BREAKOUT ROOM NUMBER EIGHTEEN

CV 171 - 180
LUNG IN THE HEART: A FATAL METASTASIS

Introduction
Metastatic disease spreading to the heart is a rare finding that complicates the clinical course of patients with malignant neoplasms. Malignant melanoma, leukemia, and lymphoma are the malignancies with high tendency to spread to the heart. The relative numbers are greater with breast and lung cancers because of their higher incidence (1).

Case presentation
A 64-year-old female with a 72 pack-year smoking history presented with cough and left chest tenderness. Physical examination was unremarkable. CT scan showed large left upper lobe mass extending from the chest wall to the mid mediastinal area and involving the area of the left hilum. There was encasement of the left pulmonary artery branches, as well as portion of the aortic arch. Biopsy following bronchoscopy confirmed non-small cell lung cancer. PET scan revealed an intense focus at the body of pancreas. Biopsy showed poorly differentiated adenocarcinoma consistent with lung primary. She was started on radiation therapy but a month later she developed dyspnea on exertion. An echocardiogram was done to further evaluate dyspnea which showed a mobile echo dense mass in the left atrium which was likely coming from the left pulmonary vein. The right atrium looked normal. The mass was measured at 0.73 cm X 0.94 cm. Given that patient had active lung malignancy, the characteristic of the mass was consistent with metastatic lung carcinoma in the left atrium attached to the pulmonary vein. Our patient was later started on chemotherapy; however, she passed away in hospice care due to progression of cancer.

Discussion
Intracardiac metastases usually originate from lung cancer (30-40%) with incidences up to 18% in the literature and 10% clinically. They are discovered mostly incidentally but sometimes in symptomatic patients as part of a routine workup of tachycardia and arrhythmia (2). Tumor cells reach the heart through one of four pathways: retrograde lymphatic extension, hematogenous spread, direct contiguous extension or transvenous extension (3). Most patients with cardiac metastases have disseminated disease, the therapy generally consists of treatment for the primary tumor or palliative care. A standard treatment modality for cardiac metastases has not yet been established (4).
A DOUBLE-EDGED SWORD: NIVOLUMAB-INDUCED PNEUMONITIS

Immune checkpoint inhibitors have been shown to be a promising and novel therapy for many malignancies. Nivolumab, a Programmed Death-Ligand 1 (PD-L1) monoclonal antibody, is now the mainstay of the National Comprehensive Cancer Network (NCCN) recommended adjuvant systemic therapies for advanced melanoma. Unfortunately, these immunotherapies may induce immune responses against non-tumor targets, resulting in immunotherapy-related adverse event (IrAE). A 61-year-old gentleman with history of melanoma of the left shoulder (stage IIIa, negative BRAF V600 mutation) status post excision and three courses of Nivolumab presented with progressive dry cough and dyspnea on exertion (DOE) for one month, worsened over one week. Significant findings include preserved saturations on room air, a prolonged expiratory phase, generalized coarse lung sounds, with fine “velcro-like” crackles in the bibasilar lung fields. Labs were remarkable for leukocytosis with left shift and an elevated liver enzymes. Infectious workup was negative including viral, bacterial, and fungal etiologies. Chest X-ray (CXR) demonstrated diffuse reticular nodular opacities in both lung fields, with Computed Tomography (CT) revealing extensive reticulonodular infiltrates and ground-glass opacities (GGOs), concerning for IrAE - Grade 2 pneumonitis. He was initiated on intravenous methylprednisolone 40mg twice daily and empiric levofloxacin, with rapid improvement in his symptoms. He was discharged with oral prednisone 100mg daily with taper over 10 weeks, and Bactrim for pneumocystis prophylaxis. At two weeks post-discharge, the patient remained symptom free with interval CXR demonstrating resolution of infiltrates. Although immunotherapy is rapidly becoming a cornerstone of treatment for malignancy, it is associated with a wide range of IrAEs. Of the documented IrAEs, pneumonitis is relatively common and potentially life-threatening, occurring in up to 11.8% of cases. It is often clinically suspected in the setting of the development or worsening dry cough or DOE, confirmed on imaging with evidence of reticular opacities or GGOs, and laboratory diagnostics ruling out other differential diagnoses. Our patient’s presentation and imaging are consistent with those findings reported from prior studies. Prompt recognition and initiation of steroid therapy is critical for symptomatic management and resolution.

