Recurrence of hyperhidrosis after endoscopic transthoracic sympathectomy—case report and review of the literature

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Summary

We describe a patient who underwent upper thoracic sympathectomy for palmar hyperhidrosis, and whose symptoms subsequently deteriorated, becoming worse than those on initial presentation.

Hyperhidrosis is defined as sweating above and beyond physiological needs, particularly in response to heat or emotional stimuli. It is associated with significant psychosocial, educational and emotional handicaps. Distinguishing normal sweating from hyperhidrosis is largely subjective, but this may be achieved objectively by means of a palmar sweat test, (levels greater than three standard deviations from mean controls providing evidence of hyperhidrosis).[1] There are few epidemiological data available, but in a pilot study in 1977 Adar *et al.* estimated the prevalence at 0·6–1% in a poorly defined population of young Israelis.[2]

A number of therapeutic options for the treatment of hyperhidrosis are available, including surgery for patients whose condition is unresponsive to medical intervention. Sympathectomy is the surgical treatment of choice for axillary disease and is the only surgical option for palmar and plantar disease. Minimally invasive techniques have been developed, including endoscopic surgery, and more people are undergoing this procedure which is promoted as a cheap, safe and effective alternative to open surgery. To date, approximately 900 cases have been treated using these techniques, but there is little information regarding their long-term outcome.

Case report

A 24-year-old woman presented 3 years following endoscopic transthoracic sympathectomy (ETS) for palmar hyperhidrosis, with recurrent severe palmar and axillary hyperhidrosis as well as marked gustatory and compensatory sweating. Hyperhidrosis of the palms began at the age of 9, and was sufficiently severe to interfere with her schoolwork. She later developed axillary and plantar hyperhidrosis. Clinical examination and laboratory investigations at that time and subsequently excluded secondary causes of hyperhidrosis. Treatments included topical 20% aluminium chloride hexahydrate, tap water iontophoresis, and later iontophoresis with the addition of anticholinergic agents, but these were ineffective.

In 1991 she was referred for ETS. This was performed first on the right, then the left side, and involved ablation of the second to fourth thoracic ganglia and their interconnections by electrocautery. Her postoperative course was complicated by a pneumothorax requiring drainage.

Initially, she had an excellent result with complete cessation of palmar sweating, and some improvement in both axillary and plantar sweating. However, mild compensatory sweating of the trunk developed almost immediately postoperatively, and gustatory sweating (particularly with vinegary foods and chocolate) a month later. Over the following 18 months she noticed progressive clamminess of both hands and axillae, very marked compensatory sweating of the trunk and thighs, and increasing gustatory sweating.

Discussion

Primary hyperhidrosis frequently presents in childhood or early adolescence. Its aetiology is unknown, but a family history is present in 25% of cases. An autosomal dominant form with incomplete penetrance has been described.[1] Our patient's younger sister was also found to have hyperhidrosis of the palms, but both parents and grandparents in this family were unaffected. Hyperhidrosis affects the palms in 20% of cases, the axillae in 37% and both palms and axillae in 43%.[3] There is often associated increased plantar sweating.

Secondary causes of hyperhidrosis should be excluded, including neurological (hypothalamic lesions and hyperpituitarism) and endocrine diseases (diabetes, hypoglycaemia, thyrotoxicosis, phaeochromocytoma, carcinoid syndrome), chronic infections (TB, brucellosis), malignancies (lymphoma), obesity and anxiety states.

Treatment is difficult. Initially, medical treatments should be prescribed, including topical aluminium chloride hexahydrate, glutaraldehyde and tannic acid. They are effective predominantly in patients with mild disease, and are thought to act by obstructing sweat gland pores or by causing atrophy of secretory cells. Effects are transient, lasting approximately 48 h. Use of aluminium chloride may be complicated by local irritation, although this may be alleviated by mild topical steroids.[4] Both glutaraldehyde and tannic acid cause a brown discoloration of the treated areas making them cosmetically unacceptable, except on the soles.[5]

Iontophoresis is a second line of treatment and relies upon the principle of introducing ions into the skin by means of an electrical current supplied by a galvanic generator via electrodes. It is thought to act by coagulating sweat glands thereby inhibiting secretion. Tap water alone[6] or with the addition of anticholinergic agents such as glycopyrrhonium bromide[7] or aluminium chloride may be used and can be effective in up to 87% of patients, particularly older patients with no family history.[8] It is relatively time-consuming treatment, since sessions last 20 min and often need to be repeated three to six times weekly to achieve optimal effect. Average remission times are 30 days, but maintenance treatments can be repeated as often as necessary thereafter.

