

(continued) their mind or if symptoms worsen. The best possible alternative therapy must be provided. In effect, patients who choose to leave AMA should be offered as much continuing therapy, social support and follow up assistance as we would arrange for all other patients.

References:

Taqueti, VR, Leaving Against Medical Advice, NEJM 2007; 357(3): 213-215

CARE OF THE HOSPITALIZED PATIENT 2009
SATURDAY, MAY 2, WASHINGTON UNIVERSITY
SEE CALENDAR FOR DETAILS

CASE OF THE MONTH ABIGAIL EMERSON, MD & ROBERT LANCEY, MD

A 33 year old African American male was admitted to the University of Missouri I.M. Service, referred from a community hospital for uncontrolled hypertension. He was initially diagnosed with hypertension approximately ten years ago; the condition had been worsening over the past 2-3 months despite escalating doses of antihypertensive agents. In the office on the day of admission, his blood pressure was 210/110 and he was "not feeling good."

The patient was transported to the stepdown unit where more information was obtained. He complained of increasing fatigue, occasional headaches with blurred vision and chest pressure, worse in the morning. He reported increasing periorbital puffiness but was unable to specify a time period; he had lost 55 lbs over the past 8 months via a diet modification program. He had been diagnosed with diabetes 3 months ago and was controlling his glucose with diet and Metformin. Daily glucose checks range from the 130s-140s. He also reported that he had been treated for hypokalemia over the past 2 months and was currently taking K-Dur 60 mEq twice daily.

His past history was remarkable for essential hypertension, diabetes mellitus, GERD and obesity. His current medications included lisinopril, amlodipine, triamterene/HCTZ, Metformin, K-Dur, metoprolol, omeprazole and cyclobenzaprine (as needed). He had no medical allergies. The patient reported a 40 pack-year history of tobacco use (and was still smoking) but denied excessive use of alcohol. He works as a machine operator and is engaged to be married. Family history was remarkable for hypertension and diabetes.

Physical examination revealed a blood pressure of 174/122, T 36.2 C, P 78, R 16 and O2 sat 96% (RA). Significant findings included mild periorbital edema, acanthosis nigricans at the nape of the neck and striae over the trunk and antecubital fossae. There was no JVD, chest was clear to auscultation and no murmur, rubs or gallops were noted. He was fully alert and oriented with clear, fluent speech and no focal neurologic deficits.

Labs revealed Na 140, K 2.5, Cl 97, BUN 12, Cr 1.0, Glucose 190, Ca 8.8 and Alb 3.4. CBC was normal; UA showed trace protein but was otherwise normal. His EKG revealed NSR with no ischemic ST-T changes and his troponin I was 0.02.

Let's stop and consider a few points:

- *What factors could be contributing to his poorly controlled hypertension?*
- *How should his blood pressure be managed at this point?*
- *Should he be evaluated for secondary causes of hypertension or is this just another "routine" case of hypertensive urgency?*
- *What are your thoughts regarding his weight loss, newly diagnosed diabetes and hypokalemia?*

A CT of the abdomen and pelvis was obtained which showed a "large right adrenal region heterogeneously enhancing mass, 7.5 cm x 5.1 cm x 5.2 cm. Multiple enhancing lesions in the liver, multiple bilateral lung parenchymal soft tissue nodules, a small T9 sclerotic focus and minimal pericardial effusion were also noted.

