Hodgkin Lymphoma/Hodgkin Disease (HL; HD)

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Cure4Kids Release Date: 1 September 2006

Hodgkin Disease (Hodgkin Lymphoma) is a (A – 1) lymphoid system malignancy, first described by Sir Thomas Hodgkin in 1832. Though the exact cause of Hodgkin lymphoma is unknown, molecular studies suggest that the characteristic Hodgkin cell is an aberrant B lymphocyte. However, the three distinct forms of the disease suggest several different causes for Hodgkin.

Childhood form – patients 14 years and younger
Young Adult form – patients 15 to 34 years old
Older adult form – patients age 55 years and older

Hodgkin lymphoma is one of the success stories in cancer therapy. Previously fatal, the disease can now be successfully treated with multimodal and multi-agent therapy.

Risk Factors

Epidemiological studies indicate that besides a bimodal age distribution, there is a difference in disease incidence between developing and industrialized countries.

In industrialized countries, the disease first peaks in the mid 20’s, with a second peak after age 50; it is also associated with a higher socioeconomic status.

In developing countries, the incidence increases in adolescence, peaking during early adult years.

Overall, Hodgkin disease seems to be more common in the United States, Latin America, Africa and Israel than in other regions (Liebhausser, 2002, p. 525).

Rarely seen in children less than 5 years of age, the disease is slightly more common in males than females, clustering within families (suggesting a possible genetic predisposition and/or common exposure to a causative agent), especially in same-sex siblings and twins. Hodgkin disease is also more common in persons who have (A – 2) immune deficiency.
Hodgkin’s Lymphoma/Hodgkin’s Disease (HL; HD)

Classification and Staging

Sternberg (1898) and Reed (1902) first described the presence of abnormal giant cells (Reed-Sternberg Cells) common in Hodgkin tumor specimens. Classical Hodgkin lymphoma is divided into 4 subtypes according to the number of Reed-Sternberg cells, the characteristics of the inflammatory milieu, and the presence or absence of fibrosis.

A-4 Classical Hodgkin lymphoma is divided into 4 subtypes:

- Lymphocyte-rich classical Hodgkin lymphoma (LRCHL).
- Nodular sclerosis Hodgkin lymphoma (NSHL).
- Mixed-cellularity Hodgkin lymphoma (MCHL).
- Lymphocyte-depleted Hodgkin lymphoma (LDHL).

Another type of HL is the nodular lymphocyte predominant (NLP) Hodgkin lymphoma. NLP is a category of its own. In NLP, the typical Reed-Sternberg cells are rare to non-existent; instead, variants called L & H cells (popcorn cells) are seen. Other distinctive clinical features of NLP include:

- an indolent though relapsing course with an excellent prognosis
- occasional cases relapsing as high-grade B-cell non-Hodgkin lymphoma
- a peak incidence in males in their 30’s and 40’s with out the bimodal age pattern of classic HL
- a greater tendency to be restricted to cervical lymph nodes.

Staging determines the extent of the disease at the time of diagnosis. The Ann Arbor Staging classification describes the stage of Hodgkin disease at the time of presentation.

Clinical Stage (CS) uses history, physical examination, radiologic and other imaging studies, laboratory tests, and initial biopsy results to determine the extent of disease.

Pathologic stage (PS) is used when a staging laparotomy provides histologic conformation of the presence or absence of lymphoma involvement of specific sites. Because of the risks involved in the staging laparotomy procedure, it is done only if the findings will significantly alter the therapy.

Clinical Presentation:

- Slowly expanding, non-tender, firm, rubbery, and movable lymph nodes mostly in the cervical, axillary, or inguinal areas.

(A – 9) Mediastinal mass commonly seen with cervical adenopathy; the patient may complain of pressure on the trachea and bronchi causing them to have respiratory difficulties such as dyspnea and a dry, non-productive cough. The
mediastinal mass may cause the patient to experience superior vena cava syndrome (SVCS)

- Organomegaly (spleen and liver)

### Systemic Symptoms:

Non-specific systemic symptoms include anorexia, weight loss, fatigue, unexplained fever (>38°C or 100.4°F), night sweats, and pruritus. Since some systemic symptoms correlate with prognosis, they are also classified according to the staging process as “A” (asymptomatic) or “B” (constitutional) disease. The triad of symptoms that define “B symptoms” includes:

1. Presence of unexplained fever for 3 consecutive days
2. Drenching night sweats
3. Unexplained weight loss of more than 10% of body weight within 6 months of diagnosis.

