Case Report

Hodgkin's Lymphoma

Sharad Bansal[1*], Lalit Une[2], Shraddha Bhutada(Chandak)[3]

[1]Associate Professor, [2]Professor, [3]JR, Department of Paediatrics, JIIU's, IIMS and R Warudi, Jalna, Maharashtra, INDIA.
Email: drsbansal@hotmail.com, lalit_68@yahoo.com

Abstract

Lymphoma is the third most common cancer among children. Hodgkin's lymphoma (HD) is a malignant process of lymphoreticular system, majority B – cell origin that constitutes 6% of childhood cancers.1 HD arises in a single node or chain of nodes and spreads first to the anatomically contiguous nodes. It is characterized morphologically by distinct presence of neoplastic giant cells called Reed Sternberg cells that induce accumulation of reactive lymphocytes, granulocytes and histiocytes.2 Course of the disease is variable but prognosis has improved with modern treatment.
Key Word: Hodgkin's Lymphoma.

[*]**Address for Correspondence:**
Dr. Sharad Bansal, Associate Professor, Department of Paediatrics, JIIU's, IIMS and R Warudi, Jalna, Maharashtra, INDIA.
Email: drsbansal@hotmail.com
Received Date: 03/03/2015 Revised Date: 11/03/2015 Accepted Date: 15/03/2015

Access this article online

Quick Response Code:

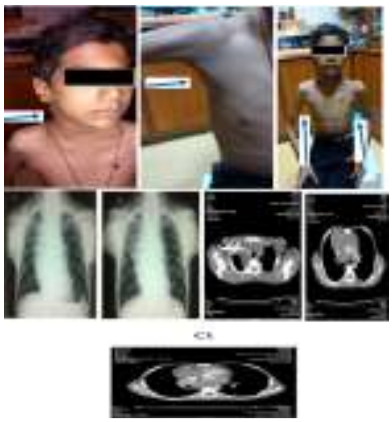
Website: www.medpulse.in

DOI: 18 March 2015

INTRODUCTION

14-year-old male child from village Babhulgaon Tal: Fulambri, Dist: Aurangabad, has come with history of swelling in right side of neck noticed since 2- 3 weeks by parents. There was history of occasional cough, low-grade fever, no loss of appetite, swelling was painless. On examination child was afebrile, vitas were stable, right sided cervical lymphadenopathy +, 2 cm * 2 cm, non tender, non painful, soft in consistency, systemic examination reveals no abnormality. Child was given oral antibiotic and symptomatic treatment and asked to follow up after a week. However, symptoms did not resolve and child also complained of loss of appetite. Hence child was investigated, CBC showed HB – 8.5%, WBC – 7,900/cmm, N 68%,L 28%, Platelets adequate, urine and stool examination was normal, USG abdomen – normal, MT test – negative, CXR showed anterio superior mediastina widening due to ill defined radio density ? lymph node? Thymic shadow. Hence chest CT was done, showed mediastina, hilar as well as lower cervical lymphadenopathy. In view of above reports biopsy of right supraclavicular lymph node was done, showed classic Hodgkin's lymphoma. The HRS cells express CD15, CD 30 and pax 5 and are immunonegative for CD 20 and LCA. Child was advised to start chemotherapy but parents were non willing and they took child to some quack and started some treatment details of which are not available. He continued that treatment for two months but his condition deteriorated further. He developed obvious lymphadenopathy (cervical, axillary), he became more cachexic, fever continued. And hence he came back to us. We started standard protocol of chemotherapy for hodgkins lymphoma which included inj. Adriamycin, inj. Bleomycine, inj. Vinblastine, inj. Dacarbazine for seven cycles in standared recommended doses. Weekly CBC , electrolyte monitoring was done. Clinically child improved gradually, lymphadenopathy regressed, appetite improved, fever decreased. At end of chemotherapy USG abdomen repeated showed decreased size of lymphnodes and no significant abnormality. PET scan done which showed residual nodal lesion in mediastinum in perivascular, right paratracheal and subcarinal regions without significant metabolic activity suggestive of good response to therapy.

