KALA-AZAR WITH PREGNANCY—CASE REPORTS

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Kala-azar or visceral Leishmaniasis is a disease of tropical country and is caused by Leishmania Donovani which is a protozoa. It used to be quite prevalent in Bihar in previous years, but later on it was totally eradicated. Stray cases started appearing since 1972 but during 1976-78 however it almost took an epidemic form in few districts of Bihar.

Kala-azar with pregnancy is very rare. General debilitation may be affecting fertility directly. Two such cases seen by us are being reported here.

Case 1

Smt. S. D. 25 years an established case of Kala-azar was referred to us by her treating physician on 16th Feb. 1977 for amenorrhoea of 16 weeks. She had an attack of Kala-azar in 1975 when she was diagnosed and treated in Tropical Disease Hospital of Calcutta. L.D. bidies were demonstrated in marrow. The treatment continued for six months and she remained symptom free for one year. Her two brothers also suffered from Kala-azar.

In December 1976 she started having high fever and complained of nausea, vomiting and amenorrhoea of two months. Her last menstrual period being 16th Oct. 1976. Her previous menstrual cycles were regular. Relapse of Kala-azar was diagnosed and she was hospitalised. Anti Kala-azar treatment was restarted.

On admission in the medical ward, she was pale with a pulse rate of 108/mt. The B.P.

being 110/56 mm of Hg. There was no glandular enlargement.

On abdominal examination, the liver was found to be 5 cms. firm and the spleen was 14-16 cms. below the costal margin. The laboratory investigations that were done showed sugar in urine and albumin free and sterile on culture. The Hb% was 40 per cent, Blood examination showed low count of total R.B.C., being 2,250,000/cumm., Total W.B.C. being 2,250/cumm., Polymorph 26 per cent, Lymphocytes 69 per cent, Large mononuclear 5 per cent, Eosinophil and Basnophil was nil. Aldehyale test was positive. L. D. bodies were demonstrated on sternal puncture. Diagnosis of Kala-azar was confirmed.

A course of stibanate was started 6 ml. I.M. daily till (each ml. containing 100 mg Pentavalent antimony) 10 injection and then 6 ml. I.M. on alternate day till 20 injections the total dose being 18 gm. of Pentavalent antimony 2 ml. of Imferron was injected I.M. every alternate day to combat anaemia. Inspite of all treatment her nausea, vomiting, persisted and amenorrhoea continued. She developed jaundice also. It was then that she was referred to us for consultation and management.

On general examination she was anaemic and deeply jaundiced. The pulse being 110/mt., B.P. 110/70 mm of Hg. Cardiovascular and respiratory systems were normal.

On abdominal examination the liver was 5 cms. firm and the spleen was 20 cms below the costal margin. The uterus was 16 weeks, soft and the lower border of spleen was almost touching the uterine fundus. The pregnancy was confirmed by vaginal examination and also by a positive pregnancy test.

The following laboratory tests were carried

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(i) Routine Examination of Urine: Urobilinogen ++ Albumin trace Sugar trace Blood Urea 21 mg. Total protein 7.2 gms. Albumin 2.7 gms. Globulin 4.5 gms. A. G. Ration 1:1.7 S.G.P.T. 12 I.U./L Total Bilirubin 2.7 mg./100 ml. Conjugated Bilirubin 1.6 mg/100 ml. Hb. 40 per cent. Prothrombine Time-14 seconds. Serum Sodium 143 Meg/L Serum Potassium 4.1 MEq/L

The patient was kept in the hospital under strict bed rest. Her Hb% was raised by repeated small packed cell transfusions. 10 per cent fructose used to be given as I.V. drips. Diet rich in carbohydrate and protein was given. A course of Peduculine was also given in the dose of 1 amp. I.M. weekly. She became afebrile in four weeks and her general health showed marked improvement. Her jaundice had also disappeared. The foetus kept on growing according to date, but at 30 weeks of pregnancy the relapse occurred again. She developed fever of remittant type. Jaundice reappeared and she became very weak.

S.G.P.T. had risen to 83/I.U. and serum alkaline phosphatase to 36 A.K. Unit/1000 ml.