Familiarity with the complex pattern of IrAEs, early recognition of IrAEs, and prompt management are critical to prevent further clinical deterioration and irreversible end-organ damage.

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HYPONATREMIA: AN IMPORTANT ASSOCIATION WITH OSTEOFOROSIS

It is known that hyponatremia is associated with fragility fractures and decreased bone mineral density. A recent meta-analysis showed that hyponatremia increased the odds of fracture at all sites (Odds ratio [OR] 2.34), the odds of osteoporosis (OR 2.67) and mortality risk was high. Chronic mild hyponatremia is associated with cognitive impairment and gait instability, leading to a 67-fold increased OR of falling. Research has revealed that when adjusting for potential confounders, patients with moderate and severe hyponatremia (sodium <129mEq/l), had a significantly lower T score in the lumbar spine (p=0.030). We suspect that many clinicians overlook this important risk factor and may not screen for osteoporosis in patients with chronic hyponatremia, spurring us to present this case.

A 63-year-old Caucasian male former cigarette smoker with a past medical history of bipolar disorder, obesity, hypertension and hyperlipidemia, presented to the emergency department with low back pain after bending over a sink. The MRI revealed an acute compression fracture of the L2 vertebral process and he underwent a kyphoplasty. Two months later, after a fall, he was diagnosed with another compression fracture of the L5 vertebral process and underwent a second kyphoplasty. Given his age and fragility fracture, he was diagnosed with osteoporosis. An initial DXA scan revealed a T score of -2.8 in the lumbar spine (excluding the area of the fracture). Evaluation of secondary causes revealed that his home medications included carbamazepine. His labs were significant for sodium 123mEq/l, serum osmolality 275mOsm/kg, urine sodium 44mEq/l and urine osmolality 201mOsm/kg, consistent with the syndrome of inappropriate antidiuretic hormone secretion (SIADH). His 25 hydroxy vitamin D was 20ng/ml, early morning cortisol, thyroid stimulating hormone, protein electrophoresis, parathyroid hormone and 8am total testosterone were within the reference ranges.

He was managed with teriparatide, 2-liter fluid restriction and ergocalciferol, with improvement in his sodium levels, 25 hydroxy vitamin D and bone density on DXA scan. This case illustrates that hyponatremia is a commonly associated risk factor for severe osteoporosis with recurrent fractures. Therefore, hyponatremia should be included in the workup for secondary causes of osteoporosis and screening for osteoporosis with DXA scan should be considered in patients with chronic hyponatremia.
Worsening Neurologic Symptoms Following Gastrointestinal Infection in a Post-Stroke Patient.

Bidirectional interactions between the brain and the gut have been well-described in pre-clinical and clinical literature. This clinical vignette poses a compelling example of this brain-gut interaction.

A 56-year-old man with a recent prior stroke presented to the Emergency Department with new onset worsening hemiparesis and dysarthria. Four months prior, patient had suffered a right pontine infarct with residual left-sided hemiparesis and mild dysarthria, for which he had been following at a rehabilitation facility. On the morning of presentation, he experienced sudden onset generalized weakness, lightheadedness, and diaphoresis. He also had ambulatory dysfunction and inability to mobilize from a seated position, which was a significant departure from his baseline. Patient denied associated numbness or paresthesia of the face, upper extremities, or lower extremities. However, patient's family members at this time noted significant worsening of his dysarthria, stating that his speech had become incomprehensible. Interestingly, review of systems was significant for a 1-week history of diarrhea, up to 7 episodes per day, productive of brown, non-bloody liquid bowel movements, associated with diffuse abdominal pain and cramping. No recent antibiotic use was noted.