How to site this article: Sharad Bansal, Lalit Une, Shraddha Bhutada(Chandak). Hodgkin's Lymphoma. *MedPulse – International Medical Journal* March 2015; 2(3): 132-135. http://www.medpulse.in (accessed 20 March 2015).



DISCUSSION

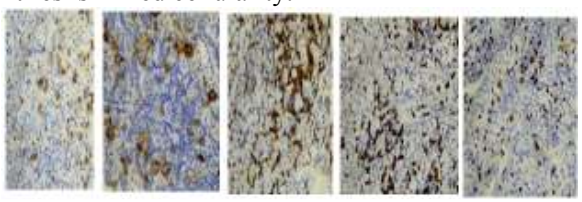
Incidence: Hodgkin's disease (HD) is more common in developing countries than in developed countries.1 It is sixth most frequent childhood malignancy after leukemia, brain tumor, neuroblastoma, non-Hodgkin's lymphoma and nephroblastoma.2 It accounts for 5-6% of childhood malignancy with preponderance in male. (male to female ratio is 2.5:1 to 5:1).[3-8]

ETIOLOGY

The etiology of HD is still subject to various speculations9. The detection of clonal EBV genome in the tumor cells of HD, indicates that EBV infection preceds the expansion of the neoplastic clone10. EBV associated childhood HD is more prevalent in developing countries than in developed countries , and in male below 10 yrs of age and in mixed cellularity type11. Other mechanism involved in pathogenesis of HD, such as disturbance of the cell cycle and apoptosis regulation. A genetic susceptibility to HD has been suggested by rare cases of familial HD.[12-14]

PATHOLOGY

Histopathological features of HD include partial or total effacement of nodal architecture and replacement by an inflammatory cellular background of histiocytes, lymphocytes, eosinophills and plasma cells containing Reed – Sternberg cells. RS cells are binucleate or multinucleate giant cells with prominent nucleoliand abundant cytoplasm. Hodgkin's cells are variant of Reed Sternberg cells. Tumor cells of classical HD are characterized by a CD 30 positive, frequently CD 15 positive and CD 45 negative phenotype, while T cell and B cell associated antigens are usually negative. Classical Hodgin's lymphoma includes four subtypes : mixed cellularity, nodular sclerosis, lymphocyte depletion and lymphocyte rich classical Hodgkin's lymphoma. The most common subtype found in children from developing countries is mixed cellularity.[15-18]



CLINICAL FEATURES

Clinically child with HD presents with painless cervical or supraclavicular lymphadenopathy usually unilateral , firm and rubbery, sometimes fluctuant. It originates in single lymphnode and then spreads to involve contigious nodes and organs. Mediastinal lymphadenopathy is seen in 50 % patients and may cause superior mediastinal syndrome.[19]

Recommended work up for clinical staging:[19]

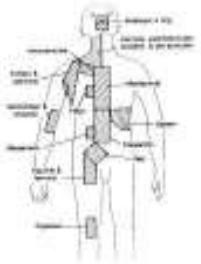


Fig. 1: Anatomic definition of separate lymph node regions (from Hudson and Donaldson[9])

Measurement of palpable lymph nodes, liver and spleen

Complete blood count, ESR

Liver Function tests, Serum Alkaline phosphatase (SAP)

Chest radiograph

Chest contrast enhanced computerized tomography (CECT)

Neck CECT, if high cervical nodes palpable

Abdominal CECT or MRI

Bone marrow biopsy except in stage IA

Bone scan, if elevated SAP level and/or bone pain

Ann Arbor staging classification:[19]

