Letters to the Editor are welcomed. They may report new clinical or laboratory observations and new developments in medical care or may contain comments on recent contents of the Journal. They will be published, if found suitable, as space permits. Like other material submitted for publication, letters must be typewritten, double-spaced, and must not exceed two typewritten pages in length. No more than five references and one figure or table may be used. See “Information for Authors” for format of references, tables, and figures. Editing, possible abridgment, and acceptance remain the prerogative of the Editors.

Ilioinguinal-Ilioinguinal Nerve Block for Hernia Repair

To the Editor: A recent article and editorial describing ilioinguinal-iliohypogastric nerve block (IHNB) for inguinal hernia repair, while thought provoking, is also troubling. The authors have performed a prospective randomized clinical trial (PRCT) and then presented the data as a retrospective study. Patients must grant informed consent to be randomized. A human subject committee (often called an IRB) should review the trial before the beginning of the study.

There are several other study designs that have a sounder ethical basis than the approach taken by Yilmazlar et al. First, one can describe the alternative treatments to the patient. The investigator states that on the basis of current medical knowledge, we do not know which treatment is better. Therefore, the patient should select the therapy which sounds best. The patient is informed that in the future, the data will be reviewed to try to determine which therapy is really better. Naja et al. have published a study of anesthesia techniques for hernia repair where treatment allocation was based on patient choice.

A second ethical approach is to seek experiments of nature. For example, physician A feels that in his experience, spinal anesthesia for hernia is followed by an unacceptable rate of postdural puncture headache (PDPH) and urinary retention. The only regional technique that physician A offers to his patients is IHNB. Physician B feels that IHNB has a 20 to 30% failure rate. Physician B only offers his patients spinal anesthesia. Both physicians have a sound basis in the literature and their personal experience to justify their positions. The regional block that the patient receives will be determined by whether they are assigned to Dr. A or Dr. B, often a fairly random process. The IRB is asked to approve a retrospective review of the two therapies.

Finally, some very sophisticated designs have been proposed to reconcile randomization with patient choice and autonomy. These designs may be impractical for most clinical researchers who lack formal biostatistical support.

Despite the ethical problems with the article, Dr. Wang’s accompanying editorial is encouraging. Ultrasound-guided IHNB may prove to be a very good anesthetic choice for inguinal hernia repair. Nerve stimulator-guided paravertebral block (PVB) is another attractive option. PVB provides the ideal triad: segmental anesthesia, good muscle relaxation, and prolonged analgesia. PVB is not plagued by the common spinal anesthesia sequelae of PDPH and urinary retention.

The anesthesia community awaits a prospective randomized clinical trial comparing ultrasound-guided IHNB versus PVB. Which of these exciting new techniques will prove most efficacious and cost-effective for open inguinal hernia repair?

Richard K. Baumgarten, MD

References

Response to Dr. Baumgarten’s Comments

To the Editor: In response to Dr. Richard K. Baumgarten’s letter, we would like to emphasize the following:

First, our study is a retrospective one on 126 patients who had undergone inguinal hernia repair.

Second, in our hospital, all patients sign a consent form which gives us permission to use their medical records in future scientific studies.

Third, several anesthetic techniques can be used for inguinal herniorrhaphy. The goal of our study was to compare spinal anesthesia (SA) with ilioinguinal-iliohypogastric nerve block (IHNB).

Fourth, we reviewed the charts of patients who had undergone surgery between 1999 and 2002. That was before
the publication of articles by Willschke et al\(^1\) and Eichenberger et al\(^2\) on ultrasound-guided ilioinguinal-iliohypogastric nerve block. Hence, we were not able to evaluate their technique in our study. I agree with the authors who point out that paravertebral block (PVB) is an ideal alternative technique that can be used in inguinal herniorrhaphy.\(^3\)

We appreciate Dr. Baumgarten’s interest in our article.

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Aspartame-induced Thrombocytopenia

To the Editor: The cause of thrombocytopenia is often not found. Owing to my interest in reactions to aspartame products,\(^1\) I have encountered four cases of thrombocytopenia which may be attributed to products containing aspartame, especially based on its recurrence on two or more occasions after rechallenge, and the absence of any other definable factor.

