When I started my career, thirty years ago, the general internist was a full service provider, involved in outpatient preventive care, inpatient management, ICU treatment and nursing home care. At that time, management decisions were at the discretion of the physician and his/her patient, with little influence from hospitals and insurance companies.

By the mid 1980’s, healthcare began to change dramatically as HMOs entered the scene, attempting to cut cost by contracting with employers to provide service at standardized rates; of course, this also led to HMO-imposed restrictions on patient care and, if you, as a physician, did not join their legion of providers, many of your patients would soon be lost to the network. This trend has continued and the physician-patient relationship is constantly assaulted by a variety of cost-conscious parties, from Medicare to private insurance companies to Utilization Review Committees.

In the 1990s, the concept of hospital-based physicians began to take hold, offering shift work for internists, a source of coverage for outpatient physicians and more efficient care for hospitalized patients. These benefits were offset by the lack of continuity between inpatient and outpatient care and by the unfortunate fact that a patient’s most life-threatening and stress-filled illnesses were often managed by relative strangers. Nevertheless, financial pressures have favored this system which has been shown to reduce hospital length of stay for most common inpatient conditions. At the same time, the divergence of inpatient and outpatient responsibilities has created a more acceptable life style for most general internists, a relatively overworked and underpaid group.

As we entered the 21st Century, cost-containment measures have continued to expand, attempting to chip away at the soaring cost of new technologies and medications. Terms such as “admission criteria,” “hospital acquired condition” and “evidence-based treatment” have entered our vocabulary. And, while primary care physicians were once slated to be the “gatekeepers” of healthcare, hospitalists have now been placed at the forefront of cost control, compliance and “denial prevention.”
The documentation of pre-existing conditions (decubiti, UTIs, etc) on admission, efforts to prevent hospital-acquired conditions (pneumonia, DVT, falls, etc) and attempts to eliminate hospital readmissions have placed new demands on the hospitalist, who may soon face outcome-based reimbursement. On the horizon is the prospect of “bundled payments,” whereby the government or insurance company will pay a specified amount for a given admission diagnosis, to be distributed among the hospital and all providers (including consultants).

Hospitalists will certainly play a vital role in creating a balance between cost-containment measures and the quality of health care that we provide; after all, inpatient care accounts for a huge percentage of total health care costs. Beyond our adherence to government regulations, insurance demands and utilization decrees, it is important to realize that good care and cost control are not mutually exclusive. More attention to the prophylaxis of hospital-acquired conditions and the adoption of evidence-based standards will address both issues. Finally, a return to the involvement of primary care physicians in patient care decisions, especially when it comes to the aggressive care of elderly and chronically ill patients, could have a significant impact on both cost control and patient satisfaction.

CASE REPORT

Kyle Moylan MD

A 44 year-old man with a history of recurrent deep vein thromboses and pulmonary embolism was admitted to the hospital with bilateral pulmonary emboli and an acute, non-occlusive DVT of the left common femoral vein. He had recently been hospitalized and diagnosed with Coombs-positive hemolytic anemia and was treated with prednisone.

On physical examination, the patient was noted to have a eunuchoid body habitus, with complete absence of chest, axillary and abdominal hair. Gynecomastia was present and his testes were found to be 2cm and firm to palpation.

Hypogonadism was suspected and confirmed by a low total testosterone level of 53 ng/dL (normal 175-781). Gonadotropin levels were elevated, with a follicle-stimulating hormone level of 37.2 munits/ml (normal 1.3 to 19.3) and a luteinizing hormone level of 23.0 munits/ml (normal 1.2-8.6), suggesting a diagnosis of hypergonadotropic hypogonadism, secondary to primary testicular failure. Karyotype results were mosaic 47 XXY/46 XY, consistent with Klinefelter Syndrome.

Additionally, the patient had a moderately positive IgM Cardiolipin at 41 MPL; this serologic finding, combined with his history of recurrent DVTs and Coombs-positive hemolytic anemia, is consistent with the diagnosis of antiphospholipid syndrome. The patient was started on testosterone replacement therapy and continued on long-term anticoagulation prior to discharge.  

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