Medscape® www.medscape.com

	Description
Stage I	Involvement of a single lymph node region or lymphoid structure (eg, spleen, thymus, Waldeyer ring)
Stage II	Involvement of 2 or more lymph node regions on the same side of the diaphragm
Stage III	Involvement of lymph node regions on both sides of the diaphragm
	III1: With or without involvement of spleen or hilar, celiac, or portal nodes
	III2: With involvement of para-aortic, iliac, or mesenteric nodes
Stage IV	Involvement of extranodal site(s) beyond that designated E
A	No symptoms
B	Unexplained fever ≥ 101.5°F, drenching night sweats, loss of >10% body weight within the previous 6 mo
X	Bulky disease: >1/3 the width of the mediastinum; >10 cm maximal dimension of nodal mass
E	Involvement of a single extranodal site, contiguous or proximal to a known nodal site
CS	Clinical stage
PS	Pathologic stage

Source: American College of Nurse Practitioners © 2007 Elsevier Inc.

DIAGNOSIS

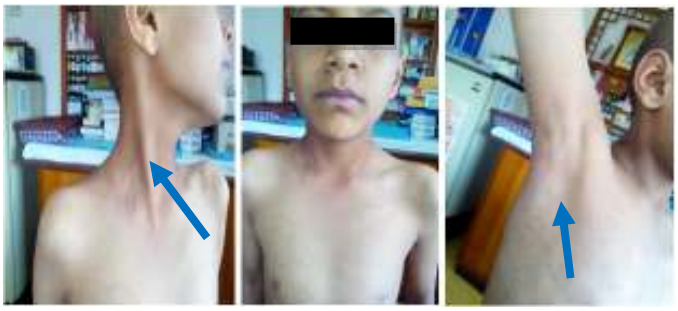
Diagnostic work up for HD includes complete hemogram, ESR, liver and renal function tests. Imaging studies include upright postero-anterior X-ray of chest with lateral view. Contranst enhanced computerized tomography of chest , abdomen and pelvis. An excision biopsy or needle biopsy of at least one enlarged lymph nodes is mandatory to establish the diagnosis.[19]

TREATMENT

Most pediatric protocols prescribe multiagent combination chemotherapy, either alone or with low dose involved field radiation. (IFRT) Addition of radiation to combination chemotherapy has improved disease free survival in patients with bulky disease and presence of B symptoms.

The most commonly used combination chemotherapy regimen includes 6-8 cycles of MOPP (nitrogen mustard, vincristine, procarbazine, prednisolone). Another regimen called ABVD (Adriamycin, Bleomycin, Vinblastine, Dacarbazine) either alone or alternating with COPP (Cyclophosphamide, Oncovin, procarbazine,prednisolone) may be used with more success.

With the highest current cure rate in Hodgkin's disease an increasing attention is focused on minimizing the late complication of therapy. In growing children low dose ,involved field radiation therapy is preferred along with combination chemotherapy in advanced stage Hodgkin's disease. High dose radiation therapy should be avoided in young children because of its late complications such as diminished growth of soft tissue and bone, hypothyroidism, gonadal dysfunctions, secondary malignancy.[20-22]



CONCLUSION

All enlarged cervical lymphnodes is not tuberculosis. Protocol based work-up is required for the correct diagnosis of the cause of cervical lymphadenitis. Early diagnosis and timely treatment of Hodgkin's Lymphoma yields good outcome. Treatment results of childhood HD have enjoyed such progress over years that HD is one currently of the most curable cancers in children. With the multiagent chemotherapy either alone or in conjunction with low dose involved field radiation therapy; 5 year survival rate is over 90% in early stage disease and 50- 70% in advanced stage disease. Yet more aggressive protocols are required to improve long term survival in unfavorable and advanced disease as well as relapsed cases.