If no “B” symptoms are present, the patient is classified as “A”. Favorable prognosis is associated with the absence of “B” symptoms and bulky disease. Unfavorable prognosis is correlated with the presence of “B” symptoms, bulky mediastinal or peripheral lymphadenopathy, extra nodal, and stage IIIB and IV disease.

### Diagnostic Workup:

- Complete history of the illness: fever, night sweats, weight loss, fatigue, and pruritus.
- Physical exam: peripheral lymphadenopathy and presence of hepatosplenomegaly.
- Chest x-ray: presence of a mediastinal mass and hilar adenopathy.
- CT scan or MRI: chest, abdomen, pelvis, neck, lymphadenopathy, hepatosplenomegaly with areas of abnormal density.
- Ultrasound imaging: bulky tumor margins, spleen, and liver size.
- Gallium scan: increased uptake in untreated disease; persistent uptake in the presence of residual disease. Most useful in supra-diaphragmatic HD.
- Bone scan: clinically indicated if the patient has bone pain, increased serum alkaline phosphatase (indicative of bone metastasis).
- Lymphangiogram (LAG): retroperitoneal lymph node involvement; also identifies specific nodes for biopsy; also used to design radiotherapy treatment fields. The dye stays in the lymph nodes for several months and is used to monitor nodal response to treatment. LAG is rarely used now.
- Lymph node biopsy: histologic classification and the presence of Reed-Sternberg cells are often diagnostic.
- Laboratory studies: CBC with differential; ESR; renal and hepatic function tests; urinalysis; some centers perform additional optional tests such as measurement of serum copper, fibrinogen, haptoglobin, immunoglobulins, ferritin, transferritin, LH/FSH, T-cell and B-cell counts, T-cell function studies.
- Surgical pathology: peripheral lymph node biopsy, possible exploratory laparotomy and splenectomy in rare cases where staging is unclear.
- Staging: Ann Arbor Classification System, modified at the Costwold Conference. (Hockenberry-Eaton, 1998)
Medical Management

Therapeutic goal for HD is cure with minimal treatment-related toxicities and sequelae. The Children’s Oncology Group (COG) uses “response-based therapy” to improve efficacy. Response-based therapy provides adjusted intensity of therapy according to the patient’s treatment responses. Patients who are good responders will have optimal therapy while poor responders will have more intensive therapies.

Most current pediatric protocols include three to six cycles of multi-agent chemotherapy and 20 to 25 Gy of radiation therapy to involved fields. Combined multi-modal and multi-agent chemotherapy regimens offer efficient disease control and cure. Through combination multimodal therapy, cumulative chemotherapy dose and radiation volume are reduced, resulting in reduced toxicity and late effects of therapy. In children with localized disease (limited to the upper neck or inguinal lymph nodes), radiotherapy alone appears may be sufficient, but chemotherapy is usually used so that the dose of radiation therapy can be reduced.

Chemotherapy

Chemotherapy is usually given before radiation therapy. The most common rationale for chemotherapy is to reduce bulky disease, allowing lower doses of radiation. To date, there are several (A – 10) chemotherapy regimens used in the treatment of children with Hodgkin lymphoma.

Radiotherapy

The size and extent of radiation therapy depend on the tumor involvement and whether chemotherapy is also used. In young children, the standard of care is low-dose (A – 11) involved field radiation plus chemotherapy. This practice prevents retardation of bone growth and soft tissue development. For patients who are fully grown (adolescents and adults), the standard is high-dose extended field radiation.