In the hospital, patient underwent head CT, which showed chronic appearing right pontine infarct without evidence for an acute intracranial process. MRI was not able to be performed due to the patient's body habitus. Neurological symptoms did not progress. However, the patient had 3-5 episodes of diarrhea, including one described as bloody and mucoid. Stool examination revealed abundant fecal leukocytes. The patient then received supportive care for presumed infectious diarrhea; in parallel, he demonstrated spontaneous progressive improvement of dysarthria back to baseline. On hospital day 3, stool cultures resulted positive for Salmonella enteritidis. Given patient's HIV status, patient was treated with a 14-day course of ciprofloxacin on discharge.

Neurodegenerative disorders and brain injury (TBI, stroke) have been associated with pathophysiological changes in the gastrointestinal tract; moreover, gut dysbiosis has been demonstrated to exacerbate underlying injury or inflammatory processes in the brain. In the absence of acute cranial pathology, we theorize that this presentation of worsening post-stroke neurologic deficits was associated with the presence of Salmonella infection in the gut. Concurrent functional neuroimaging would be necessary to assess the veracity of this association.
A NOT SO TINY GLAND: A CASE REPORT OF A Pineal Parenchymal Tumor TREATED WITH PROTON THERAPY

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Introduction
Among CNS tumors, pineal neoplasms are extremely rare, with a relative incidence rate of 0.4%. Of these pineal tumors, PPTIDs (Pineal Parenchymal Tumors of Intermediate Differentiation) are uncommon enough that treatment is often based on institutional experience and case reports rather than a “gold standard”. Radiation therapy is a common treatment modality with targeted photon methods becoming increasingly popular. Here we present a novel case of a PPTID being treated with proton therapy (PRT) as opposed to photon therapy (XRT).

Case Description
A 26 year old male with no significant PMH presented with rapid onset altered mental status consisting of mainly anterograde amnesia and perseveration. MRI scan revealed chronic hydrocephalus and a 4.1x2.7x3.3 cm pineal mass obstructing the cerebral aqueduct with infiltration into the 3rd ventricle. Due to tumor’s size and location, neurosurgery determined that the patient was not a surgical candidate. Instead, the patient underwent an endoscopic biopsy and third ventriculostomy procedure with post-op course complicated by one episode of status epilepticus resulting in diplopia and gait instability. Pathology showed low grade PPTID and PRT was recommended. Patient began a course of 30 sessions for a total of 54 Gy, with all remaining symptoms completely resolving one week into course. Last known MRI demonstrates size to be significantly decreased at 3.6x1.8x2.4 cm and possible opening of CSF flow through aqueduct.

Discussion
Based on literature review, gross total resection (GTR) of PPTIDs is a strong prognostic marker for long term survival. However, GTR is often technically unfeasible, such as in this case, which leads to the use of radiation. XRT, as adjuvant or alone, has been shown to be effective at reducing tumor size and improving survival outcomes in PPTIDs. In comparison, there is limited data for PRT due to its novelty and expense. In this report, we demonstrate PRT resulting in significant reduction of tumor volume and resolution of clinical symptoms, which lends support to PRT being a treatmnt option in these tumors. lends support to PRT being non-inferior to XRT in these tumors.

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BREAKOUT ROOM 18

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ACUTE EOSINOPHILIC PNEUMONIA: AN UNUSUAL REACTION TO ADO-TRASTUZAMAB
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Daniel Desmond M.D. – Walter Reed National Military Medical Center

INTRODUCTION: Ado-trastuzumab emtansine (T-DM1) is a monoclonal antibody drug conjugate approved for the treatment of HER2- positive breast cancers. Pulmonary toxicity is rare with T-DM1 but can cause serious or life-threatening pneumonitis. Outlined here is a case of T-DM1-induced eosinophilic pneumonia, the first that we are aware of.