Patient 1
A 10-year-old girl developed a decline of her platelet count to 1,000 cu/mm, coupled with striking enlargement of the liver and spleen, and a marked increase in histiocytes in the bone marrow. There was dramatic clinical and hematological normalization when additives were eliminated from her diet, enabling the cessation of prednisone. Similar recurrences occurred twice after ingesting aspartame—first in a cereal, and later with aspartame gum—with remissions when abstaining from aspartame products.

Patient 2
An 11-year-old girl was hospitalized for thrombocytopenia. She had been chewing various sugar-free gums. There was a remission after avoiding aspartame. Severe thrombocytopenia recurred after visiting a relative who indulged her with aspartame sweets.

Patient 3
A 61-year-old man developed a decline of his platelets to 54,000 cu/mm after drinking diet colas for two years, coupled with headaches, dizziness, two convulsions, and a nonspecific rash. Extensive studies failed to uncover an underlying problem. He improved after aspartame abstinence. One retest trial resulted in an immediate exacerbation.

Patient 4
A registered nurse began using diet colas after her second pregnancy. Her platelets declined to under 30,000 cu/mm, coupled with headaches and hypertension. The platelet count and blood pressure normalized after avoiding aspartame products.

A reaction to aspartame products should be considered in patients with thrombocytopenia in whom no underlying cause can be determined.

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Vitamin D: Deficiency or no Deficiency?

To the Editor: Vitamin D deficiency is a common disorder that can have adverse effects on bone metabolism. There is no universal agreement on the levels of serum 25-hydroxyvitamin D [25(OH)D] that define vitamin D deficiency. Several cut-off points were suggested, including levels less than 12.5 ng/mL (30 nmol/L), 20 ng/mL (50 nmol/L) and 30 ng/mL (75 nmol/L).\(^1,2\) Levels higher than 30 ng/mL (75 nmol/L) have been recently recommended to achieve better bone and dental health and lower the risk of fractures.\(^3\) Vitamin D deficiency is generally associated with increased levels of parathyroid hormone (PTH), and the level of 25(OH)D at which PTH concentration starts to rise has been postulated to define vitamin D deficiency.

This study was undertaken to determine the level of 25(OH)D that defines vitamin D deficiency in a population with year-round sun exposure. The study population consisted of ambulatory individuals residing in the eastern province of Saudi Arabia. Enrolled patients were aged 15 years or more, attending primary care clinics for conditions not related to metabolic bone disease, not taking medications known to affect bone metabolism and who were suspected to have vitamin D deficiency based on a quantified history of sun exposure, dietary intake and musculoskeletal symptoms. Levels of serum 25(OH)D, calcium, and PTH were measured. To determine the cut-off point to define vitamin D deficiency, a multivariate adaptive regression analysis of PTH on 25(OH)D level was performed. A total of 71 patients were evaluated (87% females, 13% males), with a mean age of 41 years. Mean 25(OH)D concentration was 7.75 ng/mL (18.6 nmol/L), 95% confidence interval, 15.9 to 21.3. Serum 25(OH)D was less than 30 ng/mL (75 nmol/L) in 100% and less than 20 ng/mL (50 nmol/L) in 97.2% of patients. Gender and age were not significant factors in determining vitamin D status. Normal PTH concentrations were
found in 35.7% of patients with severe deficiency [serum 25(OH)\textsubscript{D}, below a level of detection of 5 ng/mL (12 nmol/L)]. PTH levels correlated negatively with 25(OH)\textsubscript{D} \( r = -0.23; P = 0.04 \), but we could not identify a threshold level for serum 25(OH)\textsubscript{D} at which PTH would have started to rise.

Even though vitamin D deficiency is a very common condition worldwide, identifying a criterion for its definition remains unresolved. Some suggested certain levels of 25(OH)\textsubscript{D} based on data correlating with PTH concentrations, while others proposed a population-based estimate, considering factors that can affect the definition such as geographical location, age, race and time of the year.\textsuperscript{4}

Our study revealed that in a population with abundant sun exposure, PTH concentrations were not helpful to define the cutoff level that defines vitamin D deficiency; this confirms findings from a study of vitamin D status in a Finnish population.\textsuperscript{5} Larger scale population-based studies are suggested to delineate individual characteristics that define vitamin D status.