Systemic anticholinergic agents may also be effective, since sweat glands are innervated by sympathetic choli-nergic fibres. However, doses sufficient to relieve symptoms are usually associated with side-effects, such as blurred vision, dry mouth and urinary retention, making them unacceptable for all but short-term use.

There are anecdotal reports of success with diltiazem, a calcium channel blocker, thought to act by preventing the initial influx of calcium into the secretory cells necessary for the active secretion of ions and water.[1] Clonazepam, an anxiolytic, has also been reported to be of benefit, although its mechanism of action is unknown.[9] In those patients who fail to respond to medical therapy, surgical intervention may be recommended. For axillary disease, these include local measures as well as open and endoscopic sympathectomy. Surgical excision of skin bearing eccrine glands at the apex of the axilla may be effective for localized disease[10] but fails in over 30% of cases and may be complicated by painful scarring and decreased arm mobility.[11] Suction-assisted removal of axillary subepidermal tissue, including sweat glands, has also been attempted but long-term results are unknown.[12]

The surgical treatment of choice for axillary disease and the only surgical option for palmar and plantar disease is sympathectomy. The sympathetic autonomic outflow comes from all 12 thoracic and the first two lumbar segments of the spinal cord. Cells arise in the lateral horn of the spinal cord, emerge in an anterior root to enter the corresponding sympathetic ganglion and then either synapse here or travel up or down the
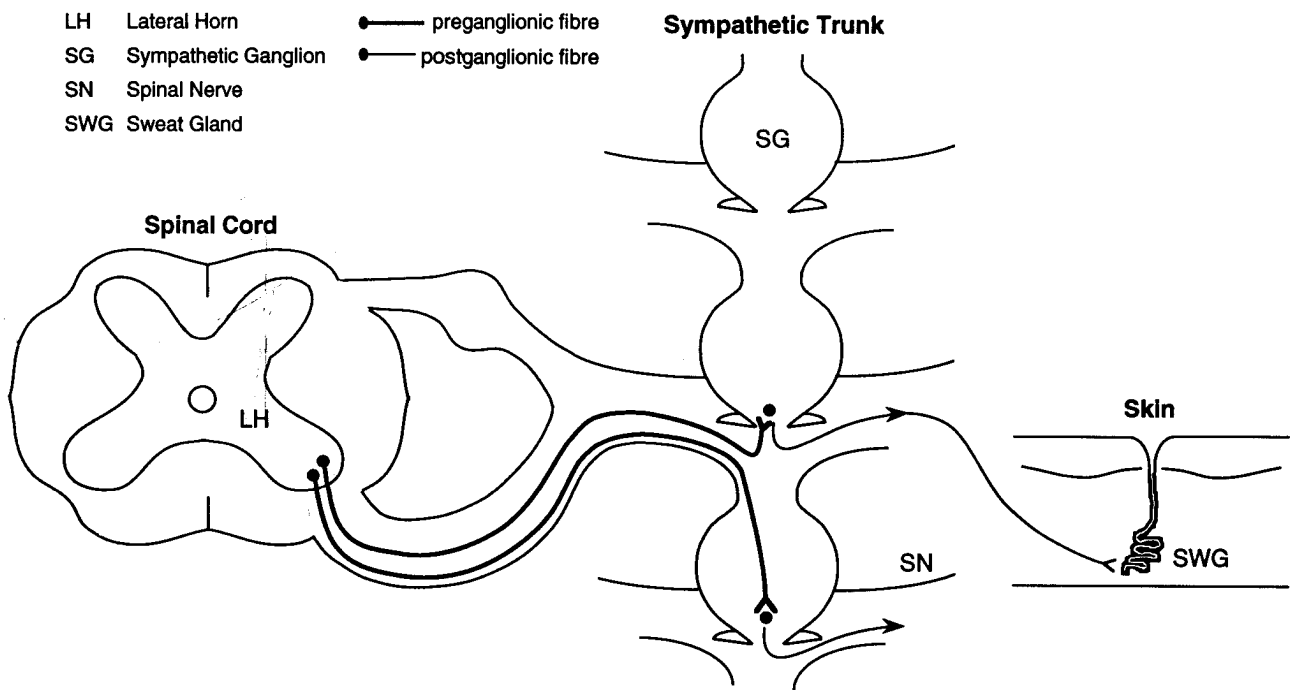

