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IATROGENIC NEUROLOGY

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The incidence of iatrogenic illness is probably underestimated, and iatrogenic illness accounts for a considerable number of all hospital admissions. Iatrogenesis refers to the consequences of medical actions and results from errors of omission or of commission. Although iatrogenesis has a long history, it represents a considerable part of the complexity of modern medicine and dictates the need for rapid dissemination and exchange of medical information. In light of the expansive use of technology, the incidence of iatrogenic illness increases in hospitalized patients, especially the elderly as well as neonates, and with the increasing use of polypharmacy.

Many iatrogenic disorders are due to drug exposures, often due to either prescribing error, dispensing errors, medication administration errors, or patient compliance errors.

Polypharmacy increases the risk of iatrogenic disease, and strategies to reduce polypharmacy are essential. However, errors follow patterns that can be uncovered; most are management errors and many are serious or severe. These human errors may be due to communication problems, premature discharges, delayed transferred to operating rooms, delay transfer to tertiary hospitals, or delayed transfer to intensive care units.

This presentation will review some of the more frequent undesirable neurologic events of interest to internal medicine specialists as well as strategies to prevent their occurrence. Astute and vigilant care and careful monitoring for untoward events are needed to prevent unnecessary distress and morbidity and to reduce the risks of iatrogenic neurologic illness.

Case 1: 31 year old woman had hyperemesis gravidarum in January 1999. At that time, she began to have intermittent oscillopsia and dizziness and was hospitalized for about two days. She also felt lethargic, and “drooping” of one side of the face. While in hospital, she aspirated and had acute respiratory distress that required treatment with a ventilator for five weeks. Ophthalmology consultation obtained during hospitalization found her to have swelling of both optic discs with few peripapillary hemorrhages. A spinal tap did not reveal increased pressure. At the time of hospital discharge, she was fed via a Dobbhoff tube. She has continued to have imbalance and oscillopsia.

On examination: she was an ill appearing woman. Blood pressure was 130/80 mm Hg. Pulse was 85 pm. Neurologic examination showed normal attention and reasoning. Affect and mood were unremarkable. Speech was dysarthric. Language was intact. Immediate and intermittent memory was intact. Cranial nerve examination showed normal visual fields to confrontation. Color vision: she missed 3 out of 15 plates with the right eye and 4 plates with the left eye. There was mild temporal pallor in the right eye; the appearances of the disc, maculae and vessels were otherwise normal. She had gaze-evoked horizontal nystagmus. Additionally, she had small amplitude down-beat nystagmus in upward and downward gaze. Pupils were 6 mm in size and sluggishly reactive to light more on the right than on the left. Facial sensation was normal. Strength of masticatory muscles was normal. There was no facial weakness. Palatal movements were intact. Tongue protruded in the midline and showed no tremor or fasciculations. Motor examination showed normal muscle bulk, tone and strength throughout. Serial finger tapping and rapid alternating movements were normal. Sensory examination showed normal light-touch, pin-prick and position sense. Coordination examination showed normal finger-finger, finger-nose and heel-knee-shin testing. Muscle stretch reflexes were 1+ and symmetric throughout. Plantar responses were flexor bilaterally. Erect posture was normal. There was mild postural instability. She was able to walk with standby assistance. Gait was wide based and unsteady. She could not do tandem, heel or toe walking.

Case 2: a 64 year old man was admitted for cardiac catheterization. He had a history of coronary artery disease with previous bypass surgery in 1984 and previous coronary angioplasty in 1992 and 1996. His last cardiac

catheterization was in January 1998 when no further angioplasty or stenting was done. He was subsequently treated medically. For the last three months he has had recurrent increasing chest pain on exertion and with rest. He was admitted with severe chest pain and was treated with intravenous nitroglycerin and heparin and has been pain free since. At previous catheterizations, he was found to have poor left ventricular function with an ejection fraction calculated at the last catheterization of 9%.

On examination: blood pressure was 92/52 mm Hg and pulse was 88 pm and regular. Jugular venous pressure was not elevated. There was minimal ankle edema. There were bilateral basal crackles. There was a 3/6 holosystolic murmur at the apex and a 1/6 ejection murmur at the left sternal border. Peripheral pulses were intact. Neurologic examination was unremarkable.

ECG showed sinus rhythm and a left bundle branch block. Echocardiography showed poor left ventricular function at the apex and a thrombus at the apex of the left ventricle. Cardiac catheterization was carried out through a 6 French sheath in the right femoral artery with a 6F pigtail and Judkins coronary catheters. A multipurpose catheter was used for the right graft. The left ventricle was not entered because of the thrombus and left ventriculography was not undertaken. There was evidence of severe three vessel coronary artery disease with an occluded LAD, obtuse marginals, and right coronary artery. There were patent vein grafts to the LAD on the right. There was an occluded vein graft in the left circumflex system. No critical lesion that were seen at this time were available to angioplasty or stent.

The patient was treated medically. Intravenous heparin was given for the left ventricular thrombus and the patient was then started on warfarin therapy. He subsequently developed pain in his left thigh, hip and left leg associated with numbness. At the same time, the hemoglobin had been falling to around 8 gm/dl. Neurologic consultation was obtained. On examination he had an antalgic flexed posture and external rotation of the left thigh. There was weakness of the left iliopsoas and left quadriceps with adequate strength of dorsi and plantar flexors, obturators and hamstrings. He had an absent left patellar reflex. Sensory examination showed decreased pin-prick and light-touch on the anterior aspect of the left thigh and slightly below the knee.