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References


Rhabdomyolysis Associated with Pulmonary Tuberculosis

To the Editor: Rhabdomyolysis, a syndrome of muscle breakdown, is caused by a variety of infectious and noninfectious etiologies. To my knowledge, this is the first reported case of rhabdomyolysis caused by active pulmonary tuberculosis.

A 60-year-old African-American gentleman presented to the emergency room with productive cough associated with fever, weight loss and occasional night sweats.

The patient denied muscle weakness or tenderness. He also denied a recent history of trauma or falls. The patient is known to be diabetic. He was taking only multivitamins and had no history of illicit drug abuse. The physical examination revealed diminished breath sounds over the right lower and upper lobe segments on auscultation and dullness over the right lung on percussion. The rest of the examination was unremarkable.

Laboratory studies revealed a serum glucose level of 280 mg/dL, a blood urea nitrogen level of 8 mg/dL, a creatinine level of 0.7 mg/dL, a sodium level of 130 mmol/L, a potassium level of 2.6 mg/dL, a magnesium level of 1.7 mg/dL, and a calcium level of 1.22 mg/dL. The creatine kinase (CK) level was 13,918 IU/L at admission and increased to above 16,000 IU/L the following day. The patient was treated with IV fluid and antituberculous drugs. The patient’s creatine kinase level started declining after one week of therapy and the patient was later discharged home on an oral antituberculous drug regimen.

Rhabdomyolysis is a life threatening disease resulting from the disintegration of skeletal muscle cells. The causes are generally secondary to trauma, exertion, drugs, toxins, electrolyte abnormalities, endocrine disorders, inflammatory myopathies and infections. Active pulmonary tuberculosis has not been reported yet as a cause. Review of the literature revealed only one case by Cases et al reporting rhabdomyolysis associated with pleural tuberculosis.\textsuperscript{1} This case did not involve pulmonary tuberculosis. Other reported cases of pulmonary infections associated with rhabdomyolysis have generally been related to viral or bacterial etiologies such as influenza, chlamydia, Legionella, mycoplasma, streptococcus and staphylococcus. The pathophysiologic mechanism in these infections leading to rhabdomyolysis is presumed to be muscle injury from direct toxin effect, hemodynamic alterations, immune-mediated process, or direct invasion.\textsuperscript{2–4}

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References


Phytophotodermatitis Due to Puncture from Lime Tree Thorn

To the Editor: Phytophotodermatitis is a cutaneous phototoxic inflammatory
erupted due to exposure to light-sensitizing botanical substances and long-wave ultraviolet radiation that usually begins approximately 24 hours after exposure and peaks at 48 to 72 hours. The primary skin lesions of phytophotodermatitis may range from delayed erythema (24–48 h) to a frankly nodular eruption. The photosensitizing compounds in the plants are furanocoumarins. The phototoxic reaction is entirely independent of the immune system; thus, prior sensitization or an intact immune system is not required. The clinical history is essential in establishing the diagnosis of phytophotodermatitis.

While picking limes from a Tahitian lime tree located in the Paradise Park area of Kea‘au, Hawaii, a 69-year-old Caucasian man developed phytophotodermatitis after puncture of the skin in the right scapular area by a thorn. The patient’s back was exposed to the sun the following day. Lesions first developed after a puncture wound by the same evening, and complete resolution of the lesions was noted with 2% diphenhydramine spray and triple antibiotic ointment (bacitracin, neomycin, and polymyxin B), beginning resolution of the lesions was noted by the same evening, and complete resolution occurred over a week’s time. There was no pruritus or residual cutaneous hyperpigmentation. The past history was negative for asthma, hay fever, or allergic reactions to medications.

The present case had some unusual features. The patient had an asymptomatic lime thorn scratch over the right forearm, whereas the central scapular lesion developed after a puncture wound from the lime thorn, and the satellite scapular lesions apparently resulted from spread due to scratching by the patient. This suggests that more of the irritating substance was introduced through the puncture.