CASE DESCRIPTION: A 73-year-old female with stage II b triplepositive (estrogen/progesterone/HER2 receptor) intraductal carcinoma of the left breast was admitted to the general medicine floor for progressively worsening dyspnea and dry cough. She was being seen by her oncologist for a toxicity check prior to cycle 10 (of 14 planned) of adjuvant T-DM1 and was noted to be hypoxic with ambulation. CT Chest upon admission revealed multifocal bilateral consolidations with diffuse ground-glass opacities. The patient was afebrile and initial laboratory values were unremarkable apart from peripheral eosinophilia. Bronchoscopy with bronchoalveolar lavage revealed 51% eosinophils with normal percentages of neutrophils/lymphocytes, consistent with a diagnosis of acute eosinophilic pneumonia (AEP). Robust infectious disease work-up found no infectious etiology, to include bacterial, viral, parasitic or fungal causes of AEP. High dose corticosteroids were initiated, however the patient’s respiratory status continued to decline, progressing to ARDS and requiring intubation. Her hypoxemic respiratory failure progressed despite maximal critical care support and after 2 weeks of failure to wean from ventilatory support she was compassionately extubated.

CONCLUSION: Presented here is an apparent case of fatal TDM1-induced acute eosinophilic pneumonia (AEP) which did not respond to corticosteroid treatment. AEP has been reported in patients treated with trastuzumab however, this is the first case we are aware of involving this drug conjugate. This could represent the feared consequence of compound related toxicity related to multiple HER-2 directed agents. With the recent FDA approval of a second HER-2 directed antibody drug conjugate (trastuzumab deruxtecan) and tyrosine kinase inhibitor, tucatinib (dosed with trastuzumab), in the 3rd and 4th line settings compound toxicity should be considered in managing these patients with apparent treatment-related toxicity.

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BREAKOUT ROOM 18

ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
ACUTE DIGITAL ISCHEMIA FROM INTRA-ARTERIAL AUTOINJECTION MASQUERADING AS NECROTIZING FASCIITIS. Maldarelli M, MD, Traver E, MD, Schmalzle S, MD. The University of Maryland School of Medicine Baltimore MD.

Among people who inject drugs intravenously, intraarterial injection can be a common practice in the later stages of addiction. Complications of this practice include severe infection, ischemia, and compartment syndrome, all of which may have overlapping presenting symptoms. Limb ischemia from thrombus is an unusual complication of long-term intravenous drug use (IVDU). Here we present a case of a 35 year old man with limb ischemia as a result of intraarterial injection, initially diagnosed as necrotizing fasciitis.

A 35-year-old man presented 3 days after heroin injection to the left foot with severe pain, swelling, and discoloration. Labs showed white blood count 10.1 K/ml, erythrocyte sedimentation rate 51 mm/hr, C Reactive Protein 8.8 mg/dL, lactate of 1.7, and creatinine kinase 1,414 u/L. Given these results and his history of flossing needles, he was suspected to have necrotizing fasciitis with compartment syndrome. Antibiotics were administered, and he underwent surgical exploration of the affected extremity. Surgery revealed healthy tissue. After several days with intact motor and sensation in his affected limb, he developed black discoloration of the toes. Doppler and ankle-brachial index (ABI) studies demonstrated a left ABI of 1.23, a right ABI of 1.28, and 0 mmHg flow to the left great toe, concerning for ischemia. The patient disclosed that he often injected into his arteries, including the inadvertent injection of clotted blood. The patient was unsuccessfully treated with therapeutic anticoagulation and hyperbaric oxygen therapy and subsequently required transmetatarsal amputation.

People who inject drugs, particularly those who intentionally perform intraarterial autoinjection, are at risk for ischemia due to intraarterial thromboemboli, localized toxic vasculitis, or arterial spasm. Education on safe injection techniques may reduce the risk of these serious complications. There is no standard algorithm for limb ischemia from intraarterial drug use, and therefore multimodal treatment approaches have been used. In this case, salvage of the extremity was attempted with intravenous heparin therapy and hyperbaric oxygen therapy but was unsuccessful. In patients with a history of IVDU, limb ischemia should be suspected even in patients who clinically appear to have infection.

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BREAKOUT ROOM 18

ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
CASTLEMAN DISEASE: AN ALGORITHMIC APPROACH TO A RARE MULTIFACTORIAL CONDITION
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Introduction: Castleman disease is a group of lymphoproliferative disease with systemic inflammation associated with HHV-8 and HIV. While relatively rare, the early recognition and initiation of appropriate therapy can significantly improve an otherwise poor prognosis. Both multicentric Castleman disease and Kaposi’s sarcoma are proliferative disorders associated with human herpes virus 8 (HHV-8), most commonly seen in the US in patients with HIV. In the case of Kaposi’s sarcoma, HHV-8 drives dysregulation of cell growth and proliferation of the classic spindle cells while multicentric Castleman disease leads to the development of lymphoma.

