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RESEARCH ARTICLE

CHANGING CLINICAL PROFILE OF KALA-AZAR IN CHILDREN PATIENT: A HOSPITAL BASED STUDY

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ABSTRACT

Aim: Study to evaluate the clinical Profile, atypical presentation, the drugs in its treatment and to follow up the cases with atypical presentation of children patient with Kala-azar. Methodology: Patients were studied on the basis of history, examination, investigation and diagnosis. Atypical cases were screened and followed up at 1 wk, 3 months and 6 months. Results: Data was analyzed by MS Office software. Conclusions: Kala-azar, in most cases still presents with typical clinical features but cases with atypical presentation is also very common and 10% patients with kala-azar were atypical presentation.

Key Words: Kala-azar, Typical presentation, Atypical presentation.

INTRODUCTION

Visceral Leishmaniasis is the disseminated intracellular protozoal infection of reticuloendotheluial system caused by parasites of genus leishmaina and other kineto-plastedia[1] It is transmitted by the bite of female sandfly phlebotomus argentipus on human host in India. The disease affects both children and adult and nearly half of cases are reported in children (Chatterjee, 2009). The disease got its name kala-azar (kala means black azar means fever) because of dark pigmentation of body in this disease. Other names used for this disease are Dum-Dum fever, sarkari bimari, sahib disease, burdwan fever and Ponus but the name kala azar is mot common term used for visceral leishmaniasis. It is world wide in distribution and occurs in all continents except Australia and Antarctica (Park's, ?). WHO estimated that 350 million people are at risk of infection with leishmania in endemic area (WHO, 1996). An estimate of 5 lacks new cases of visceral leishmaniasis occur annually worldwide and 90% of which occur in India, Bhutan, Nepal Bangladesh & Brazil. Anually 1-3 lakhs kala azar cases are reported in india of which 90% occurs in bihar alone (WHO, 1996). Other areas from where this disease is reported are eastern U.P. and Eastern states like Bengal and Assam i.e. it is prevalent in Gangetic and Brahmaputra belts. Kala-azar is a chronic infection of Reticuloendothelial system characterized by irregular fever of long duration, large spleen and liver (Aiket, 1979). Anemia, leucopenia and progressive emaciation. In recent past increasing number of cases are being observed in the wards which do not have usual documented clinical features and exhibit some unusual presentation like, kala–azar without splenomegaly, Kala-azar with lymphadenopathy, Kala-azar presenting with hepatic encephalopathy causing a lot of confusion in suspecting & diagnosing these cases (WHO, 1996; Aiket, 1979).

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In the present study were studied the 100 cases of Kala-azar and our aims were to evaluate clinical Profile to Kala-azar in Children, evaluate the cases with atypical presentation, evaluate the drugs in its treatment and to follow up the cases with atypical presentation.

MATERIALS AND METHODS

The patients were diagnosed to have kala-azar on the basis of spleen or bone marrow aspirate showing leishmania parasites known as L. D. bodies. Or serologic diagnosis with rk 39 antigen strip were carried out in department of Pediatrics, Katihar medical college, Katihar between Oct. 2012 to september 2014 children aged 2 to 13 years were included in the study and who fulfilled the inclusive criteria were selected. The attendant of entire subject signed an informed consent approved by institutional ethical committee of Katihar Medical college, Katihar, Bihar, India.

Design of Study: Prospective.

Setting of Study: Hospital based Study.