LH   Lateral Horn   ●—— preganglionic fibre   **Sympathetic Trunk**
SG   Sympathetic Ganglion ●—— postganglionic fibre
SN   Spinal Nerve
SWG   Sweat Gland

Figure 1. Schematic representation of sympathetic autonomic outflow.

sympathetic chain to synapse in another sympathetic ganglion. Cholinergic postsynaptic fibres join the corresponding spinal nerve from whence they are distributed to the sweat glands (Fig. 1). The sympathetic outflow to the upper limb is usually from the T2 to T6 ganglia; in 10% of patients, fibres from T1 (in the lower part of the stellate ganglion) may also contribute to arm innervation. In a further 10% the nerve of Kuntz, with fibres arising in T2 and T3, joins the brachial plexus bypassing the sympathetic chain.[13] These anatomical variations may account for some of the treatment failures.

Most surgeons agree that denervation of the palms requires ablation of the T2 ganglion and its connections[14] and many would also include T3.[15] However, this procedure yields complete axillary drying in only 8% of cases,[2] hence axillary denervation additionally requires ablation of the T4 ganglion[16] and some would also include T5.[17]

For plantar hyperhidrosis, lumbar sympathectomy may be performed, ablating L2–L4 ganglia and their connections, and this results in denervation of the distal third of the leg.

There are several different open and endoscopic surgical techniques for treatment of palmar and axillary hyperhidrosis. Complications directly related to the open operative techniques occur in over 40% of patients and include vascular and brachial plexus injuries, Horners syndrome, pneumothorax and with the axillary route, extensive and painful scarring, postoperative neuralgia and wound infections.[18] ETS, first described by Kux in 1951,[19] is reported to cause fewer postoperative complications, and involves shorter operation times (30–45 min), hospital stay (2–3 days) and convalescent periods.[15]

The immediate failure rate post-sympathectomy is 2% for open and 1% for endoscopic procedures. The long-term failure rate for open procedures ranges from 0 to 5%, usually within 2–18 months of surgery,[18] but there are few data regarding long-term outcome following endoscopic procedures. Series reported to date suggest a similar incidence of 2–5%,[17,20] but in one study 5% of patients 3 years postoperatively had worse than original symptomatology because of marked compensatory sweating.[17]

Recurrence of hyperhidrosis may occur several years after a successful outcome. The causes may include only partial denervation by diathermy and/or subsequent nerve regeneration (evidence of nerve regeneration has been found in surgical specimens at second operation).[21] Failure to identify the nerve of Kuntz or some other anatomical variation may also be contributory.

Three other complications common to both endoscopic and open sympathectomy are compensatory, gustatory and phantom sweating. The majority of patients find them only a minor inconvenience and preferable to their original symptoms. In one series further investigation and treatment was warranted in only 5% of patients and initially severe symptoms considerably improved in 10 of 54 at a mean of 10·1 months postoperatively.[17]

Compensatory sweating usually occurs in response to heat or emotional stimuli and involves the trunk and thighs (but may also include the soles). There is no satisfactory explanation for its occurrence, but it has been suggested that as up to 40% of sweat gland function is lost following bilateral upper thoracic sympathectomy, it may be a thermoregulatory response.[22] It occurs in 37–75% of patients in whom the T2–T4 ganglia are ablated.[16] Axillary hyperhidrosis may occur postoperatively if the axillae are not denervated.[23] The incidence parallels the extent of the sympathectomy[3] and may be reduced to 16–20% if only T2 and its connections are destroyed.

Gustatory sweating may occur in 0–56% of patients;[15] lower incidences are reported in patients not directly asked about their symptoms. It usually occurs with spicy or acidic foods or cheeses and is thought to be due to collateral sprouting or aberrant regeneration of preganglionic fibres synapsing in the superior cervical ganglion. Symptoms may be reduced by extending the sympathectomy to involve the lower third of the stellate ganglion (which also increases the likelihood of postoperative Horners syndrome).[3] Phantom sweating is the sensation of impending hyperhidrosis in the absence of sweating; the mechanism is poorly understood. It is reported in 4–26% of patients usually within 18 months of surgery.[18]

Our patient, who developed recurrent hyperhidrosis, compensatory and gustatory sweating following endoscopic upper thoracic sympathectomy, illustrates that this is not a procedure without complications. Other recently developed minimally invasive techniques, including percutaneous radiofrequency ablation (successful in 24 of 27 reported cases[24]), computerized tomography-guided percutaneous phenol injection,[25] and a stereotactic thermocoagulation technique,[26] will require further investigation to assess their long-term efficacy and complications.

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