Relapse

Most relapses for Hodgkin disease occur within the first 3 years after their initial diagnosis; however, patients are at risk for relapse up to 10 years after diagnosis. Following primary therapy, patients who experience relapse from Hodgkin disease are categorized into the following subgroups:

1. Never achieved a complete remission
2. Brief complete remission (≤12 months)
3. Multiple relapses after one or more conventional chemotherapy regimens
4. Prolonged complete remission (≥12 months)

Patients in the first three subgroups are candidates for autologous bone marrow or stem cell transplantation procedures.
Relapses, especially at untreated sites, tend to maintain the histologic subtype of the original lesion. Localized relapse is also more responsive to salvage treatment than widespread relapse. The chemotherapy regimen for advanced-stage disease is usually the regimen prescribed for patients who have relapsed. Radiotherapy is suitable for disease that recurs outside the previous radiotherapy fields and salvage chemotherapy can be administered with non-cross resistant drug combinations. Complete response rates of 25% to 50% have been achieved with treatment of relapsed disease. (Baggott, et al, 2002).

Second Malignancies

Hodgkin's disease is associated with several secondary malignancies that are often treatment-related. Secondary acute non-lymphocytic leukemia (s-ANLL) and myelodysplastic syndrome (MDS) are the most common secondary hematologic malignancies and commonly occur 5 to 10 years after treatment. Other associated malignancies include non-Hodgkin lymphoma and other solid tumors of the lungs, breast, gastrointestinal tract, and thyroid. The risk of breast cancer is greatly increased when the breast is included in the radiation therapy port.

Future Directions

As the biology and clinical characteristics of lymphoma become better known, novel therapies for this group of diseases will evolve. Although combined modality therapy has greatly increased survival rates for children with Hodgkin lymphoma, novel risk-adapted protocols are currently being investigated to further reduce acute and long-term treatment side effects without compromising cure rates.

The use of antibodies to deliver radiation directly to the cancer cells and drug combinations with fewer side-effects is currently being studied. Such a treatment is expected to improve patient quality of life and to benefit those patients who are not able to withstand the side-effects of current treatments.

One new, experimental approach to immunotherapy is a non-myeloablative allogeneic stem cell transplant (“mini transplant”). In this procedure, the patient receives stem cells from a compatible donor (sibling or parent) plus enough chemotherapy to allow the transplant to take. Once the transplant takes, the subsequent immune response to the cancer may shrink the tumor.

Other novel therapies include drugs that trigger self-destruction of lymphoma cells with minimal side-effects. Such drugs are currently in Phase I studies and are being combined with monoclonal antibody (Rituximab and Fenritinde) therapies in an attempt to increase their efficacy.
Hodgkin’s Lymphoma/Hodgkin’s Disease (HL; HD)

Helpful Web Links:

Wikipedia.org
Hodgkin Lymphoma

St. Jude Children’s Research Hospital, Memphis, TN
http://www.stjude.org/disease-summaries

Information-on-Hodgkins
http://www.information-on-hodgkins-disease.com

The National Cancer Institute
http://www.cancer.gov/cancertopics/pdq/treatment/childhoodhodgkins/healthprofessional

Lymphoma Information Network
http://www.lymphomainfo.net/childhood/hodgkins.html

Related www.Cure4kids.org Seminars:

Seminar #768 Hodgkin Disease
Presenter: Monika Metzger, MD
http://www.cure4kids.org/seminar/768

Seminar #420 ALL, Hodgkin & Non-Hodgkin (in Spanish)
LLA, Hodgkin & No Hodgkin
Scott Howard, MD, MS
http://www.cure4kids.org/seminar/420

Seminar #366 Refractory and Relapsed Hodgkin Disease
Gregory Hale, MD, Melissa Hudson, MD and Matthew J. Krasin, MD
http://www.cure4kids.org/seminar/366

Seminar #97 Hodgkin’s Disease
Melissa Hudson, MD, Sue C. Kaste, DO, Mihaela Onciu, MD and Matthew J. Krasin, MD
http://www.cure4kids.org/seminar/97

Seminar #165 Childhood Hodgkin Lymphoma and Epstein-Barr Virus (EBV)
Scott Howard, MD, MS and Jeffrey T. Sample, PhD
http://www.cure4kids.org/seminar/165
Appendix:

A – 1 The Lymphatic System and sites of Lymphoma

Lymphoma can present anywhere normal lymphocytes – blood cells that provide immune defense – are found.