Total bilirubin 1.8 mg/100 ml. Conjugated Bilirubin 1.1 mg. Free Bilirubin 0.7 mg/100 ml.

She was again kept on strict bed rest, parentaral glucose, with Vit. B Complex and Vit. C the growing uterus pushed the spleen laterally. The spleen laterally. The spleen did not regress in size.

On 3rd June, 1977 she went into premature labour at 34 weeks and an alive dysmature female baby weighing 2.5 kg. was delivered by low forceps. The Apgar-score was 6 only and movements and relexes were sluggish. The respiration was shallow, and gradually the baby became cyanosed. Inspite of all resuscitative efforts by the paediatrician it could not be revived. The patient left hospital seven days after the delivery when her episiotomy stitches healed.

During this period her spleen had shown slight regression. Another course of Pedunculine 1 amp. I.M. weekly given for 6 weeks.

The patient reported for check up after 6 months. It was very surprising to find that the spleen had almost regressed and was just palpable under the costal margin. Aldehyde test was negative and marrow did not show any L.D. bodies.

In July, 1978, she again attended our out patients department with 2½ months amenor-rhoea with vaginal bleeding. Her general health was good and liver and spleen was not palpable. She was 10 weeks pregnant, with slight bleeding. She was put on conservative treatment for threatened abortion, but 12 hours later she went into inevitable abortion and spontaneously aborted.

Case 2

P. Devi 25 years was admitted on 10th May, 1974 at 32 weeks of pregnancy with history of high fever for last one month. Her general health was good. Her pulse rate was 88/mt. B.P. being 126/80 mm. of Hg. On abdominal examination spleen was just palpable, uterus was 32 weeks, head high with regular foetal heart sound.

Urine examination showed no albumin or sugar. B.Coli was isolated which was sensitive to Septran. Blood picture was within normal limits. Malaria parasites was not seen in blood film.

The temperature responded to Septran and she was discharged with an advice to attend antenatal clinic. Next she reported with high fever 14 days after the delivery had taken place in her village home. Mother and the baby both were running high temperature.

The patient looked ill, anaemic with enlarged spleen of 12 cms. Her Hb. was 58 per cent and the total R.B.C. only 2.8 mm/cm. Total and Diff. W.B.C. count was normal. Blood film showed no malarial parasite. Urine culture was sterile. Aldehyde test was positive, and L.D. bodies could not be easily seen in sternal puncture.

Diagnosis of Kala-azar was confirmed and treatment started with Stibanate. The baby's general condition was also very poor with higher fever and anaemia. The spleen and liver was enlarged. Suspicion of Kala-azar in the baby was also suspected. Aldehyde test was positive but done marrow study was not carried out. Baby was also given Stibanate injection in the dose of 0.1 ml. as test dose and then one ml. I.M. daily for 10 days. Mother

and baby both showed remarkable and rapid improvement with treatment and was discharged after 3 weeks.

Comments

To the best of our knowledge we could not come across any case of Kala-azar complicating pregnancy in the literature. Richard V. Lee (1975) observed that pregnancy in a case of Kala-azar is rare and ends in abortion of still birth. In case 1 inspite of her chronic and prolonged infection she conceived. The risk of splenic rupture or any of its vessel was apprehended but in her condition termination of pregnancy even could not be carried out with safety margins. The baby, though dysmature, showed no congenital malformations.

We missed the diagnosis in the second case on her first visit since urinary infection was more plausible reason for fever and it did respond to treatment.

Since the infection was recent and mild

the pregnancy continued uneventfuly. The development of Kala-azar in the new born baby shows that L.D. bodies cross placental barrier easily. The baby responded very well to the usual Kala-azar treatment.

These two cases showed that pregnancy is not so rare with the Kala-azar and it seems to agravate the condition. In the first case relapse occured during pregnancy only and her condition showed improvement only after delivery. Regression of spleen occured only after foetus was born. In the second case also Kala-azar aggravated as the pregnancy advanced. Another point to be noted that the baby can contact infection from the mother. Foetal salvage is, however, poor in such cases.

References

 Richard V. Lee, Gerandn and Thomas: Medical Complications during pregnancy, Philadelphia, W. B. Saunders, 1975.