be identified here. Also a seroepidemiological study of children from Azad Jammu and Kashmir has been discussed. [25]

REFERENCES

1. Sagher, F. Cutaneous leishmaniasis, in clinical tropical dermatology by Orlando Canzares. Oxford, Blackwell, 1975, p. 186.
2. Lewis, D. J. Phiebotomid sandflies. Bull. WHO., 1971;44:535.
3. Lysenko, A.J. Distribution of leishmaniasis in the old world. Bull. WHO.,1971; 44:515.
4. Farah, F. S. and Malak, J. A. Cutaneous leishmaniasis. Arch. Dermatol., 1971; 103:467.
5. Farah, F. S. Leishmaniasis, In clinical dermatology. Edi. ted by Demis, D. J. Philadelphia, Harper and Low, 1984;4,p.15.
6. Zuckerman, A. Current status of immunology of blood and tissue protozoa. I. Leishmania. Ecp. Parasitol., 1975; 38:370.
7. Fife,E. H.Jr. Advancesin methodologyforimmunodiagnosis of parasite diseases. Exp. Parasitol., 1971; 30:132.
8. Garnham, P.C.C. and I-Iumphrey, J.H. Problems in Leishmaniasis related to immunology. Curr. Top. Microbiol. Immunol., 1969; 48:29.
9. Turk, J. L. and Bryceson, A.D.M. Immunological phenomenon in leprosy and related diseases. Adv. Immunol., 1971; 13:209.
10. Bittencourt, A. L., Sodre, A. and Andrade, Z. A. Pesquisa de antocorpos circulantcs pelo metodo de immunofluorescencia na leishmaniose tegumentar. Rev. do Inst. Med. Trop. Sao Paulo, 1968; 10:247.
11. Ambroise - Tmas, P. Interet de limmunofluorescence dans le diagnostic, le controle post therapeutique et la surveillance epidem oloique des parasitoses. J. Parasitol., 1970; 56:4.
12. Schumnis, G. A. and Flerman, R. Characteristic of so-called natural antibodies in various normal sera against culture forms of Leishmania. J. Parasitol., 1970; 56:889.
13. Bray, R. S. Leishmaniasisin the old world. Br. Med. Bull., 1979; 28:39.
14. Adler, S. Leishmania, in advances in parasitology. Edited by B. Dawes. New York, Academic press,

1964, vol. 2, p.35.
15. Taliaferro, W. H. and Stauber, L. A. Immunology of protozoan infections, in research in protozology. Edited byTze- Teran Chen Pergamon, New York, 1968, v. 3, p.506.
16. Stauber, L. A. Leishmanias, in immunity to parasitic animals. Edited by G. J. Jackson, R. Herman and I. Singer. New York, Appleton-Century-Crofts, 1970, v. 2, p.739.
17. Heyneman, D. Immunology of Leishmaniasis. Bull. WHO., 1971; 44:499.
18. Neal, R. A., Garnham, P.C.C. and Cohan, S. Immunization against protozoal diseases. Br. Med. Bull., 1969; 25:194.
19. Bxyceson, A.D.M., Bray, R.S., Wolstencroft, R. A. and Dumonde, D.C., Immunity in cutaneous leishmaniasis of the guinea pig. Clin. Exp. Immuno., 1970; 7:301.
20. Soulsby, EJ.L. Cell mediated immuno responses and parasitic infections; in immunity to animal parasites. Edited by E.J.L. Soulsby. New York, Academic Press, 1972; p.57.
21. Burney, M. I. and Lari, F. A. Status of leishmaniasis in Pakistan. Pakistan J. Med. Res., 1986; 25:101.
22. Sheikh, N. A. Cutaneous leishmaniasis. JPMA., 1975;25:235.
23. Burney, M. I. Leishmaniasis in Northern areas. Pakistan Armed Forces Med. J., 1962; 12:111.
24. Rahman, M., Rab, S.M., Kazmi, A.K. and Ahmed, A. Visceral leishmaniasis (Kalaazar) in Karachi. JPMA., 1989;39:1243.
25. Rab, M.A., Iqbal, J., Azmi, F.H., Munir, M.A. and Saleem, M. Visceral leishmaniasis “A Seroepidemiological study of 289 children from endemic foci in Azad Jammu and Kashmir by indirect fluorescent antibody technique. JPMA., 1989;39:225.