Heat-Triggered Reticular Telangiectatic Erythema Induced by a Spinal Cord Stimulator

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Abstract

In recent years, cutaneous complications have been reported after implantation of medical devices as a result of their widespread use. We report a case of reticular telangiectatic erythema (RTE) after replacement of a spinal cord stimulator. To date, the pathogenesis of RTE has been poorly understood. Some reports have suggested that heat is involved, whereas others seem to contradict this observation. In our thermographic study, we found that heat can cause RTE.

CASE REPORT

A 72-year-old man with a 4-month history of progressive telangiectatic erythema on his abdomen was referred to our department in December 2010. The erythema had appeared 10 days after replacement of an SCS pulse generator and connector block (PrimeAdvanced Neurostimulator, model 37702). The patient had a history of refractory angina pectoris (third degree according to the grading of the Canadian Cardiovascular Society), which led to implantation of an SCS 8 years earlier. Examination of the skin revealed a 30 x 15-cm warm, reticular, slightly scaly erythematous patch on the left side of the abdomen (Figure, A).

Routine laboratory investigations revealed only a slightly elevated C-reactive protein concentration (15.4 mg/L [reference range, 5 mg/L]). Allergy skin patch testing of all relevant components of the SCS that were provided by the manufacturer yielded normal results. Histologic investigation of a punch biopsy specimen from the abdomen revealed telangiectasia with isolated eosinophilic granulocytes and an isolated lymphocytic infiltration in the upper and middle dermis. Together, these findings suggested the diagnosis of RTE after implantation of a medical device. However, to elucidate the marked hyperthermia accompanying the erythema, we performed an examination using a thermal camera (ThermaCam PM 595; FLIR Systems, Inc) in a special climatic chamber under stable environmental conditions (temperature, 21°C; humidity, 48%). The image indicated substantial overheating in the region around the SCS (Figure, B), which caused us to suspect heat-triggered RTE induced by the SCS. To confirm this hypothesis, we removed the heat source by switching off the SCS; after 10 days, the erythema had largely disappeared (Figure, C). The SCS electrode and the pulse generator were replaced. Technical examination revealed that the removed device had a broken extension electrode sheath (Figure, D). The damaged extension may have caused leakage of current and consequent overheating of local tissue.
DISCUSSION
Since 1967, spinal cord stimulation has been used in the treatment of many chronic pain disorders including severe angina. It is an adjustable, nondestructive neuromodulatory procedure that delivers therapeutic doses of electrical current to the spinal cord. Adverse events associated with permanent SCS implantation include paralysis or severe neurologic deficit, infection of the implanted hardware, leakage of cerebrospinal fluid, painful stimulation, and persistent pain at the implant site. Moreover, breakage of the implanted hardware or migration of the electrode has been observed in 11% to 45% of implanted systems.

In the present case, a broken extension electrode sheath of an SCS may have caused leakage of current. Because the electrical circuit remains closed, leakage of current is derived from the local body tissue. Flowing current causes (localized) heat, which usually is followed by a functional telangiectatic expansion of blood vessels. We speculated that this may have produced the particular clinical pattern. This is strongly supported by results of the thermographic study and the observation that no skin changes occurred after replacement of the broken device. Nevertheless, other differential diagnoses such as an irritant or allergic contact dermatitis and (bacterial) inflammatory...
processes were discussed. Because skin changes developed in about 10 days, an irritant co-factor cannot be definitely excluded. Allergic contact dermatitis was unlikely because (1) results of patch testing with all material of the SCS yielded normal results; (2) the SCS with the broken extension electrode sheath was replaced by an identical device consisting of the same material and has resulted in no adverse reaction; and (3) in the detailed medical history, the patient gave no information about any contact substances with sensitizing potential such as topical creams, ointments, or cosmetics.

In the present case, the most likely diagnosis of heat-triggered RTE was made. To date, the pathogenesis of RTE has been poorly understood. Some reports have suggested that heat is involved in RTE, whereas others seem to contradict these observations. In many cases, RTE is classified as idiopathic and is a diagnosis of exclusion. Inasmuch as the use of implanted medical devices has increased substantially in recent years, the frequency of cutaneous complications may also be expected to increase, and the diagnosis of RTE might become more prevalent.

**CONCLUSION**

In the present case of heat-triggered RTE induced by an SCS, thermography suggested that a broken extension electrode sheath may have caused leakage of current and consequent overheating of local tissue. After switching off the SCS, the erythema largely disappeared after 10 days. Results of the thermographic study and the observation that no skin changes occurred after replacement of the broken device confirms that heat can cause RTE.

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