N  lymph node
H  liver (hepatic)
L  lung (not shown)
B  bone marrow
S  spleen
P  pleura (lung lining – not shown)
O  bone
D  skin
M  mucosal linings – nose, stomach, eyes, etc.

Lymphoma is not one cancer, but an aim for a group of related cancers that arise when a lymphocyte (an immune cell) becomes malignant.

When a lymphocyte becomes malignant it’s biological behavior is arrested at its stage of development.

Lymphoma cells may grow to form a solid, accumulate to form tumors in the body, most commonly in the lymphatic system – the network of lymph nodes and channels that filter blood and

Lymphomation.org, Reigelsville, PA
http://www.lymphomation.org/about-details.htm

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A – 2 Immune Deficiency Conditions Associated with Hodgkin’s Disease

Wiskott-Aldrich Syndrome (WAS) is a rare, X-linked, recessive, combined immunodeficiency disorder caused by partial defects in T and B lymphocytes. WAS is usually expressed fully in males and characterized by thrombocytopenia, scaly, itchy skin, and rashes (eczema).

Ataxia-telangiectasia (A-T) is a complex multi-system autosomal recessive disorder characterized by progressive neurologic impairment, cerebellar ataxia, variable immunodeficiency with susceptibility to sinopulmonary infections, impaired organ maturation, x-ray hypersensitivity, ocular and cutaneous telangiectasia, and a predisposition to malignancy.

Human Immunodeficiency virus infection (HIV) causes AIDS-related lymphoma--a disease in which cancerous (malignant) cells are found in the lymph system in patients who have AIDS (acquired immunodeficiency syndrome). (HIV slowly damages the immune system for a number of years after infection.)

Epstein-Barr Virus (EBV) is the herpesvirus that causes infectious mononucleosis and is also associated with various types of human cancers. It is a pervasive virus, infecting a large percentage of people in both industrialized and low resource nations. Latent EBV infections are often associated with AIDS and lymphomas in immunocompromised patients.
A – 3  **Reed Sternberg Cells** are large cells that have 2 nuclei, which give them an “owl’s eye” appearance. Although common in Hodgkin disease, they can also be found in other disorders such as infectious mononucleosis.

The characteristic cell, the Reed-Sternberg cell, is a bizarre, gigantic cell with 2 or more large nucleoli, each enclosing a large, central, scarlet nucleolus with clear space around it.

The two center cells, Reed-Sternberg cells, are characteristic for Hodgkin lymphoma.

**Sydney Children’s Hospital, Australia**  
http://www.kids-cancer.org/what_is.htm  
**Surgical-Tutor.org.uk**  
www.surgical-tutor.org.uk/pathology/pathology1.htm
## Rye Histologic Classification of Hodgkin Disease

<table>
<thead>
<tr>
<th>Histologic Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphocytic predominance</td>
<td>Numerous small benign-appearing lymphocytes and/or reactive histiocytes</td>
</tr>
<tr>
<td></td>
<td>Nodular or diffuse</td>
</tr>
<tr>
<td></td>
<td>No necrosis; fibrosis</td>
</tr>
<tr>
<td></td>
<td>Rare Reed-Sternberg cells (difficult to find)</td>
</tr>
<tr>
<td>Nodular sclerosis</td>
<td>Lymphoid tissue is divided into nodules by collagen bands</td>
</tr>
<tr>
<td></td>
<td>Nodules contain atypical histiocytic cells in clear spaces (lacunar cells)</td>
</tr>
<tr>
<td></td>
<td>Presence of eosinophils and necrosis</td>
</tr>
<tr>
<td></td>
<td>Rare Reed-Sternberg cells (difficult to find)</td>
</tr>
<tr>
<td>Mixed cellularity</td>
<td>Intermediate between lymphocytic predominance and depletion</td>
</tr>
<tr>
<td></td>
<td>Variety of histologic components (eosinophils, plasma cells, mature neutrophils, lymphocytes, histiocytes, and Reed Sternberg cells)</td>
</tr>
<tr>
<td></td>
<td>Necrosis and fibrosis may be present</td>
</tr>
<tr>
<td>Lymphocytic depletion</td>
<td>Decreased number of lymphocytes</td>
</tr>
<tr>
<td></td>
<td>Diffuse fibrosis with decreased number of all other cells on a disordered connective tissue</td>
</tr>
<tr>
<td></td>
<td>Reticular type with atypical histiocytes and increased number of Reed Sternberg cells</td>
</tr>
<tr>
<td></td>
<td>Necrosis common</td>
</tr>
</tbody>
</table>