METHOD

After informed consent, each patient was included in this study. Thorough history taking, clinical examination and investigation were done in every case according to Performa: Patients Particular: Name, Age, Sex, Date of Admission, Address with telephone number, Presenting complaints in chronological Like: Fever, Abdominal Distension, Pallor, Loss of Appetite, Loss of weight, Abdominal Pain. History of present illness with particular emphasis on onset, pattern of fever, abdominal distension and its progression with time, pallor and response to earlier treatment. Past History, Family History, Drugs History. General Examination: Pallor, Icterus, Cyanosis, Clubbing, Edema, Lymphadenopathy, Height and weight of patients, Nutritional assessment and general condition, Vitals : Pulse rate, respiratory rate, temperature &

blood pressure and Condition of skin and hair. System examination: for Abdomen: Spleen, Liver, Cardiovascular, Respiratory, and Central Nervous system were examined for any other associated illness. Investigations: Complete Blood Count, Total WBC Count, Differential WBC Count, RBC Count, Hemoglobin Count: Platelets count, Chest X-ray, Liver Function Test, SGOT, SGPT, Serum Bilirubin, Prothrombin Time, Renal function Test, Serum creatinine. Investigation to confirm the diagnosis: Serological test by rK 39, L.D bodies demonstration by splenic or bone marrow smear spienic aspiration

Statistical Analysis: The data was analyzed using the MS Office Software.

Observations: Table 1 to XXX details the result of present study.

Table 1. Age group of patients

Age group	No. of Cases (n-100)	Percentage
2-5 yr	17	17
5-10 yr	49	49
10-14 yr	34	34

Above Table shows age group of patients presenting with kala-azar. Maximum number of patients belonged to 5-10 year age group.

Table 2. Sex distribution of patients

Sex	No. of Cases (n-100)	Percentage
Male	66	66
Female	34	34

Above Table shows sex distribution of patients There was significant male predominance.

Table 3. **Presenting complain of patients**. ***Fever and its Nature***

Fever	No.of Cases (n=100)	Percentage
Nature of fever	100	100
Intermittent	74	74
Continuous	20	20
Double Quotidian	6	6

Above Table shows presence and pattern of fever: all cases presented with fever and pattern was intermittent in most of the cases.

Table 4. ***Abdominal Distension***

Abdominal Distension	No.of Cases (n=100)	Percentage
Present	61	61
Absent	39	39

Above Table shows abdominal distension as presenting complaint of patients presenting with kala-azar. In more than half of the patients, abdominal distension was one of the chief complaints.

Table 5. ***Progressive Paleness of Body (Pallor***)

Pallor	No.of Cases (n 100)	Percentage
Present	54	54
Absent	46	46

Above Table shows progressive paleness of body as a presenting complaint.

Table 6. ***Poor Weight Gain***

Poor weight gain	No.of Cases(n-100)	Percentage
Present	36	36
Absent	64	64

Above Table shows poor weight gain s a presenting complaint of patients. It was present in about one third of patients.

Table 7. ***Abdominal Pain***

Abdominal pain	No. of Cases(n-100)	Percentage
Present	19	19
Absent	81	81

Above Table shows pain in abdomen as as a presenting complaint of patients of Kala-azar.19 % of Patients complained of pain in abdomen.

Table 8. Jaundice with loss of Consciousness *(Hepatic Encephalopathy)*

Jaundice	No.of Cases (n-100)	Percentage
Present	3	3
Absent	97	97

Above Table shows jaundice with loss of consciousness in 3 diagnosed cases of Kala-azar in the present series.

TABLE 9. Painful Red Nodular Lesions Over Leg *(Erythema Nodosum)*

Erythema Nodosum	No. of Cases (n=100)	Percentage
Present	1	1
Absent		99

Above Table shows Erthema Nodosum as an accidental finding in patients of Kala- azar.

Table 10. Findings of clinical examintion. ***(pallor)***

Pallor	No. of Cases (n=100)	Percentage
Present	72	72
Absent	28	28

Above Table shows pallor as finding on clinical examination pallor was present in 72% of patients.

Table 11. Table showing presence of *Splenomegaly* as a Clinical Finding

Splenomegaly	No. of Cases (n=100)	Percentage
Present	98	98
Absent	2	2

Above Table shows splenomegaly as a finding on clinical Examination. Splenomegaly was present in almost all (98%) of cases with exception of 2 atypical cases.