The onset of dermatitis is variable, however, and may begin within a few hours to days after exposure. Recent evidence supports it being a type of irritant contact dermatitis. The sap of the tree and scratches by the thorns may cause rashes in sensitive individuals. A variety of reactions have been noted as a result of exposure to citrus peel. Many plants, including limes, contain furanocoumarins (psoralen) that cause phytophotodermatitis by forming phototoxic compounds on exposure to ultraviolet A radiation. After resolution of the phototoxic reaction, postinflammatory hyperpigmentation may occur.

Phototoxic reactions to exposure to lime can be polymorphous in nature. Due to the bizarre configurations of lesions induced by phytophotodermatitis, it can be confused with many other diseases, especially in children. A detailed clinical history is crucial in establishing the correct diagnosis of phytophotodermatitis. The treatment of acute phytophotodermatitis is primarily symptomatic with the lesions responding well to the application of topical corticosteroids and cold compresses. Mild cases may not require treatment.

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References

Letters to the Editor
Retroperitoneal Fibrosis as a Long-term Complication of Radiation Treatment for Prostate Cancer

To the Editor: A 70-year-old gentleman presented with a six week history of abdominal pain associated with generalized weakness and anorexia. The patient reported insidious onset, 5 to 6/10 aching, and mid-abdominal pain which was aggravated in the supine position with no relation to food. Past medical history included a 30-pack year history of smoking and heavy alcohol consumption. Careful questioning revealed that six years prior, the patient had completed eight weeks of radiotherapy for carcinoma of the prostate. Physical examination revealed poorly localized lower quadrant abdominal tenderness, but there were no masses or organomegaly.

Laboratory workup was significant for blood urea nitrogen (BUN) of 35 and serum creatinine of 2.7. Ultrasound of the abdomen was followed by computed tomography (CT) scan without contrast of the abdomen, which revealed bilateral hydronephrosis and an ill-defined soft tissue mass surrounding the distal abdominal aorta mimicking a leaking aortic aneurysm (Fig. top panel). Cystoscopy showed evidence of diffuse fibrosis involving the posterior urethra and bladder, suggestive of secondary changes from radiation treatment. Retrograde pyelogram demonstrated bilateral hydrourerteronephrosis and bilateral extrinsic ureteral compression with medial deviation. With difficulty, bilateral ureteric stents were placed over a guidewire through extremely small ureteral orifices.
The patients BUN and serum creatinine returned to baseline after stent placement. Subsequently, biopsy of the retroperitoneal mass was obtained through exploratory laparotomy. Pathology results from the biopsy specimens revealed chronic inflammatory cell infiltrates admixed with plasma cells, histiocytes and eosinophils, confirming the diagnosis of retroperitoneal fibrosis (RPF) (Fig. bottom panel).

RPF is a rare entity with incidence varying from 1:200,000 to 1:500,000. Although two thirds of these cases are idiopathic, it has been reported as a sequel of pelvic irradiation delivered for the treatment for testicular seminoma, colon carcinoma, pancreatic carcinoma, cervical carcinoma and uterine fibroma.1–3

Prostate cancer is the most common malignancy of men in the United States. External beam radiotherapy is the curative treatment for localized carcinoma of the prostate and has an important role in the adjuvant therapy for advanced cases.4 In the patient reported here, prostate carcinoma was classified as localized adenocarcinoma of the prostate on histopathology and was clinical stage T1c with a Gleason score 2+3. Baseline prostate specific antigen (PSA) level was 5.7 ng/mL. Patient received 3 D conformal external beam pelvic radiotherapy. Total cumulative dose of 75.6 Gy radiations was delivered to the prostate in forty-two fractions over a span of eight weeks. Definitive remission was documented (PSA level less than 0.1) in subsequent follow-up visits. Patient seemed to be fine until he presented with acute renal failure and bilateral hydronephrosis and was found to have retroperitoneal fibrosis. To the best of our knowledge, only one case of localized fibrosis has been described in a patient receiving radiotherapy for prostate carcinoma.5 In that case, the patient underwent transurethral resection of the prostate (TURP) for stage C adenocarcinoma of the prostate and postoperatively received 6000 rads of cobalt radiotherapy. One year later, he was found to have localized fibrosis involving urethras. In our case, the patient had rather extensive fibrosis involving multiple abdominal and pelvic viscera. Moreover, recognition of RPF as a sequel of radiotherapy for prostate carcinoma after such a long interval (six years in our case) is the first one of its kind.