REFERENCES

1. Macfarlane GJ, Evstifeeva T, Boyle P, Grufferman S. International patterns in the occurrence of Hodgkin's

disease in children and young adult males. Int J Cancer 1995; 61: 165-169
2. Young JL jr.,Ries LG, Silverberg E, Horm JW, Miller RW. Cancer incidence, survival and mortality for children younger than 15 years. Cancer 1986;58:598-602.
3. Stiller CA, parkin DM. Geographic and ethnic variations in the incidence of childhood cancer. Br Med Bull 1996;52(4):682-703.
4. Yeole BB, Advani S.H. and Sunny L. Epidemiological features of childhood cancers in greater Mumbai. Indian Pediatric 2001;38(11):1270-1277
5. Mac Mathon B. Epidemiology of Hodgkin's disease. Cancer Res 1966;26:1189-1200
6. Sackmann – Muriel f, Zubizarreta P, Gallo G, Scopinaro M, Alderete D, Alfaro E, *et. al...* Hodgkin's disease in children: results of prospective randomized trial in a single institution in Argentina. Med Pediatr Oncol 1997;29:544- 552
7. Cavdar AO, Pamir A,Gozdasoglu S,Babacan E, Yavuz G, Umal E, *et. al...* Hodgkin's disease in children: clinicoepidemiologic and viral (EBV) analysis. Med Pediatr Oncol 1999;32:18-24
8. Arya LS, Thavaraj V, Dawar R, Rath GK,Jain Y, Kumar R *et. al...* Hodgkin's disease in Indian children:report from a major referral centre. Med Pediatr Oncol 2000;35(3) :203 (abstract)
9. Levine PH, Ablshi DV, Berald CW, Carbone PP,Waggoner DE, Malan L. Elevated antibody titres to EBV in Hodgkin's disease. Cancer 1971;27:416-421.
10. Weiss LM,Strickler JG, Warnke RA, purtillo DT, Sklar J. Epstein Barr viral DNA in tissues of Hodgkin's disease. Am J pathology 1987;129:86-91
11. Dinand V , Arya LS. Epideilogy of childhood Hodgkin's disease: is it different in developing countries? Indian pediatr 2006;43(2):141-147
12. Robertson SJ,Loerman JT, Grufferman S, KostyuD, vander Horst CM, athews TJ, *et. al...* Familial Hodgkin's disease: a clinical and laboratory investigation. Cancer 1987;59:1314- 1319
13. Mack TM, Cozen W, Shibata DK,Weiss LM, Nathwani BN, Hernandez AM,*et. al...* Concordance for Hodgkin's disease in identical twins suggesting genetic susceptibility to the young adult form of the disease. N Engl J Med 1995;332(7):413-418
14. Shugart YY,Hemminki K, Vaittinen P, Kingman A, Dong C. A genetic study of Hodgkin's lymphoma an estimate of heritability and anticipation based on familial cancer database in Sweden. Hum Genet 2000;106(5):553-556.
15. Hodgkin T. On some morbid appearances of the absorbent gland and spleen. Med Chir Trans 1832;17:62-114
16. Sternberg C. Uber eine Eigenartige unter dem blide der Pseudoleukemia verlaufende Tuberculose des lymphatischen. Apparates Z Heilkd 1898;19:21-90
17. Reed DM, On the pathological changes in Hodgkin's disease , with special reference to its relation to tuberculosis. Johns Hopkins Hosp Rep 1902; 10:133- 396
18. Lukes RJ , Butler JJ. The pathology and nomenclature of Hodgkin's disease. Cancer Res 1966; 26: 1063-1083.
19. M. R. Lokeshwar, Nitin Shah, Bharat Agrawal, Anupam Sachdeva, IAP speciality Series on Pediatric Hematology oncology 2006; 32: 352-355
20. Hudson MM and Donaldson SS. Hodgkin's disease. Pdeatric Clin North Am 1997;44: 891-906
21. Kapoor G, Advani SH, Dinshaw KA, *et. al...* Treatment results of Hodgkin's disease in indian children. Pediatr Hematol oncol 1995; 12 : 559-69.
22. Landman –Parkar J, Pacquement H, Leblanc T, *et. al...* Localized childhood hodgkin's disease: response adapted chemotherapy with etoposide, bleomycine, vinblastine and prednisolone before low dose radiation therapy – Results of the French Society of Pediatric Oncology study MDH -90. J Clin Oncol 2000; 18: 1500-1507.

Source of Support: None Declared
Conflict of Interest: None Declared