Table 12. Table showing presence of *Hepatomegaly* as a Clinical Finding

Hepatomegaly	No.of Cases (n=100)	Percentage
Present	71	71
Absent	29	29

Above Table shows hepatomegaly as a finding on clinical examination. Hepatomegaly was found in 71% of cases.

Table 13. Table showing presence of *lymphadenopathy* as a clinical finding

Lymphadenopathy	No.of Cases (n=100)	Percentage
Present	4	4
Absent	96	96

Above Table shows that four cases presented with significant lymphadenopathy which is uusual for kala-azar.

Table 14. Table showing presence of *ascites* as a clinical findings

Ascites	NO. of Cases (n=100)	Percentage
Present	8	8
Absent	92	92

Above Table shows Ascites as a finding on clinical examination.

INVESTIGATIONS

Table 15.Table Shows *Haemoglobin Level* Of Patients Studied According To Who Grading Of Anemia

Grade	Severity	Haemoglobin levels	No.of patients	percentage
0	None	Normal	00	00
1	Mild	10 to Normal	16	16
2	Moderate	8-10	58	58
3	Severe	6.5-7.9	17	17
4	Life threatening	<6.5	9	9

Above Table shows that all patents of Kala-azar were anemic and about one fourth cases were having life threatening or severe anemia.

Table 16. Table shows *total leukocyte count* of patients studied

Total No. of cases (n=100)

WBC Count (mm)	Definition	No. of patients	Percentage
<4000	Leukopenia	84	84
4000-11000	Normal WBC Count	13	13
>11000	Leukocytosis	3	3

Above Table shows leukocyte count of patients of kala-azar 84 % of patients having leukopenia,

Table 17.Table Shows *Platelet Count* of Patients Studied

Total No. of cases (N=100)

Platelet Count (per ul)	Category	No. of patients	Percentage
150000-450000	Normal Count	38	38
50000-100000	Mild thrombocytopenia	24	24
20000-50000	Moderate thrombocytopenia	29	29
<20000		9	9

Above Table shows platelet count of patients of kala-azar 62% of patients were having thrombocytopenia of various grades.

Table 18. Table show liver Functions tests (as S.Bilirubin and SGPT) in patients

LFT	Values	No. of cases (n=100)	Percentage
Serum Bilirubin	<0.8mg/dl(Normal)	97	97
	>0.8mg/dl(high)	3	3
SGPT	<45 1U/L(Normal)	97	97
	<45 1U/L(High)	3	3

Above Table shows liver function tests of patents of Kala-azar.3 patients with atypical features showed marked derangements of Liver function tests.

Table 19. Table shows *Renal functions tests* (Blood Urea and Serum Creatinine)

Renal function tests	Normal Values	N0. of cases (n=100)	Percentage
Blood Urea Nitrogen	10-20(mg/dl)	100	100
Serum Creatinine	0.3-1.0(mg/dl)	100	100

Above Table shows renal function tests inn patients of kala—azar. All patients presented with normal renal parameters.

Table 20. Table shows Findings of *Chest X* ray to search for associated diseases and complications

Findings on chest x ray	No. of cases (n=100)	Percentage
Normal	92	92
Pneumonia	4	4
Suggestive of Tuberculosis disease	4	4

Above Table shows finding of chest X-ray in patients of Kala-azar

EVALUATING THE DRUGS IN ITS TREATMENT

Table 21. Table showing *mean Body temperature*, as a response to treatment During Evaluation of Drug in Treatment of Kala-azar Evaluation of Drug in treatment of kala-azar

Time of observation	No. of cases (n=100)	Mean body temperature(in oF)
At Admission	100	101.05
At Discharge	100	98.44

Study showed that mean body temperature reduced to normal levels during treatment.

Table 22. Table showing *Mean Splenic size* as a response to treatment during Evaluation of Drug in Treatment of Kala-azar

Time of observation	No.of cases (n=100)	Mean± S.D
At Admission	98	6.27 c.m
At Discharge	98	1.46 c.m

Study showed significant reduction of splenic size after proper treatment with Amphotericin B.