The astute senior resident then orders: ACTH 2 pg/ml (7-69), plasma rennin 0.4 ng/ml (0.2-1.6), plasma aldosterone 112 ng/ml (<31), AM cortisol 31.2 (6.7-22.6), 24-hr urine free cortisol 925 mcg (<85), a low dose dexamethasone suppression test 35.7 mcg/dL, a high dose dexamethasone suppression test 38.9 mcg/dL, DHEA 14.6 ng/ml (1.9-7.6), testosterone 185 (400-1080) and free testosterone 45 (47-244). 24 hr urine metanephrines, VMA and catecholamines were normal

More points to consider:

- *What is your differential diagnosis for this patient?*
- *How do the above results change our management of this patient's hypertension?*
- *What are your next steps in the diagnostic workup?*

Once the patient's blood pressure was stabilized, he was sent home and brought back in four weeks for a laparoscopic adrenalectomy and partial hepatectomy with biopsies. His postoperative course was complicated by hypertensive crisis, respiratory failure and altered mental status. Pathology confirmed the diagnosis of adrenocortical carcinoma and further workup showed metastases in the liver, lung, bone and brain (stage IV disease). After consultation with Hematology-Oncology, the patient opted for hospice care.

Discussion: This case demonstrates a rare cause for secondary hypertension, adrenocortical carcinoma (ACC). The patient's presentation raises the possibility of several endocrinologic causes for hypertension, including Cushing's Syndrome and hyperaldosteronism.

ACC is a rare adrenal malignancy, with a population incidence of 1-2 per million. There is a bimodal age distribution, with a peak under 5 years of age and another in the 4th-5th decades of life; median age at diagnosis is 44 years. Hyperfunction of the adrenal is common, with 60-80% demonstrating hormone secretion. The primary differential diagnoses for this adrenal mass are neuroblastoma and pheochromocytoma. ACC, while usually sporadic, has been associated with hereditary cancer syndromes, including MEN 1 (parathyroid, pituitary and pancreatic neuroendocrine tumors and carcinomas), Beckwith-Wiedemann Syndrome (Wilms' tumor, neuroblastoma, hepatoblastoma, ACC) and Li-Fraumeni Syndrome (breast cancer, soft tissue and bone sarcomas, brain tumors, ACC).

(continued)

ACC is largely a silent disease unless hormone secretion leads to an early diagnosis; 70% of cases have metastasized at the time of diagnosis and common sites include liver, lung, bone and peritoneum. Adult disease tends to be more aggressive and has a poorer prognosis than ACC in children. Treatment includes surgical resection and adjuvant therapy (mitotane and possible radiation). Prognosis is often poor and is related to the completeness of surgical resection and the presence of distant metastases. Several small patient series, published in recent years, suggest an improvement in survival over time.

References: Luton, JP et al., Clinical features of adrenocortical carcinoma, prognostic factors and the effect of mitotane therapy, *NEJM* 1990, 322 (17): 1195-1201

Brennan MF, Adrenocortical carcinoma, *CA Cancer J Clin* 37 (6): 348-65, Nov-Dec 1987

FROM THE JOURNALS

Robert Folzenlogen MD

Efficacy of Esomeprazole for the Treatment of Poorly Controlled Asthma

Mastornade, JG et al., *Am Lung Assoc Asthma Clinical Research Centers*

NEJM, Vol 360, No 15, 1487-1499, April 9, 2009

Conclusion: despite a high prevalence of GERD in asthma patients, PPI use does not improve control

Update in COPD 2008

Maclay, JD et al., *Am J Resp Crit Care Med*, Vol 179, No. 7, 533-541, April, 2009

Review of pathophysiology, genetics,, systemic features, imaging and treatment of COPD

The Role of D-Dimer Testing in Patients with Suspected Venous Thromboembolism

Prisco, D and E. Grifoni, *Seminars Thrombosis Hemostasis*, Vol 35, No. 1, 50-59, Feb 2009

An overview of the rationale, utility and limitations of d-dimer testing

ID CORNER

William Salzer MD

INTRAABDOMINAL INFECTIONS

Attached are the evidence-based guidelines for the antibiotic treatment of intraabdominal infections from the IDSA.

Solomkin, JS et al., Guidelines for the selection of anti-infective agents for complicated intraabdominal infections, *Clin Infect Dis* 2003; 37:997-1005

<http://www.journals.uchicago.edu/doi/pdf/10.1086/378702>