Aggressive Pacemaker Twiddler's Syndrome*

Dislodgement of an Active Fixation Ventricular Pacing Electrode

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Pacemaker twiddler's syndrome is the phenomenon whereby a pacemaker patient may actively dislodge a pacing electrode by manipulating, often unconsciously, the permanent implanted pacemaker. This unusual case is remarkable in several respects: surgical precautions, originally taken to secure the pulse generator against migration, proved ineffective; the displaced lead featured an active fixation "screw-in" electrode; and the reported dislodgement occurred not in the early postimplant stage, but fully three months later. It can be concluded that even with advanced lead technology and operative precaution, electrode dislodgement cannot be absolutely prevented. Awareness of this phenomenon, especially by pacemaker follow-up care personnel, could lessen its occurrence.

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Twiddler's syndrome, first described by Bayliss et al.,1 is characterized by a loss of pacing capture that occurs secondarily to electrode retraction. Retractile forces occur because of conscious or unconscious pacemaker manipulation by the patient, who winds (or twists) the lead body up within the pulse generator pocket. This phenomenon has been reported in a variety of conditions.1,2 The authors believe this case to be the first report of a dislodgement due to twiddler’s syndrome of an active fixation electrode, and where surgical precautions, purposely taken to prevent pacemaker "migration," proved ineffective.

CASE REPORT

An 89-year-old man was referred because of pacemaker pulse generator erosion through his skin and consequent infection of the immediate pocket site area. Indications for pacing were confirmed. We removed the infected pacemaker and lead and initiated appropriate antibiotic treatment; contralateral implantation of a new VVI pacing system was planned.

Eight days following removal of the infected system, with continuing antibiotic treatment, we implanted the new pacemaker and lead. Under general anesthesia, an active fixation "screw-in" electrode was inserted via a subclavian introducer. Good lead position within the right ventricle was confirmed by both PA and lateral view fluoroscopy, as well as by measurement of electrical pacing, sensing and impedance data: at .5 ms pulse duration, pacing thresholds were .6 volts, .9 mA, and 650 ohms; the amplitude of the R wave was measured at 7.0 mV.

After the electrode was screwed into position, we reconfirmed thresholds. Multiple sutures were used to secure the lead body to the anchoring sleeve, supplied with the lead for this purpose, and then to the fascia. A small pacemaker was implanted. We took two other precautions to prevent pacemaker migration within the pocket of this thin patient. The pacemaker pocket was created beneath the pectoralis major muscle, and the pacemaker was sutured to the pectoralis minor muscle.

Proper pacemaker function was confirmed intraoperatively and throughout hospitalization. A chest x-ray film, taken three weeks after the implant, revealed maintenance of proper electrode position (Fig 1).

Fourteen weeks postoperation, we received a report of pacemaker failure to pace or capture. Telephone monitoring to evaluate this report revealed a lack of sensing and ineffective bipolar pacing spikes (at 72 ppm) which failed to capture. The underlying heart rate was approximately 80 ppm. The patient was brought by ambulance to the hospital emergency room, where the diagnosis was confirmed. Perforation of the lead electrode was initially suspected, since pacemaker-synchronized twitching at the base of the left rib cage was observed.

Chest x-ray films revealed electrode retraction, from the right ventricle to the left subclavian vein. Another view showed the lead body to be neatly coiled around the pacemaker within the pocket (Fig 2). The observed twitching of the lower left side was considered to be caused by pacemaker stimulation of the phrenic nerve.

We loosened the lead from its anchorings, and with a stylet reinserted, repositioned it. Proper position was confirmed by both PA and lateral fluoroscopy, and by measurement of electrical data. With some extra diligence, the lead was re-anchored using the suture sleeve, and the pacemaker was again sutured securely to the pectoralis minor muscle. Anti-twiddler defenses were supported by instructions to the patient's nursing home staff to be alert to pacemaker manipulations that might reinitiate this problem. Now, 20 months later, pacemaker function continues to be appropriate.

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FIGURE 1. X-ray film three weeks postimplant confirms proper electrode position.
Lung Cancer with Skin Metastasis*

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Cutaneous metastasis from lung cancer is rare, but physicians should understand its significance. We treated eight such patients during a 30-month period at Wilkes-Barre (Pa) General Hospital. The seven men and one woman ranged in age from 46 to 72 years (mean, 59 years). In three, the skin lesion was the first manifestation of the underlying cancer and in another three, it was found coincident with the lung mass. Pathologic findings included small-cell undifferentiated carcinoma in four patients, squamous cell carcinoma in three patients, and large-cell undifferentiated carcinoma in one patient. Seven of the eight primary lung lesions were in the upper lobes. Six patients had clinically occult visceral metastases at the time of skin biopsy. Only one patient survived more than six months following skin metastasis. Biopsy specimens must be taken from all new skin lesions, particularly in patients who smoke or who already have a history of lung cancer.

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"Webster was much possessed by death
And saw the skull beneath the skin . . . "
—T. S. Eliot (1920)
Whispers of Immortality

Cutaneous metastases are rare developments from internal malignant neoplasms. In five large autopsy studies there were 146 cases (2 percent) in 7,196 necropsies. These lesions are usually discrete, round, painless nodules, firm or rubbery, and fixed; they may be multiple. Skin metastases seem to appear more often on the anterior trunk or head and neck but may arise anywhere on the body.

In general, cancers that often metastasize to other organs also do so to the skin. Thus, lung cancer—with its recognized propensity for spread to brain, bone, liver, and adrenal glands—is responsible for the bulk of skin metastases in men and is second only to breast cancer in women. Among patients with bronchogenic carcinoma, 2.8 percent to 7.5 percent will develop cutaneous metastases.

An apparent cluster of such patients at our institution, as well as the interesting presentation of our case 1 (skin nodule of small-cell undifferentiated carcinoma with normal chest roentgenogram), prompted this review. Eight patients with biopsy-proven skin metastasis from bronchogenic carcinoma were treated at the Wilkes-Barre (Pa) General Hospital during the 30-month period, January 1986 through July 1988, and are the basis for this study.

CASE REPORTS

Case 1
A 70-year-old man, a cigarette and pipe smoker with a history of resected basal cell carcinoma of the nose and recent history of weight loss, was referred because of a 2-cm mass of the left breast area. Chest roentgenogram was normal and he had no pulmonary

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REFERENCES

Figure 2. X-ray film 14 weeks postimplant reveals lead coiled around pacemaker, with tip in left subclavian vein.