Table 23. Table showing *disappearance of L.D Bodies* as a response to treatment During Evaluation of Drug in Treatment of Kala-azar L.D Bodies

Time of observation	No of cases (n=100)	L.D Bodies status
At. Admission	100	All positive
At Discharge	100	All Negative

Above Table shows that all patients were L.D. body positive at the time of admission and became negative at the time of discharge.

FOLLOW UP OF ATYPICAL CASES

Atypical cases were followed up for all parameter. But important parameters are shown below.

Table 24 Table showing follow up of cases of Kala-azar presenting with *Hepatic Encephatopathy*.

Serum Bilirubin (mg/dl)				
Cases	Day 0	At 1 wk	At 3 mo.	At 6 mo.
Case1	7.8	5.2	0.6	0.5
Case 2	11.6	8.5	0.8	0.8
Case 3	10.2	7.8	0.7	0.5

Above Table shows that Serum bilirubin levels gradually decreased and came to normal levels during treatment and follow up.

Table 25. Table showing SGPT of Patients presenting with Jaundice. *S.G.P.T (IU/L)*

Cases	Day 0	At 1wk	At 3 Mo.	At 6Mo.
Case 1	1490	710	40	26
Case 2	968	464	38	28
Case 3				

Above Table shows that SGPT levels decreased and gradually returned to normal levels during treatment and follow up.

Table 26. Table showing *Splenic size* of patients presenting with Jaundice during follow up

Splenic Size (In cm)				
Cases	Day 0	At 1 wk	At 3 Mo.	At 6 Mo.
Case 1	4	3.5	0	0
Case 2	6	4.8	0	0
Case 3	11	8.8	2	0

Above Table shows regression of splenic size in patients presenting with atypical feature of hepatic encephalopathy.

FOLLOW UP OF ATYPICAL CASES PRESENTING WITH LYMPHADENOPATHY

Table 27. Table showing *size of lymph nods* during follow up of Atypical cases presenting with lymphadenopathy

Size of Lymph Nodes (cm)				
Cases	Day 0	At 1 wk	At 3 mo.	At 6 Mo.
Case 1	4.5	3.8	2	1
Case 2	3.5	3.0	1.5	1.5
Case 3	3	2.2	1.8	1
Case 4	3.2	2.6	1.2	1

Above Table shows that size of enlarged lymph nodes gradually reduced and became of normal size during treatment and follow up.

Table 28. Table showing *size of spleen* during follow up of Atypical cases presenting with lymphadenopathy

Size of Spleen(cm)				
Cases	Day 0	At 1 wk	At 3 mo.	At 6mo.
Case 1	8	5.4	0	0
Case 2	6	6.8	0	0
Case 3	6	5	1	1
Case 4	9.4	7	2	1

Above Table shows regression of splenic size in patients Presenting with atypical feature of lymphadenopathy.

FOLLOW UP OF ATYPICAL CASES PRESENTING AS APLASTIC ANEMIA

i.e. Absence of Splenomegaly and hepatomegaly

Table-29. Table showing *Haemoglobin levels* of patients presenting with Aplastic Anemia

Hemoglobin Level (in g/dl)				
Cases	Day 0	At 1 wk	At 3 mo.	At 6 mo.
Case 1	6.2	7.8	10	11.6
Case 2	5.9	7.6	9.8	12

Above Table shows that levels of hemoglobin gradually increased during treatment and follow up.

Table 30.Table showing *Total Leukoecyte Count* of Patients presenting with Aplastic Anemia

Total Leukocyte Count (/ul)				
Cases	Day0	At 1Wk	At3 mo.	At6mo.
Case 1	1890	2150	4300	4500
Case 2	2300	2680	4750	4320

Above Table shows that leukocyte counts gradually increased during treatment and follow up.