RPF is characterized by dense fibrous plaque formation in the retroperitoneal structures. The hallmark of RPF is fibrosis and can involve the lower third of the ureters leading to obstructive uropathy as seen in our patient. Although poorly localized back pain is the most common presenting complaint, RPF may present with many other nonspecific symptoms, including anorexia, weight loss, anemia, mild fever or malaise. On account of these, the diagnosis is usually late.1 As evident in our case, radiological evidence of RPF on IV urography includes a classic triad of upper ureteral hydronephrosis, medial deviation of the ureters and extrinsic ureteral compression.1 CT and MRI are the modalities of choice for documenting the extent of disease. Medical management of RPF includes corticosteroid as first line and immunosuppressive drugs as second line drugs. Surgical options include relieving ureteral obstruction through surgical laparotomy or laparoscopic means.1 The
long term prognosis is usually favorable, but severe complications such as chronic renal failure, requiring renal replacement therapy, may arise.1

Our case documents that retroperitoneal fibrosis can arise as a sequel of radiation treatment for prostate cancer; therefore, we recommend prolonged follow-up of several years. We urge the target audience to identify this unusual but manageable complication of prostate cancer radiotherapy.

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References

Electrical Storm and Implanted Defibrillators

To the Editor: As more patients obtain automatic implantable cardioverter defibrillators (AICDs), EMS personnel and emergency medical centers will be required to treat the unique situations that such patients present to emergency center personnel. One unique presentation is electrical storm (multiple temporally related episodes of ventricular tachycardia or ventricular fibrillation)1,2 with an appropriately functioning AICD.3

A 65-year-old female was brought to the emergency center by EMS. She was complaining of repeated discharges of her defibrillator, and she complained her chest was hurting because of the discharges. When she arrived, the patient had no IV established, as the EMS personnel were getting shocked while attempting to start an IV. The patient remained conscious throughout her EMS transport and emergency department stay. She had counted many (30) discharges by the time of arrival. She was immediately connected to a cardiac monitor and vital signs were noted: irregular rapid pulse of 160 to 200. Emergency department nurses attempted to establish IV lines but kept getting “shocked” when the patient’s defibrillator discharged. The nurses were very concerned that they would be unable to get IV access. However, cardiac monitor observations showed that it took about a 4 second continuous run of ventricular tachycardia/ventricular fibrillation (VT/VF) viewed on the cardiac monitor to trigger the patient’s AICD to discharge in about 2 seconds. One to three second runs of VT/VF that self-terminated would not trigger a discharge. By careful observation of the cardiac monitor, nurses could obtain adequate warning as to when to stop physical contact with the patient and avoid a “shock.” This practice worked well as the nurses were no longer “shocked” and in a few minutes, IV lines were established and blood was drawn. This was not a trivial success because without vascular access, and with staff too nervous and concerned to establish access, the ability to adequately provide care could have been compromised.

Conventional lidocaine therapy was followed by a 300 mg amiodarone bolus and drip (1 mg/min). This treatment markedly reduced the VT/VF runs and allowed the patient to have relief from frequent AICD discharges. This allowed time to get a more complete history from the patient, discover and treat her underlying causes for her electrical storm (low potassium and medical non-compliance).

With increasing numbers of patients obtaining AICDs, EMS and emergency center personnel need to have a working knowledge of how to handle patients that present with symptoms in which AICDs play a role. Being shocked from a patient’s AICD discharge is at best startling and at worst could cause fear in some caregivers that could compromise patient care. Discharges felt while touching a patient’s skin are not strong enough to cause fibrillation in caregivers.4 However, even with this knowledge, it is difficult to do procedures with confidence if a caregiver is frequently and randomly shocked while attempting a procedure. In this case, we describe an easy method to avoid getting shocked, which our caregivers found to be very valuable. Simply observing the cardiac monitor for VT/VF runs that last about 4 seconds will allow a caregiver about 2 seconds of warning before the AICD discharges. No caregiver was shocked once we used this method; confidence was reestablished and IV lines were started.

