Clinical Case Report

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Sciatic neuropathy caused by a pressure ulcer

A case report

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Abstract

Rationale: Sciatic neuropathy has various causes; however, cases in which a pressure ulcer led to sciatic neuropathy have not been reported to date.

Patient concerns: A 33-year-old woman with no pre-existing mobility problems visited our department with the chief complaint of an extensive pressure ulcer and necrosis in her right buttock. She had a medical history of being bedridden for 2 days while in a coma due to a drug overdose 2 months previously. Physical examination revealed loss of sensation and foot drop in the right foot.

Diagnosis: Physical examination, magnetic resonance imaging, and nerve conduction studies were conducted; the patient was diagnosed with a common peroneal branch injury of the right sciatic nerve.

Interventions: The necrotic tissue was debrided and sciatic nerve decompression was performed, followed by frequent dressing changes. In addition, psychiatric treatment and physical therapy were performed simultaneously.

Outcomes: The pressure ulcer decreased in size and healed to some extent with granulation tissue. However, gait disorders, accompanied by symptoms of sciatic neuropathy, continued. The patient was transferred to the department of gastroenterology for the treatment of toxic hepatitis, which occurred during her inpatient treatment.

Lessons: Physicians should be aware that sciatic neuropathy may occur during the treatment of patients with a pressure ulcer who exhibit no symptoms of paraplegia or quadriplegia. To prevent neuropathy, aggressive treatment of the pressure ulcer is necessary.

Abbreviations: AH = adductor hallucis, CMAP = compound muscle action potential, CV = conduction velocity, DL = distal latency, EDB = extensor digitorum brevis, EMG = electromyography, MRI = magnetic resonance imaging, NE = not evoked, SNAP = sensory nerve action potential, TA = tibialis anterior.

Keywords: pressure sore, sciatic nerve, sciatic neuropathy

1. Introduction

Peripheral neuropathy, a neurological condition, occurs as a result of injury to a peripheral nerve, causing weakness, numbness, and pain. Physicians often encounter patients with neuropathy, and making an accurate diagnosis of the underlying cause is important because the treatment and prognosis of neuropathy are dependent on cause. The most common causes of sciatic neuropathy are hip surgery and trauma, followed by external compression, direct nerve injury, and ischemia. Other known causes include intragluteal injections, compartment syndrome, infection, inflammation, intraneural tumors, vascular causes, gynecological causes, piriformis syndrome, and radiotherapy.[1–6] However, no report has described a pressure ulcer causing neuropathy. Herein, the authors report a case of sciatic neuropathy caused by a pressure ulcer in a 33-year-old female patient without pre-existing mobility problems.

2. Case report

A 33-year-old woman presented with a chief complaint of a pressure ulcer measuring 25 × 10 cm that had appeared 1 month previously on her right buttock. The patient received medication to treat paranoid type schizophrenia for 12 years. One month before the occurrence of the pressure ulcer, the patient overdosed and was bedridden for 2 days in a coma, which presumably caused the pressure ulcer to form on her right buttock. The lesion had deteriorated over the following month. At the time of the patient's visit, erythema, pain, and swelling was observed near the lesion, suggesting inflammation, and massive areas of necrotic tissue were found (Fig. 1). According to the National Pressure Ulcer Advisory Panel (NPUAP) staging system, it was classified as unstageable.[7]

Physical examination of the right lower extremity indicated symptoms of sensory deficit in the anterolateral aspect of the calf, dorsomedial and dorsolateral aspect of the foot, first dorsal web space, and great toe. Furthermore, right foot dorsiflexion exhibited significant weakness, with a Medical Research Council scale grade of 1 of 5 (Figs. 2A, B). A common peroneal branch injury in the sciatic nerve was clinically suspected. Magnetic