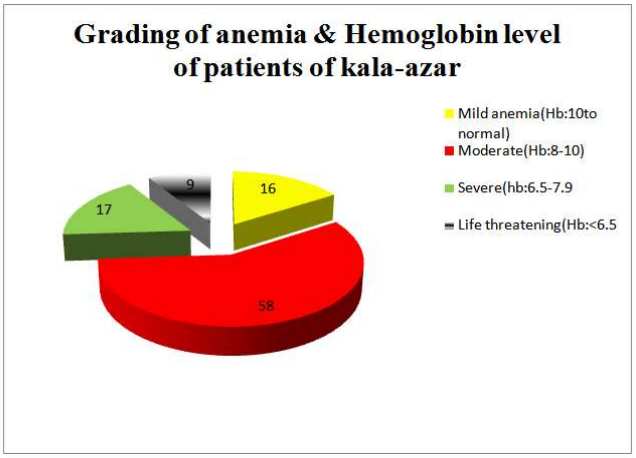

Figure1. Grading of anaemia and hemoglobin level of kala-azar patients

DISCUSSION

Kala -azar is an important public health problem in many parts of the world including India. In India itself Bihar is hyper endemic zone for kala-azar as more than 90% of the cases of Kala-azar are found in Bihar itself. Kala-azar presents with varying clinical featurs in different parts of world. In Bihar itself a number of cases were found to have variegated atypical features. In view of such findings the present study was carried out to review the clinical profile of kala-azar . Present study proved to be fruitful because a sizeable number of cases presented with atypical clinical features. These cases were studied in greater details and were followed up for response to treatment. These cases posed problem in diagnosis but high index of suspicion helped us to diagnose these cases. This study was conducted on 100 patients admitted in indoor of upgraded department of Pediatrics, Katihar medical college Katihar, which were positive for L.D. bodies either by Bone

Marrow Examination or by splenic smear. Various Epidemiological and clinical features of kala-azar Like Age, six, presenting complaints, findings on physical examination, investigations were studied. Response to treatment was also studied in terms of disappearance of fever, Rise in Hemoglobin levels, regression of splenic size and disappearance of L.D bodies.

The cases with atypical presentation were followed up for further evaluation and their response to treatment. In the present study Table 1 shows that the prevalence of kala-azar was maximum in age group 5 to 10 years (49 out of 100 i.e. 49%), next frequent prevalence was in 10 to 14 years (34 out of 100 i.e. 34%) age group while minimal prevalence was noticed in age group 2 to 5 years (17 out of 100 patients i.e. 17%). Children less than 2 years did not presented with features of Kala-azar during our study. Napier *et al*. 1946 in his study of 387 patients had observed that maximum number of patients of Indian VL were between 5 to 15 years of age. These observations are similar to the present study. This high prevalence in 5 to 10 years of age might be due to the fact that children in this age group are very active physically, spend most of their time outside the home like in play orchards, farm house, etc. where there are more chances of contracting disease by bite of sand fly. Besides this, these children usually wear shorts and vest, and therefore most of their body parts are exposed for the bite of sand fly. Children of age group 10 to 15 years had little lower prevalence than 5 to 10 years of child probably because these children usually wear trousers and full sleeves shirt, so that very little part of their body are exposed for the bite of sand fly.

In addition these children are more conscious to the bite of sand fly. Children in age group 2 to 5 years spend that maximum time inside home under guidance and supervision of parents and special care is taken for their clothing and food. So these factors might be contributing for less prevalence of VL, in this age group. Table 11 shows there were 66 males (66%) and 34 females (34%) patients in study with male to female ration 1.94:1. Naik *et al*. 1976 had also found a male predominance, Aiket *et al*. 1979 had reported male/female ration of 1.4:1 Bharat *et al*, and Park had reported a male/female ratio of 2:1 Prasad *et al*. 1987 in his study of 619 cases found male predominance. These observations are more or less in accordance with present study. Whatever the differences is, females are affected less because they spend more time inside home and culture of covering their maximum parts of body with clothes, so sand fly have less chance to bite. In addition to this in our male dominated society, males get preference over females for treatment of any disease. The factor might also be responsible for less number of female reporting with VL.