Liebhauser, P. Hodgkin Lymphoma in Baggott, C et.al. Nursing Care of Children and Adolescents with Cancer, 2001, p.526
### Ann Arbor Staging Classification System

<table>
<thead>
<tr>
<th>Staging</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stage I</strong></td>
<td>Involvement of a single lymph node or lymphoid structure (spleen, thymus, Waldemeyer’s ring (nasopharynx, tonsil, base of tongue), appendix and Peyer’s patches; or extralymphatic site (1_E))</td>
</tr>
<tr>
<td><strong>Stage II</strong></td>
<td>Involvement of 2 or more lymph node regions or lymphoid structures on the same side of the diaphragm; Number of anatomic regions involved indicated by a subscript (II_x)</td>
</tr>
</tbody>
</table>
| **Stage III**    | Involvement of lymph node regions or lymphoid structures on both sides of the diaphragm; maybe subdivided into:  
|                  | - Stage III_1 – spleen or splenic, hilar, celiac, portal node involvement  
|                  | - Stage III_2 – para-aortic, iliac, mesenteric node involvement  
| **Stage IV**     | Diffuse or disseminated involvement of one or more extra-lymphatic sites with or without associated lymph node enlargement |

### Symptoms

- **A** Asymptomatic
- **B** Symptoms include:  
  - unexplained weight loss (≥10% of body weight) in 6 months before initial staging  
  - unexplained, persistent, recurrent fevers with temperatures > 38°C (100°F) during previous month  
  - recurring drenching night sweats during previous month

### Subscripts

- **X (x)** Bulky disease: ≥ 10 cm at maximal dimension

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**Lymphomation.org, Reigelsville, PA**  
[http://www.lymphomation.org/about-details.htm](http://www.lymphomation.org/about-details.htm)  
Staging Lymphoma
Extra-nodal extension: involvement of the extra-lymphatic tissue by limited direct extension from an adjacent nodal site or a single extra-nodal deposit consistent with extension from a regionally involved node.

Pathologic Staging Site (PS)

PS at a given site is indicated by a subscript:
- D = skin
- H = liver
- L = lung
- M = bone marrow
- O = bone or skeletal
- P = pleura


A – 6  Staging Laparotomy

Staging laparotomy includes splenectomy, wedge biopsies of the both hepatic lobes, and sampling of the splenic hilar, celiac and portal hepatic, mesenteric, iliac and para-aortic lymph nodes.

A – 7  Risks of Laparotomy Staging:

- Wound complications (infections, dehiscence)
- Bacterial infections by encapsulated organisms (Streptococcus pneumoniae and Haemophilus influenzae)
- Subphrenic abscess
- Pancreatitis
- Sepsis
- Retroperitoneal hematoma
- Surgery-related pulmonary complications – atelectasis, pneumonia

Late complications:
- Adhesions with intestinal obstruction
A – 8  **Lymph Nodes Enlargement/ Lymphadenopathy**

Nodules on the neck

Courtesy of Dr. Khattabi, IOP Fellow - Morocco

A – 9  **Mediastinal Mass**

Show the presence of a large anterior mediastinal mass causing some compression of the airway.