Although the use of lidocaine was done in this case with some effectiveness, switching to amioderone was the key for suppressing the patient’s VT/VF. Electrical storm has been successfully treated by various medications.5 Currently, amioderone seems to be the most effective.5

Electrical storm in patients with an appropriately discharging AICD can be a medical emergency that challenges caregivers in unique ways. Both the patient and caregivers can obtain successful resolution of the problems of repeated AICD discharges if the caregivers proceed with knowledge of the discharge cycle of the AICD. Consequently, caregivers can avoid getting shocked. If the caregivers are inadvertently shocked, they know that the shock is unlikely to cause them harm. Patients then can have the underlying causes for the electrical storm deter-
Acute Pancreatitis Induced by Adult Precursor B-cell Acute Lymphoblastic Leukemia Associated with Complex Cytogenetics

To the Editor: We report the case of a patient with precursor B-cell acute lymphoid leukemia and poor cytogenetics who presented with acute pancreatitis related to leukemia.

The patient, a 42-year-old gentleman, presented with abdominal pain. Physical examination revealed tenderness in the epigastrium and a 6-cm mass in the left chest wall. Laboratory data revealed a white blood cell count of 7.0 k/uL, with 39% blasts, hemoglobin of 10.5 g/dL, and a platelet count of 84 k/uL. Calcium was 8.5 mg/dL, amylase 274 IU/L, and lipase was 356 IU/L. Computed tomography (CT) of the thorax and abdomen showed fat stranding and haziness around the pancreas, suggestive of acute pancreatitis (AP) along with a soft tissue mass in the left anterior chest. Bone marrow aspiration and biopsy showed hypercellular marrow, heavily infiltrated by lymphoblasts, which were predominantly precursor B lymphoblasts. Findings were compatible with precursor B lymphoblastic leukemia/lymphoma with aberrant CD33 expression.

Bone marrow cytogenetics revealed a complex pattern (47, XY, t(1;7), t(2;15), t(4;15), +8(2)/46, XY). FISH was negative for t(9;22) BCR-ABL. CT-guided biopsy of the soft tissue mass in the chest wall showed precursor B lymphoblastic leukemia/lymphoma. Despite conservative management of AP, his pancreatic enzymes continued to be elevated. After 10 days of admission, he was started on systemic chemotherapy (hyper-CVAD). His pancreatic enzymes started going down within a day of initiation of the chemotherapy, and normalized within 2 days of starting treatment.

Acute lymphoblastic leukemia (ALL) is a neoplastic disorder that is rapidly fatal if untreated. Approximately half of all adult patients with ALL have lymphadenopathy, hepatomegaly or splenomegaly. Other sites of extramedullary involvement include the testis, retina, and skin.1,2 AP in association with ALL has been reported previously, but the connection was mainly related to chemotherapy agents, namely L-asparaginase.3 There have been a few case reports of AP associated with hypercalcemia due to acute T-cell leukemia (ATLL),4 and one case report documented invasion of the pancreas by ALL lymphoblasts in a case of ATLL.5

Our patient, who was diagnosed with precursor B-cell ALL, presented with AP and showed improvement with chemotherapy. The patient had normal serum calcium at presentation, and his pancreatic enzymes continued to be elevated for 10 days despite conservative management. He improved soon after starting the chemotherapy however, suggesting that the AP was associated with ALL. The fact that the patient had a subcutaneous mass in his chest wall, biopsy proven to be ALL, suggested that his leukemia was maintaining a tendency to involve extramedullary organs. Such presentation generally indicates a poor prognosis. The presence of complex cytogenetics is also a poor prognostic factor, which may have contributed to this presentation.

We hypothesize that the main cause of his AP was pancreatic tissue invasion by acute precursor B-cell leukemia cells. Although autopsy studies have shown pancreatic involvement by ALL, it has been found only with T-cell ALL. To the best of our knowledge, our report is the first to show clinically proven AP related mostly to involvement of the pancreas by precursor B-cell leukemia.