Table 2, shows fever was presenting symptom in100% of cases and it was intermittent in 74% continuous in 20% and double quotidian in 6% of cases. Thakur *et al*. 1984 observed fever to be the presenting symptom in 98% of the patient and it was intermittent in 77%, continuous in 20% and double rise of temperature in 2% of their study and this observation coincides with the present study. Table 4 shows frequency of abdominal distension as presenting complaints of patients. Causes of Abdominal distension may be: Splenomegaly**,** Hepatomegaly and Ascites etc. Queiroz *et al*. 1995 had reported abdominal distension in 64% cases during their study of 430 cases in Brazil. Our study also matches to the study of Queiroz *et al*. 1995 Table 5. shows Pallor (Paleness)of body as presenting complaint of patients. 54 out of 100 cases (54%) presented with progressive paleness of body a complaint Queiroz *et al*. 1995 also reported symptoms of pallor in 58% of cases. Pallor was due to Anemia which is multi factorial in origin. Contributing factors could be sequestration inside autoimmune hemolysis, shorter half life of RBC's, coombs test positive, hemolysis, G.I. blood loss, malnutrition etc. Table 6 shows poor weight gain as presenting complaints of patients. It was present in 36 out of 100 patients (36%) poor weight gain is due to Malnutrition, Anemia etc. Table 7 shows Abdominal pain as presenting complaints of patients. It was present in 19 out of 100 cases (19%). Abdominal pain may be contributed to organomegaly (enlarged liver spleen) or Ascites. Large spleen may undergo ischemic infarction leading to severe abdominal pain. Associated abdominal infections may cause pain with or without diarrhea/dysentery. Table 8 shows: Cases with jaundice with loss of Consciousness (Hepatic Encephalopathy). 3 cases with jaundice and alteration of consciousness with a diagnosis of hepatic encephalopathy were found to have persistent high fever which was not explained due to any other reason. After a through clinical examination, they were found to have massive splenomegaly and as they belonged to endemic zone for Kala-azar therefore they were subjected to bone marrow examination which showed numerous L.D. bodies. This was an atypical presentation of Kala-azar. Some authors such as Queiroz *et al*,[10](2004) also reported Hepatic insufficiency a cause of death in as much as 31% of cases in a study conducted at Brazil. But jaundice and Encephalopathy is not described as usual clinical feature of Indian Kala- azar.

Table 9 shows that one of our patients presented with painful red Nodular Lesions over legs (Erythema Nodosum).This patient also had moderate grade fever for last 2 months. Fever was suppressed after treatment by local practitioner and patient came with complaints of Erythema Nodosum on detailed clinical examination massive Hepato-splenomegaly was found and Bone marrow examination showed L.D bodies. Erythema nodosum is aninflamamatory reaction in subcutaneous fat. Its occurrence is associated with infections such as beta-hemolytic streptococci, Tuberculosis, coccidiodomycosis, Histoplasmosis and Leprosy. (Robbins patho. Seventh edition 2004). So far presence of erythema nodosum is not described in association with kala-azar. So this is an atypical feature of kala-azar.

Findings of clinical examination

Table 11 shows splenomegaly as a finding on clinical examination. Splenomegaly was present in 98 out of 100 cases (98%) Most of the studies like Thakur *et al*. 1995 Napier *et al*. 1946 reported Splenomegaly in 100% of cases. But our study showed that 2 of our patents did not showed splenomegaly. Actually they both presented with high grade fever with pancytopenia. They were thought to be cases of Aplastic Anemia due to absence of Hepato-splenomegaly, lymphadenopahy and presence of severe pallor. Thus absence of splenomegaly was an atypical presentation of kala-azar. Table 12 shows Hepatomegaly as a clinical feature Hepatomegaly was present in71% of cases Napier *et al*. And Sanyal *et al.* 1976 reported Hepatomegaly in 80% of cases this observation was similar to our study. Table 13 showing Lymphadenopathy as a clinical finding. Significant Lymphadenopathy was present in 4 out of 100 cases of our study.

