

CASE REPORTS

Scleroderma, Raynaud's Phenomenon, and
Local Anesthetics

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Patients with connective-tissue diseases often pose problems for the anesthesiologist since they may have complicated drug histories, anatomic alterations, or unsuspected multiorgan involvement. We report the case of a patient who had scleroderma with an unusual degree of impaired circulation and prolonged sensory anesthesia following lidocaine.

REPORT OF A CASE

A 72-year-old man was admitted to the hospital with a long history of scleroderma with marked thickening and pigmentation of the skin of the feet, hands, and forearms; advanced digital ulcerations; loss of ability to tear; and esophageal changes causing dysphagia. There was no evidence of other organ involvement. Thirteen years prior to admission, a bilateral thoracic sympathectomy had been performed, with some improvement in the Raynaud's phenomenon associated with his disease.

On the present admission an attempt was made to evaluate sympathetic tone, if any, in the right arm, so that amputation of the fingers might be avoided. Axillary block with 20 ml of 1 per cent lidocaine with epinephrine, 1/100,000, produced no change in forearm skin temperature or blood flow (measured by constant-temperature plethysmograph); however, sensory anesthesia (pin prick) persisted for 24 hours. To explore this further, subcutaneous injections (2-3 ml) of 1 per cent lidocaine, both with and without epinephrine, 1/100,000, were made on the opposite upper arm and over the anterior chest wall. As shown in table 1, the duration of sensory anesthesia with lidocaine was seven to eight hours. In adjacent areas where lidocaine with epinephrine was injected, sensory anesthesia lasted 10-12 hours.

DISCUSSION

This case is unusual because of the prolonged effect of lidocaine, especially in an area (trunk) not severely affected by connective-tissue disease. In scleroderma, forearm subcutaneous tissue and forehead skin blood flows may be at least 30 to 50 per cent less than

those in control subjects.¹ In normal subjects the addition of epinephrine to local anesthetics injected subcutaneously may prolong the duration of anesthesia fourfold or more, depending on the vascularity of the area.² In this case, however, epinephrine lengthened the anesthesia much less than expected. This suggests that tissue blood flow was so poor as to be relatively unaffected by vasoconstriction. This is of interest because of the previous thoracic sympathectomy and the reported monoamine oxidase depletion in scleroderma,³ which should make the blood vessels more sensitive to catecholamines. Severely impaired perfusion might lower tissue pH, which would reduce both the effectiveness of epinephrine and the amount of lidocaine free base; however, regional pH data in scleroderma have not been reported. It is worth comment that Ritchie⁴ has shown the cationic form of lidocaine to be more effective than the free base in blocking the nonmyelinated desheathed nerve.

This case raises the question of lidocaine metabolism, an oxidative process believed to occur only in the liver. However, in scleroderma the liver usually is not involved unless there is right heart failure,⁵ which this patient did not have. Though the mechanism for prolonged lidocaine anesthesia is not clear, this case points out that in scleroderma patients, nerve blocks, regardless of the area to be anesthetized, can be achieved with very little anesthetic drug. It seems advisable that an ester-linked rather than an amide-linked local anesthetic be used, since procaine is less fat-soluble than lidocaine and its disposition is more rapid in all tissues.⁶

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TABLE 1. Results of Local Anesthetic Procedure in a Patient with Scleroderma

Area of Injection	Drug	Dose (ml)	Duration of Sensory Anesthesia (hours)
Right axilla (deep)	Lidocaine, 1 per cent, with epinephrine	20	24
Left upper arm (subcutaneous)	Lidocaine, 1 per cent	2	8
Left upper arm (subcutaneous)	Lidocaine, 1 per cent, with epinephrine	2	12
Left anterior abdomen (subcutaneous)	Lidocaine, 1 per cent	2	7
Left anterior abdomen (subcutaneous)	Lidocaine, 1 per cent, with epinephrine	2	10

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Malignant Hyperthermia—An Ounce of Prevention

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Malignant hyperthermia¹ is characterized by fever, often associated with rigidity, following the administration of a muscle relaxant and/or potent inhalational anesthetic agent.² In most reports describing this syndrome a plea is made for early recognition and treatment³ as a means of decreasing the alarming mortality rate, which is approximately 60 per cent.¹ The following report describes a patient in whom the symptoms were recognized and treated early, leading to a remarkably benign clinical course.

REPORT OF A CASE

A 5-year-old girl, one of identical twins, was scheduled for tonsillectomy and adenoidectomy. The child weighed 18.2 kg. Aside from a history of allergy to several antigens and several bouts of pneumonia and bronchitis, she was an active, healthy child. The hemoglobin was 12.9 gm, leukocyte count 8,900. Premedication consisted of a 60-mg pentobarbital suppository and 0.4 mg atropine, intramuscularly, an hour prior to opera-

tion. Anesthesia was induced with nitrous oxide-oxygen-halothane, and five minutes after induction a single dose of 20 mg of succinylcholine was given intravenously. The patient did not relax and the jaw became so rigid that it was impossible to open it to attempt intubation. At this time the heart rate was 160 beats/min. The possibility that this response represented an early symptom of malignant hyperthermia was entertained and the procedure was terminated. The rectal temperature was 37.5 C. The patient was taken to the recovery room and immediately wrapped in two cooling blankets of the Aqua-K-Thermia type with the servomechanism set to maintain the temperature at 37 C for the duration of the day (18 hours). The heart rate of 160 beats/min promptly decreased over a period of 20 minutes to 100 beats/min. Blood-gas values at the time of admission to the recovery room were: pH 7.47; PaCO₂ 24 mm Hg; base deficit 4; PaO₂ 96 mm Hg. These results prompted us to feel that the excessive hypermetabolic state associated with the syndrome had been aborted, and no further therapy was instituted.

A tonsillectomy and adenoidectomy scheduled to be performed on the patient's identical twin was cancelled, although the preoperative medication of 60 mg nembutal and 0.4 mg atropine had been given, and studies were undertaken to determine whether our presumed diagnosis was correct. Table 1 shows the results of the laboratory tests

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