Courtesy of Guillermo Chantada, MD, Hospital JP Garrahan, Buenos Aires, Argentina
Added to http://www.cure4kids.org/ums/oncopedia/
Oncopedia Case #122 added 4/2/2008

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### Chemotherapy Regimen for Hodgkin’s Lymphoma

<table>
<thead>
<tr>
<th>Chemotherapy Regimen Combinations</th>
<th>Chemotherapy Drugs/Agents</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABVD</td>
<td>Adriamycin (doxorubicin), bleomycin, vinblastine, dacarbazine</td>
</tr>
<tr>
<td>AOPE</td>
<td>Adriamycin (doxorubicin), Oncovin (vincristine), prednisone, etoposide (VP-16)</td>
</tr>
<tr>
<td>ChlVPP</td>
<td>Chlorambucil, vinblastine, procarbazine, prednisolone</td>
</tr>
<tr>
<td>CHOP</td>
<td>cyclophosphamide, Adriamycin (doxorubicin), Oncovin (vincristine), prednisone</td>
</tr>
<tr>
<td>COMP</td>
<td>Cyclophosphamide, Oncovin (vincristine), methotrexate, prednisone</td>
</tr>
<tr>
<td>COPP</td>
<td>cyclophosphamide, Oncovin (vincristine), procarbazine, prednisone</td>
</tr>
<tr>
<td>CVPP</td>
<td>cyclophosphamide, Oncovin (vincristine), prednisone, procarbazine</td>
</tr>
<tr>
<td>DBVE – PC/DZR</td>
<td>Adriamycin (doxorubicin), bleomycin, Oncovin (vincristine), etoposide (VP-16), prednisone, cyclophosphamide/dextrazoxane</td>
</tr>
<tr>
<td>EVAP</td>
<td>etoposide (VP -16), vinblastine, cytosine-arabinoside (Ara-C), cisplatinum (Platinol)</td>
</tr>
<tr>
<td>MOPP</td>
<td>mechlorethamine (Mustargen), Oncovin (vincristine) procarbazine, prednisone</td>
</tr>
<tr>
<td>OEPA</td>
<td>Oncovin (vincristine), etoposide (VP -16), prednisone, Adriamycin (doxorubicin)</td>
</tr>
<tr>
<td>OPA</td>
<td>Oncovin (vincristine), prednisone, Adriamycin (doxorubicin)</td>
</tr>
<tr>
<td>OPPA</td>
<td>Oncovin (vincristine), Prednisone, Procarbazine, Adriamycin (doxorubicin)</td>
</tr>
<tr>
<td>VAMP</td>
<td>vinblastine, Adriamycin (doxorubicin), methotrexate, prednisone</td>
</tr>
<tr>
<td>VBVP</td>
<td>vinblastine, bleomycin, etoposide (VP -16), prednisone</td>
</tr>
<tr>
<td>VEEP</td>
<td>vinblastine, eoposide (VP -16), prednisolone</td>
</tr>
<tr>
<td>VEPA</td>
<td>vinblastine, eoposide (VP -16), prednisone, Adriamycin (doxorubicin)</td>
</tr>
</tbody>
</table>

Liebhauser, P. Hodgkin’s Lymphoma in Baggott, C et.al. Nursing Care of Children and Adolescents with Cancer, 2001, p.530
A – 11  Radiotherapy fields in lymphoma:

Mantle (supra-diaphragmatic field: irradiates submandibular, submental, cervical, supraclavicular, infraclavicular, axillary, mediastinal, and pulmonary hilar lymph nodes (neck, chest and/or lymph nodes under the arms)

Minimantle field irradiates bilateral supramediastinal disease involving axilla, supraclavicular, infraclavicular, or cervical lymph node chains

Hemi-minimantle irradiates unilateral supramediastinal disease involving axilla, supraclavicular, infraclavicular, or cervical lymph node chains

Sub-diaphragmatic (middle; para-aortic) field covers the heart and the spleen (para-aortic nodes)

Pelvic field covers the common iliac, external iliac and the inguino-femoral nodes (from the spleen to the groin).

Inverted Y includes both the sub-diaphragmatic and pelvic fields; radiation to the lymph nodes in front of the lower spine (para-aortic) and the groin. Each groin makes up an arm of the inverted Y.

Total nodal irradiation is given to the mantle field and the inverted Y field.
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Edited by: Marc Kusinitz, PhD, St. Jude Children’s Research Hospital
Cure4Kids Release Date: 1 September 2006

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