These cases presented with features of Kala-azar but Lymph nodes were found to be enlarged in cervical area and were 3-5 cm in size multiple, discrete, firm, non tender and mobile. Lymph nodes were not explained by any pathology in these patients even after investigations including FNAC of involved nodes. Smears of Bone marrow. Showed L.D bodies and with the regression in the size of these lymph nodes with the treatment of Kala-azar these nodes were thought to be due to this disease itself. Lymphadenopathy is not a feature of Indian Visceral Leishmaniasis. It is described as a clinical feature of African visceral leishmaniasis. This presence of Lymphadenopathy shows an atypical presentation of Indian Kala-azar. Table 14 shows Ascites as a clinical feature of patients of Kala-azar. Ascites was present in 8 out of 100 cases (8%). Ascites is described as clinical feature in 6% patients by Queiroz *et al*. 2004. Thus our study matched findings of Queiroz *et al*. 2004 Table 15 shows about presence of Anemia in patients of Kala-azar. Anemia was found in all 100 patients of kala-azar. Anemia was graded according o WHO grading of Anemia. Life threatening anemia (Hemoglobin levels <6.5g/dl) was present in 9%of patients. Severe anemia (6.5-7.9g/dl) was present in 17% of cases. Mild to moderate anemia was found in 74% of patients. Anemia was multifactorial in origin contributing factors could be sequestration inside spleen, autoimmune hemolysis, shortened Half life of RBC,s, Coombs test positive Hemolysis ,G.I Blood loss, Malnutrition etc. Anemia was observed a constant feature of in the work of all observed like. Thakur et.al.[9] Table 16 shows total leukocyte count of patients under our study. 84 out of 100(84%) patients were found to have leucopenia.

Only 13% of patients were having normal WBC counts while 3% had leukocytosis. Leukocytosis is not usual but it may be due to associated infections. Table 17 shows platelet count of patient. Platelet count was normal in 38% of patients and 24% of patients showed Mild thrombocytopenia .Moderate thrombocytopenia was observed in 29% of patients. Severe thrombocytopenia <20000/ul was observed in 9% of patients only. Table 18 shows liver function tests in patients of Kala-azar as Serum bilirubin and SGPT. 3 out of 100 patients (3%) showed severe derangement of Liver function tests Actually they presented with Hepatic Encephalopathy.

Hepatic Encephalopathy is reported by Queiroz *et al*. 2004 during their study at Brazil but Indian literature does not describe Hepatic Encephalopathy as presenting feature of kala-azar. So this was an atypical feature of kala-azar. Table 19 shows Renal functions of patients under our study. All patient showed normal values of serum Creatinine and Blood urea. This denotes lack of Renal involvement in Visceral Leishmaniasis. Table 20 shows finding of chest x-ray in our patients. Chest X –ray was done to search for any associated disease with kala-azar. Results of chest X-ray showed normal in 92% of patients while. 4% of patients showed features of Pneumonia .X-ray Features suggesting tuberculosis disease was associated in 4 out of 100 patients in our study. Patients having associated disease were treated with proper antibiotics and they responded well to the therapy.

Evaluation of drugs in treatment of kala-azar

Following observations were found. Table 21 shows mean body temperature reduced during the treatment and became normal at discharge. Amphotericin B was administered to all the patents and all patients responded well to the treatment. Table 22 shows mean splenic size at the time of admission and discharge. Our study showed that splenic size decreased significantly during treatment. Table 23 shows that all patients were L.D body positive at the time of Admission and became L.D body Negative at the time of discharge.