Case: A 30-year-old woman with history of HIV complicated by noncompliance and inhaled heroin use present with a 1-week history of exertional dyspnea, nasal congestion and chills. Physical examination revealed significant submandibular and left axillary lymphadenopathy and hepatomegaly. Laboratory findings revealed CD4 count 9, HIV viral load 140 copies/mL, CD8 and B cell lymphocytosis, normocytic anemia consistent with anemia of chronic disease, negative hepatitis panel, and indeterminate QuantiFERON gold. CT-Thorax revealed large right axillary lymph node, and mediastinal and hilar lymphadenopathy. Excisional biopsy of 2 axillary lymph nodes revealed Castleman disease with features of hyaline vascular and plasma cell type and Kaposi’s sarcoma, both HHV8 positive. Bronchoscopy revealed pulmonary involvement of Kaposi’s sarcoma. Treatment included liposomal doxorubicin with rituximab along with continuation of ART, and consideration of ganciclovir for HHV-8. Standard pretreatment evaluation included transthoracic echocardiogram, which revealed normal heart function, and FDG/PET, which revealed extensive widespread lymphadenopathy with spleen hyperactivity. Shortly after initiating chemotherapy, patient developed anasarca, shortness of breath, and peripheral edema requiring readmission. Etiology of symptoms were unclear, although cardiac and pulmonary workup was largely unremarkable.

Discussion: This case illustrates the multiple potential complications of HIV immunosuppression and the algorithmic approach to treatment of HHV-8 multicentric Castleman disease with concurrent Kaposi’s sarcoma. While a rare condition with poor prognosis, continued research into current therapies and ongoing trials may continue to improve median overall survival.

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ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
COVID-19 and PFO: A Dangerous Combination?
Saroj Timilsina MD, Sunita Karki, MBBS, Lauren Berninger, DO

Introduction:
Recent evidence suggests that patients with COVID-19 infection may have increased susceptibility to venous thromboembolism (VTE). Additionally, case reports from New York City and Wuhan, China have identified a potential association between COVID-19 and large vessel strokes. Here we present a case of lower extremity DVT, saddle pulmonary embolism and acute left MCA infarct in the setting of COVID-19 infection.

Case Report:
A 55-year-old woman with a past medical history of meningioma status post resection, remote history of provoked DVT and recently diagnosed COVID-19 infection presented to the ED with two weeks of worsening dyspnea associated with fever, cough and right sided chest pain. Physical exam was remarkable for tachycardia, tachypnea, hypoxia and diminished breath sounds bilaterally. Laboratory studies revealed a leukocytosis of 16,000 with neutrophil predominance (73%) and lymphopenia (13%) as well as mildly elevated troponin and markedly elevated d-dimer, fibrinogen and c-reactive protein. Contrast CT of the chest illustrated a large saddle pulmonary embolism extending to the bilateral main pulmonary arteries in addition to scattered ground glass opacities throughout both lungs consistent with known COVID-19 infection. Due to extensive clot burden and evidence of right heart strain, she underwent catheter directed tPA administration and was subsequently placed on a heparin drip. Her hospital course was complicated by new onset right sided facial droop on day three of admission. MRA with contrast was performed which revealed small acute infarcts within the left middle cerebral artery. Echo with bubble study determined the presence of a patent foramen ovale (PFO), A subsequent ultrasound of lower extremities revealed a left popliteal DVT.

Discussion:
The prevalence of a PFO in the adult population is estimated at 25% and that number is significantly higher in patients with cryptogenic stroke. The coagulopathy and vascular endothelial dysfunction implicated as potential complications of COVID-19 could theoretically increase the risk of paradoxical emboli in the subset of the population with a PFO. Further investigation is needed to examine the incidence of stroke and VTE in COVID-19 patients especially in those patients with an underlying PFO. The most appropriate choice and duration of anticoagulation in these patients should also be explored.