# Lymphoma Case Scenario 1

HISTORY: A 23-year-old healthy female presented with a month-long history of persistent headache of increasing severity. She noted episodic nausea and vomiting in association with her headaches that were exacerbated by exertion.

FAMILY HISTORY: Paternal aunt died at age 23 years of Hodgkin’s lymphoma.

3/10/16 PHYSICAL EXAM: Revealed nystagmus with bidirectional lateral gaze and left upper-extremity dysmetria.

3/13/16 IMAGING

MRI: Demonstrated a left-sided, extra-axial posterior fossa mass that was broadly based on the retrosigmoid dura, and enhanced homogeneously. Imaging characteristics were consistent with meningioma.

3/17/16 PROCEDURE: Posterior fossa craniectomy and tumor excision without complication.

3/18/16 SURGICAL PATHOLOGIC EVALUATION:

* Fibrous tumor composed of small lymphocytes and scattered plasma cells consistent with nodular sclerosis type Hodgkin’s lymphoma.
* Immunoperoxidase staining: Reed-Sternberg cells present and positive for CD15 and CD30.
* In situ hybridization for Epstein-Barr virus–associated RNA was negative.

3/21/16 POST SURGERY IMAGING

CT: Prevascular, pretracheal and supraclavicular lymphadenopathy

MRI: T10 vertebral body metastasis with adjacent epidural enhancement

3/23/16 POST SURGERY FINDINGS

Lumbar Puncture: normal CSF protein and negative CSF cytology

Bone Marrow biopsy: negative

HIV: negative

Final Diagnosis: Hodgkin’s Lymphoma

TREATMENT:

The patient was treated with six cycles of chemotherapy with doxorubicin, bleomycin, vinblastine, dacarbazine (ABVD), with four treatments of intrathecal thiotepa, followed by 36 Gy radiotherapy to the posterior fossa, and 30.6 Gy to the mediastinum, including T10. The patient entered complete remission after three cycles of ABVD and remains in ongoing complete remission.

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| * **What is the primary site?**
* **What is the histology?**
 | * **What is the grade/differentiation?**
 |
| **Stage/ Prognostic Factors** |
| Summary Stage |  | Tumor Size Summary |  |
| TNM Clin T |  | TNM Path T |  |
| TNM Clin N |  | TNM Path N |  |
| TNM Clin M |  | TNM Path M |  |
| TNM Clin Stage |  | TNM Path Stage |  |
| TNM Clin Descriptor |  | TNM Path Descriptor |  |
| TNM Clin Staged By |  | TNM Path Staged By |  |
| CS SSF 2 |  |  |  |
|  |  | Regional Nodes Positive |  |
|  |  | Regional Nodes Examined |  |
|  |  | Mets at Dx - Bone |  |
|  |  | Mets at Dx - Brain |  |
|  |  | Mets at Dx - Liver |  |
|  |  | Mets at Dx - Lung |  |
|  |  | Mets at Dx - Other |  |
|  |  | Mets at Dx – Distant LN |  |
|  |  |  |  |
| **Treatment** |
| Diagnostic Staging Procedure |  |  |  |
| **Surgery Codes** |  | **Radiation Codes** |  |
| Surgical Procedure of Primary Site |  | Radiation Treatment Volume |  |
| Scope of Regional Lymph Node Surgery |  | Regional Treatment Modality |  |
| Surgical Procedure/ Other Site |  | Regional Dose |  |
| **Systemic Therapy Codes** |  | Boost Treatment Modality |  |
| Chemotherapy |  | Boost Dose |  |
| Hormone Therapy |  | Number of Treatments to Volume |  |
| Immunotherapy |  | Reason No Radiation |  |
| Hematologic Transplant/Endocrine Procedure |  | Radiation/Surgery Sequence |  |
| Systemic/Surgery Sequence |  |  |  |

# Leukemia Case Scenario 2

3/11/16 HISTORY: A 21-year-old female presented to the outpatient clinic with lower abdominal pain. Seven days before presentation, she had a ruptured corpus luteal cyst, which was detected on abdominal computed tomography (CT) at another clinic. Her initial platelet count was estimated to be 3,777×109/L at our clinic. Because thrombocytosis appeared to be secondary to bleeding, the patient's blood cell counts were only monitored during her clinical course. However, we decided to perform further evaluation because of thrombocytosis persisted for 2 weeks with no decrease in the platelet count. Her medical history was unremarkable, and she had no family history of hematologic disease or genetic disorders. Her vital signs were normal at admission. Except for mild lower abdominal tenderness, the patient had no other positive findings on physical examination.

3/13/16 LABWORK

* (CBC) hemoglobin level of 10.1 g/dL, hematocrit level of 30.7%,
* (WBC) count of 10×109/µL (differential count: neutrophils 63%, lymphocytes 33%, eosinophils 1%, basophils 3%, and monocytes 0%) and platelet count of 3,294×109/L.

Serum biochemistry panel:

* Total protein, 7.2 g/dL;
* Albumin, 3.9 g/dL;
* Total bilirubin, 1.2 mg/dL;
* Aspartate aminotransferase, 11 IU/L;
* Alanine aminotransferase, 13 IU/L;
* Blood urea nitrogen, 6 mg/dL;
* Creatinine, 0.6 mg/dL;
* Lactic dehydrogenase, 410 IU/L;
* C-reactive protein, 2.0 mg/dL.

Peripheral blood smear showed thrombocytosis.

* Serum iron level was 73 µg/dL,
* total iron binding capacity was 267 µg/dL,
* ferritin level was 206.5 ng/mL.
* LAP score of 127 points, (within normal range)

3/16/16 CT of Abdomen and Pelvis:

Revealed a small amount of hemoperitoneum resulting from the previous ruptured ovarian cyst.

3/25/16 PROCEDURE

Bone Marrow Aspiration and Biopsy:

Revealed a high number of megakaryocytes, but no cells undergoing malignant transformation cytogenetic abnormality was detected with the karyotype 46,XX,t(9;22)(q34;q11.2) on bone marrow. A *bcr/abl* rearrangement in the bone marrow using reverse transcriptase PCR was observed, which also showed amplified products from the b3a2 mRNA deletion in the major *bcr* gene.

3/25/16 POST SURGICAL TESTING and DIAGNOSIS

* Results were negative for the *JAK2* V617F mutation.
* Because the patient had isolated thrombocytosis (3,294×109/L), she was tentatively diagnosed with Essential Thrombocythemia before the results of the cytogenetic and molecular studies were available, even if results for the *JAK2* V617F mutation were unknown.

4/05/16 TREATMENT

Hydroxyurea was administered to the patient at a dose of 2,000 mg/day for 14 days to lower her platelet count. A follow-up CBC showed persistent thrombocytosis, platelet counts of 2,206×109/L, and leukocytopenia (1.1×109/L). Hydroxyurea was discontinued and identified the Philadelphia chromosome and *bcr/abl* rearrangement, but no *JAK2* V617F mutation. This led to the final diagnosis of chronic-phase CML, for which the patient received imatinib. In the 6 days following the treatment with imatinib, the patient's platelet count normalized to 438×109/L. Patient is currently followed up to confirm complete molecular response against  *bcr/abl* rearrangement. In the 3 months after treatment with imatinib, a major molecular response (3-log reduction of transcript levels) was observed.

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