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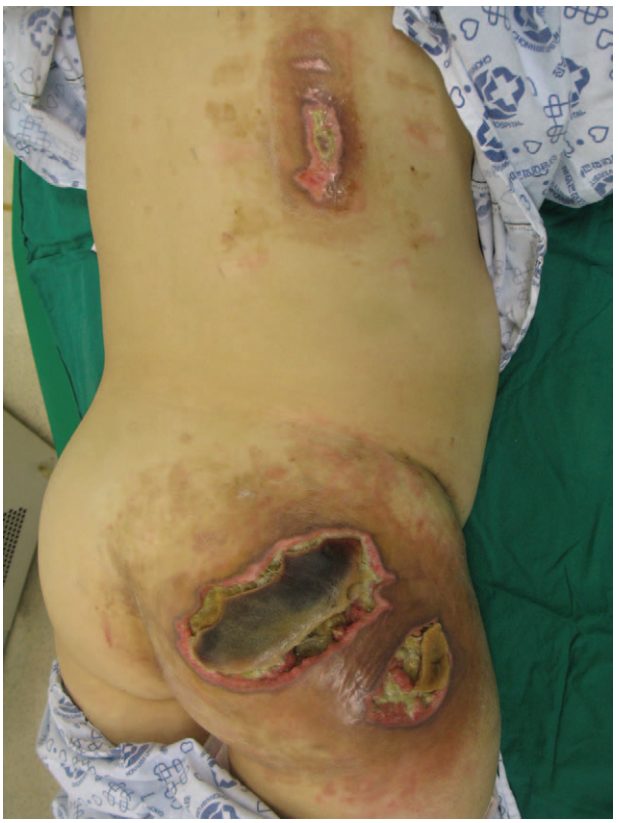

Figure 1. Photograph of the wide area of necrotic tissue and pressure ulcer on the right buttock at the patient's initial visit.

resonance imaging (MRI) revealed general edematous changes in the right buttock and inflammatory soft tissue swelling near the sciatic nerve (Fig. 3A, B). Further sensory and motor nerve conduction studies indicated signs of common peroneal branch injury in the sciatic nerve (Tables 1 and 2). It was concluded that the sciatic neuropathy was induced by inflammatory soft tissue swelling near the sciatic nerve, which was caused by the pressure ulcer.

To treat the necrotic and inflammatory soft tissue swelling, debridement and decompression were performed under general anesthesia. A 30×25 cm sized soft tissue defects remained, and greater trochanter, lesser trochanter, and neck of femur were exposed (Fig. 4). Frequent dressing changes were administered on the remaining soft tissue defects and the patient underwent psychiatric treatment with concomitant physical therapy.

After 6 months of frequent dressing changes and serial debridement, the pressure ulcer exhibited moderate improvement. Soft tissue defects decreased in size from 30×25 to 25×15 cm and healed with granulation tissue (Fig. 5A, B). Although a gait disorder was still present as a sequela of sciatic neuropathy, the patient's motor function recovered to some extent with MRC grade 2 of 5. However, valproic acid, which was administered as a psychiatric medication while the patient was admitted to the hospital, caused toxic hepatitis. The patient was transferred to the department of gastroenterology for the treatment of toxic hepatitis.

The patient provided informed consent for the publication of her clinical and radiological data. This study was approved by the Institutional Review Board of Chonnam National University Hospital (Gwangju, Korea) and was conducted in accordance with the principles of the Helsinki Declaration II.