In conclusion, AP associated with ALL is induced not only by chemotherapy or hypercalcemia, but can also be directly related to invasion of the pancreas by lymphoblasts.

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References

In a large clinical series of 2185 patients of HL, CNS involvement was extremely rare. NHL, CNS involvement by Hodgkin lymphoma (HL) is exceedingly rare. In contrast to B-cell origin account for the majority of these neoplasms. In 1985, a large clinical series of 2185 patients of HL, CNS involvement by Hodgkin lymphoma (HL) is exceedingly rare. In contrast to NHL, CNS involvement by Hodgkin lymphoma (HL) is exceedingly rare. In a large clinical series of 2185 patients of HL, CNS involvement was seen in only 0.2% cases. All of these cases with CNS involvement were seen at relapse, along with disseminated systemic disease. Presentation of HL with a primary intracranial lesion is extremely rare, with only sporadic cases reported in the literature. We report the following case of biopsy proven Classical Nodular Sclerosis Hodgkin lymphoma presenting in a young male with simultaneous intracranial and spinal involvement, along with nodal disease. A 38-year-old male presented with vertigo to our ENT outpatient clinic. MRI of the brain showed a contrast enhancing lesion located at the roof of the fourth ventricle extending into the pons with significant edema. He was then lost to follow-up and again presented a month later with diplopia. Repeat MRI showed the same pons lesion with increased surrounding edema and mass effect along with a prominent enhancing lesion along the left aspect of the cauda equina. CT scan of chest abdomen and pelvis showed enlarged retroperitoneal nodal masses. Given his progressing neurologic symptoms, he was started immediately on radiotherapy (XRT) and parental steroids, which resulted in significant symptomatic improvement. At this point, with a high clinical suspicion for primary CNS NHL, an Ommaya reservoir was placed and the patient was given intrathecal and systemic methotrexate. Cytology from the CSF was negative for malignant cells on multiple occasions. After 2 nondiagnostic CT guided biopsies, a laparoscopic biopsy of the retroperitoneal lymph nodes demonstrated, to our surprise, classical Nodular Sclerosis HL. There was extensive architectural effacement by a diffuse and vaguely nodular proliferation with birefringent collagen bands around some nodules, a sclerotic nodal capsule and some diffuse sclerosis. There were predominantly small lymphocytes with a moderate number of scattered large cells, many of which had prominent nucleoli, some of which were binucleate and some of which were in lacunar spaces. Immunohistochemical stains showed that the large atypical cells were positive for CD15 and CD30, mostly negative for CD20 and Oct 2, and negative for CD79a, CD45, B8b.1, MUM-1, PAXS (weak) and T-cell associated antigens. No clonal T-cell receptor (TCR) gamma chain gene rearrangement was detected by PCR. A definitive diagnosis of classical nodular sclerosis HL was therefore rendered. The lesions in pons and cauda equina were not biopsied due to the high risks involved in biopsying the brainstem and the visible clinical improvement on current therapy. The patient was discharged for XRT followed by chemotherapy on the Stanford V protocol as an outpatient. He showed steady improvement through the course of radiation. At the end of treatment, his cranial nerve deficits had resolved completely, and repeat imaging showed complete resolution of the CNS disease and retroperitoneal lymphadenopathy. At one year follow-up, he remained in remission.

Presentation of Hodgkin lymphoma with CNS involvement is extremely rare, with only 11 such prior case reports found in a literature search. The most common form of involvement was a dural-based parenchymal mass. Posterior fossa/brainstem involvement was seen in only 36% of the cases, all of which had a cerebellar lesion. None of the cases had pontine involvement. The most common histologic type of HL among these cases was nodular sclerosis. Treatment modalities used were neurosurgery, intrathecal chemotherapy, systemic chemotherapy and radiotherapy. Chemotherapy protocols used included ABVD, MOPP and CHOP. Regardless of the treatment modalities used, complete remission was achieved in all cases, along with resolution of neurologic manifestations. No uniform risk factors for neurologic involvement by Hodgkin lymphoma have been identified so far.

### References

References


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Grief can take care of itself, but to get the full value of a joy you must have somebody to divide it with.

—Mark Twain