Follow up of atypical cases

Table 24 shows follow up of cases of Kala-azar presenting with Hepatic Encephalopathy. Serum bilirubin of all 3 cases of Kala-azar presenting with Hepatic Encephalopathy was very high at the time of presentation. But it gradually returned to Normal in course of treatment and follow up. Table 25 shows SGPT of patents presenting with jaundice. Initially SGPT of all patients presenting with Jaundice were very high but gradually SGPT improved and became Normal during treatment and follow up. Table 25 shows SGPT of patients presenting with Jaundice. Initially SGPT of all patients presenting with Jaundice were very high but gradually SGPT improved and became Normal during treatment and follow up. 16shows splenic size of patients presenting with jaundice. All patients were having significant splenomegaly which gradual came to normal level during follow up. Similarly Table 27 shows that size of lymphnodes gradually reduced during follow up of cases presenting with lymphadenopathy. Table 28 Splenic size reduced to normal levels during follow up of cases with lymphadenopathy.

Follow up of atypical cases predsenting with aplastic anemia

Table 29 shows that Hemoglobin level of patients increased during follow up of these cases of proven kala-azar showing their good response to treatment thus further confirming the diagnosis. Similarly Table 30 shows total leukocyte count of patients Kala-azar presenting with aplastic anemia. TLC gradually raised to Normal levels during follow up.

Summary and conclusion

Following observations were made during the study: (1) Maximum number of patients were observed in 5-10 yrs of age group. (2) Males were affected more than females in all groups with male/female ration 1.94:1. (3) Fever was presenting complaint in all cases of VL and it was mostly intermittent in Nature. (4) Abdominal distension was one of the chief complaints in 61 % of patients. (5) Progressive paleness of body was due to anemia and was present in about half (54%) of patients. (6) Poor weight gain was present in 36% of cases (7) 19% of patients complained of pain in abdomen. (8) Three patients with jaundice and alteration of consciousness with a diagnosis of hepatic encephalopathy were found to have persistent high fever which was not explained due to any other reason. They had massive Splenomegaly and the bone marrow examination showed numerous L.D. bodies and they completely recovered after treatment of Kala-azar. (9) One patient complained of Erythema nodosum. It is very unusual for Kala-azar to present with Erythema nodosum. (10) On clinical examination 72% of patients were found to be having pallor. (11) Splenomegaly was found in 98% of cases but 2 patients presented with Pancytopenia with absence of Hepato-splenomegaly. Thus initially they seemed to be cases of Aplastic Anemia later proved to be cases of kala-azar on bone marrow examination. (12) Hepatomegaly was present in 71%

of cases. (13) 4% of cases presented with lymphadenopathy which was not explained by any other pathology and they regressed after treatment of kala-azar. (14) On investigations all patients were found to be anemic as much as 25% of patients had severe and life threatening anemia. (15) Most of the patients (84%) were found to be leukopenic. (16) Many patients (38%) had moderate to severe thrombocytopenia. (17)

The three patients presenting with atypical features of Hepatic Encephalopathy had marked derangements of Liver Functions tests. (18) All the patients in present study were found to have Normal Renal Function Tests. (19) During search for associated diseases and complications 8% of patient were found to have pneumonia and tuberculosis. (20) During evaluation of Drug in treatment of kala-azar, all patients including those with atypical presentation responded well to therapy with Amphotericin B without any major complications. (21) During follow up of atypical cases. All atypical cases responded well to treatment and (i) LFT's became Normal in cases with Hepatic Encephalopathy, (ii) Lymph Nodes became of Normal size after treatment,(iii) cases presenting with pancytopenia without splenomegaly and L.D. Bodies in bone marrow also responded well to Am B treatment and their blood counts improved gradually on treatment (iv) Case with Erythema Nodosum als showed gradual improvement of symptoms after treatment with Amphotericin B. After thorough study of all clinical profile and necessary investigations, it was concluded that Kala-azar, in most cases still presents with typical clinical features but cases with atypical presentation is also very common. Our study which included 100 patients found 10 cases of Kala-azar with atypical presentation i.e.10% of total cases, which is a quite significant figure. This large figure of atypical cases which were not documented till date shows change in clinical profile. Therefore high index of suspicion is needed to diagnose all cases of kala-azar in Endemic areas so that one will not miss either typical or atypical presentations of this disease.

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