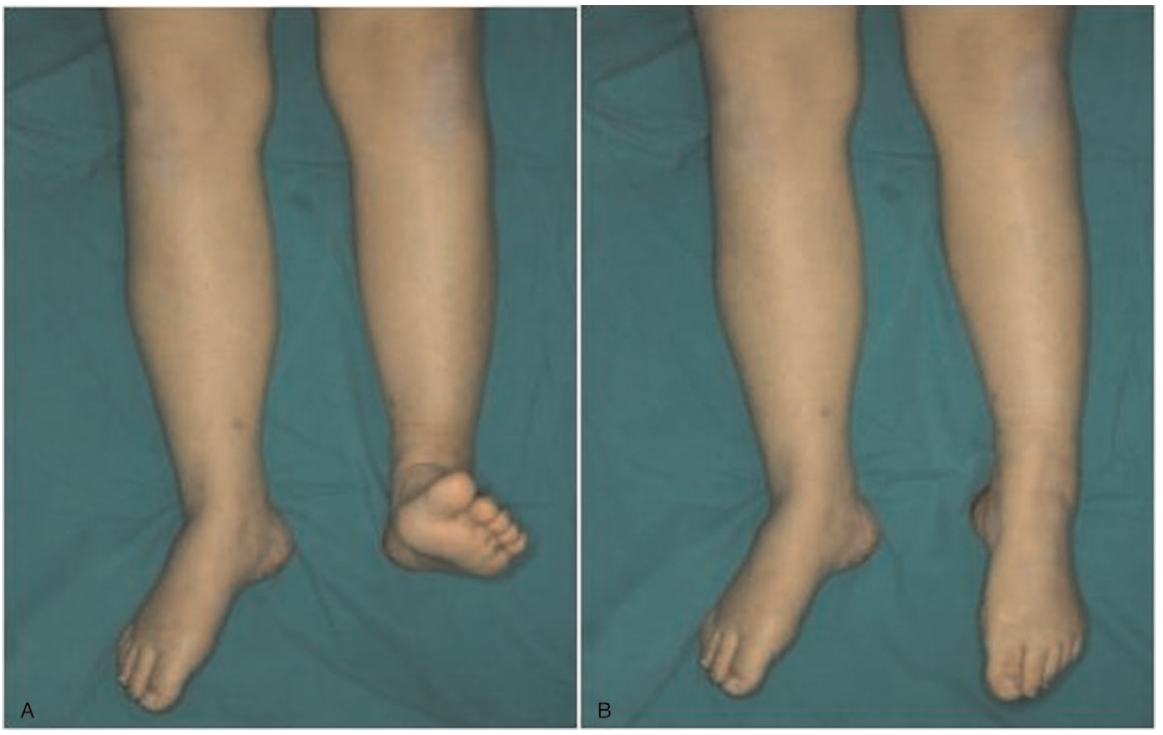

Figure 2. Full dorsiflexion (A) and plantar flexion (B) of both ankles. The patient had no dorsiflexion in the right foot.

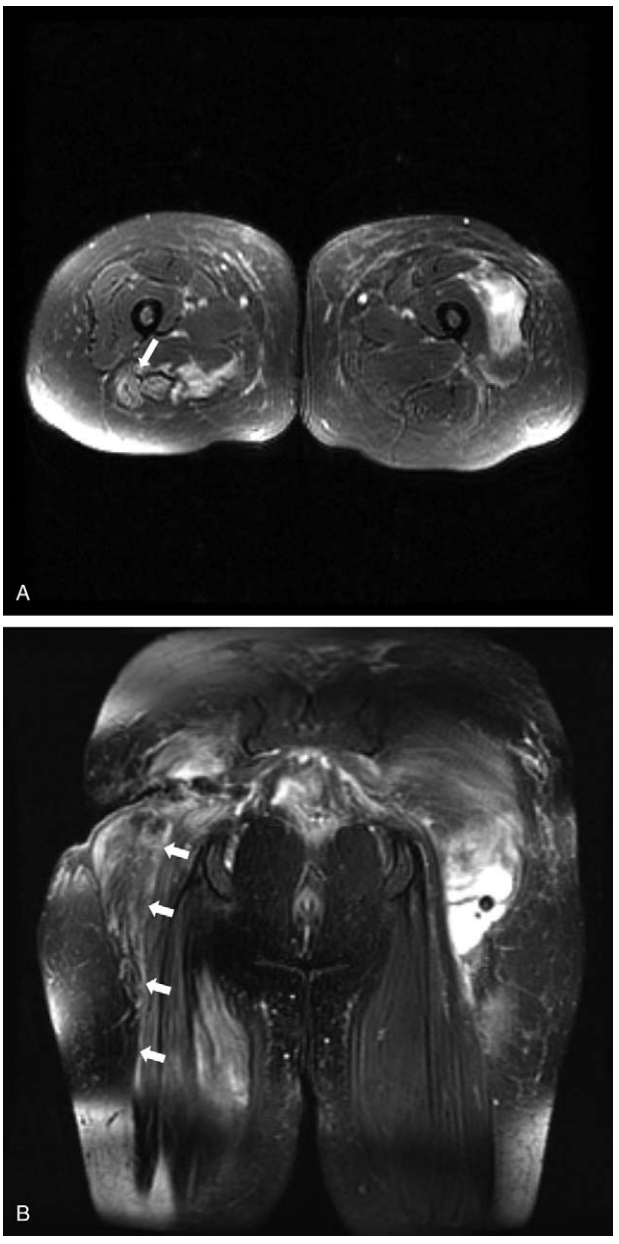

Figure 3. Axial T2-weighted fat suppression (A) and coronal T2-weighted fat suppression (B). Magnetic resonance images reveal edematous changes with a reticular pattern or inflammatory soft-tissue swelling around the sciatic nerve (arrows) from the level of the right sciatic notch to the level of the right subtrochanteric femoral neck. The left sciatic nerve appears to be normal.

3. Discussion

The sciatic nerve runs through the lower extremity and is the largest and longest nerve in the human body. The sciatic nerve divides into the tibial nerve and common peroneal nerve branches, and runs to the feet.[8] It is susceptible to injury due to its long length, and sciatic neuropathy can occur due to several causes, including fracture, dislocation, hematoma, and hip replacement surgery,[1–6] leading to neurological deficits. Sciatic neuropathy is diagnosed based on nerve conduction studies, electromyography (EMG), and MRI. One of the most common symptoms of sciatic neuropathy is foot drop; other symptoms include weakened knee flexion, pain and sensory loss in the foot, and decreased ankle jerk.[1,2] It is important to determine the exact cause of sciatic neuropathy before treatment. For example, if a patient has piriformis syndrome, the first-line therapy should be conservative therapy or botulinum toxin injection, and tricyclic antidepressants or anticonvulsants should be administered to control neuropathic pain. In contrast, surgery is the treatment of choice if it is accompanied by compartment syndrome.[1,4] In this study, debridement and decompression were combined with rehabilitation to treat a decubitus ulcer that was accompanied by inflammation and edema, which caused sciatic neuropathy.

Electrodiagnosis is a very useful method for diagnosing sciatic neuropathy. The following electrodiagnostic findings are characteristic of sciatic neuropathy. Motor nerve conduction studies demonstrate typically decreased amplitudes of compound muscle action potentials in the peroneal and tibial nerves. The F latency of the peroneal and tibial nerves is absent or prolonged, and sensory nerve conduction studies reveal decreased amplitudes of the superficial peroneal sensory and sural sensory nerve action potential.[1,2] In addition, a needle EMG, in addition to a nerve conduction study, is necessary to distinguish between sciatic neuropathy and peroneal mononeuropathy.[1] However, needle EMG was not performed in this case because of the high risk of infection from the soft tissue defects in the right buttock.

MRI is the optimal medical imaging technique for evaluating pathologies of the sciatic nerve.[1] Depending on the severity of the injury, the nerve exhibits high signal intensity on T2-weighted MRI[9–11]; moreover, MRI can detect the extent of the lesion and identify inflammatory etiology.[1] In this case, we confirmed inflammatory soft tissue swelling using MRI, which was helpful in diagnosing sciatic neuropathy and identifying its cause.

The currently known complications of pressure ulcers include cellulitis, osteomyelitis, bacteremia, endocarditis, meningitis, septic arthritis, abscesses, and squamous cell skin cancer, among others.[12] The present findings suggest that neuropathy can also occur as a complication of pressure ulcers.

Table 1

Motor nerve conduction studies.

	Nerve	Segment	Latency, ms	Ampulitude, mV	CV, m/s	F-wave, ms
Right	Peroneal	Ankle – knee/EDB	*NE			
		ankle – knee/TA	3.2–7.2	*0.1	*30.2	
	Tibial	Ankle – knee/AH	*NE			
Left	Peroneal	Ankle – knee/EDB	4.8–11.2	*1.3	41.0	37.9
	Tibial	Ankle – knee/AH	3.1–9.5	20.9	51.5	39.1

The compound muscle action potentials (CMAPs) in the right peroneal [tibialis anterior (TA) recording] and left peroneal nerves exhibited a decreased amplitude. A decreased conduction velocity was found for the CMAPs in the right peroneal (TA recording) nerves. CMAPs were not evoked in the right peroneal [extensor digitorum brevis (EDB) recording] and tibial nerves.
AH = abductor hallucis, CV = conduction velocity, EDB = extensor digitorum brevis, TA = tibialis anterior.

Table 2

Sensory nerve conduction studies.

	Nerve	Segment	DL, ms	Amplitude, μV	
Right	Superficial peroneal	Leg/ankle	*4.4	*2.8	Distance: 14 cm
	Sural	Midcalf/ankle	*5.6	*5.0	Distance: 14 cm
Left	Superficial peroneal	Leg/ankle	2.9	*6.8	Distance: 14 cm
	Sural	Midcalf/ankle	2.9	*5.9	Distance: 14 cm

The sensory nerve action potentials (SNAPs) in the right superficial peroneal and sural nerves exhibited decreased peak latency values. The SNAPs in all examined nerves exhibited decreased amplitudes.
DL = distal latency, NE = not evoked.
* Abnormal finding.

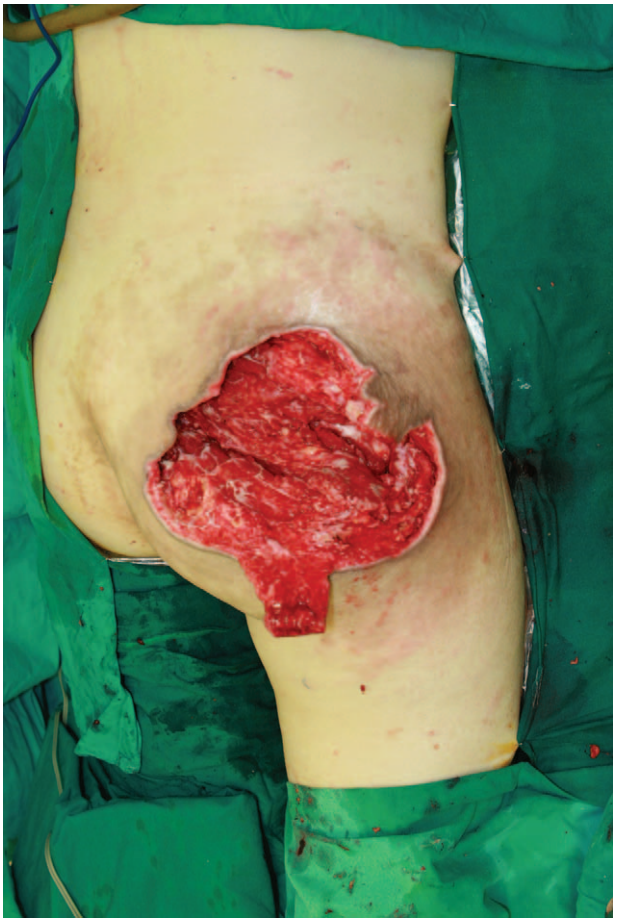

Figure 4. To treat sciatic neuropathy caused by inflammatory soft tissue swelling, debridement was performed on the necrotic tissue and decompression was performed on the sciatic nerve.

Khaykin et al[13] noted that patients with schizophrenia have an increased risk of adverse events including decubitus ulcer and it may be related to medication resulting in oversedation and decreased mobility. Gefen [14] noted that pressure ulcers in subdermal tissues under bony prominences very likely occur approximately between the first hour and 4 to 6 hours after sustained loading. In our case, the patient overdosed on the schizophrenia medication and was bedridden for 2 days in a coma. It is sufficient to cause the pressure ulcer.

This patient ultimately should have undergone flap surgery to cover the soft tissue defects. However, in general, if a patient's risk factors remained, operative reconstruction must be delayed. Furthermore, if there is a high possibility of a surgery failing or ulcer recurrence, chronic wound care is more effective. In the case study presented here, because of uncontrolled moods or refusal of psychiatric treatment, we anticipated her poor cooperation and prolonged care postoperatively. Moreover, toxic hepatitis occurred during psychopharmacological treatment. The patient was susceptible to hepatic failure, and liver transplantation was a possibility. As a result, we could not perform flap surgery under general anesthesia; instead, the patient received conservative treatment while her general condition improved. Some examples of conservative treatment include debridement, frequent dressing changes, and irrigation of deep areas of the ulcer.[12,15,16]

There are several limitations to our study. First, follow-up nerve conduction studies after 6 months do not exist. Those data provide a better idea of the treatment method and the expected duration of treatment. Second, EMG is helpful to distinguish between sciatic neuropathy and peroneal mononeuropathy, but we could not perform needle EMG because of the high risk of infection. And third, further studies and long-term data are needed to confirm our finding due to small sample size.

4. Conclusion

Because pressure ulcers most commonly occur in patients with paraplegia or quadriplegia, physicians are generally less interested in sensory and motor disorders in the lower extremity caused by pressure ulcers. However, as this case reveals, the possibility of sciatic neuropathy occurring due to a pressure ulcer should be considered when treating pressure ulcer patients who do not have paraplegia or quadriplegia. Sciatic neuropathy should be prevented by aggressive pressure ulcer treatment and, if neurological symptoms occur, an accurate diagnosis of sciatic neuropathy should be made based on a physical examination, nerve conduction studies, and imaging studies to provide the appropriate treatment.

Author contributions

Conceptualization: Jae Ha Hwang.
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Formal analysis: Dong Wan Kim.
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Supervision: Kwang Seog Kim, Sam Yong Lee.
Writing – original draft: Jae Ha Hwang.
Writing – review & editing: Jae Ha Hwang.

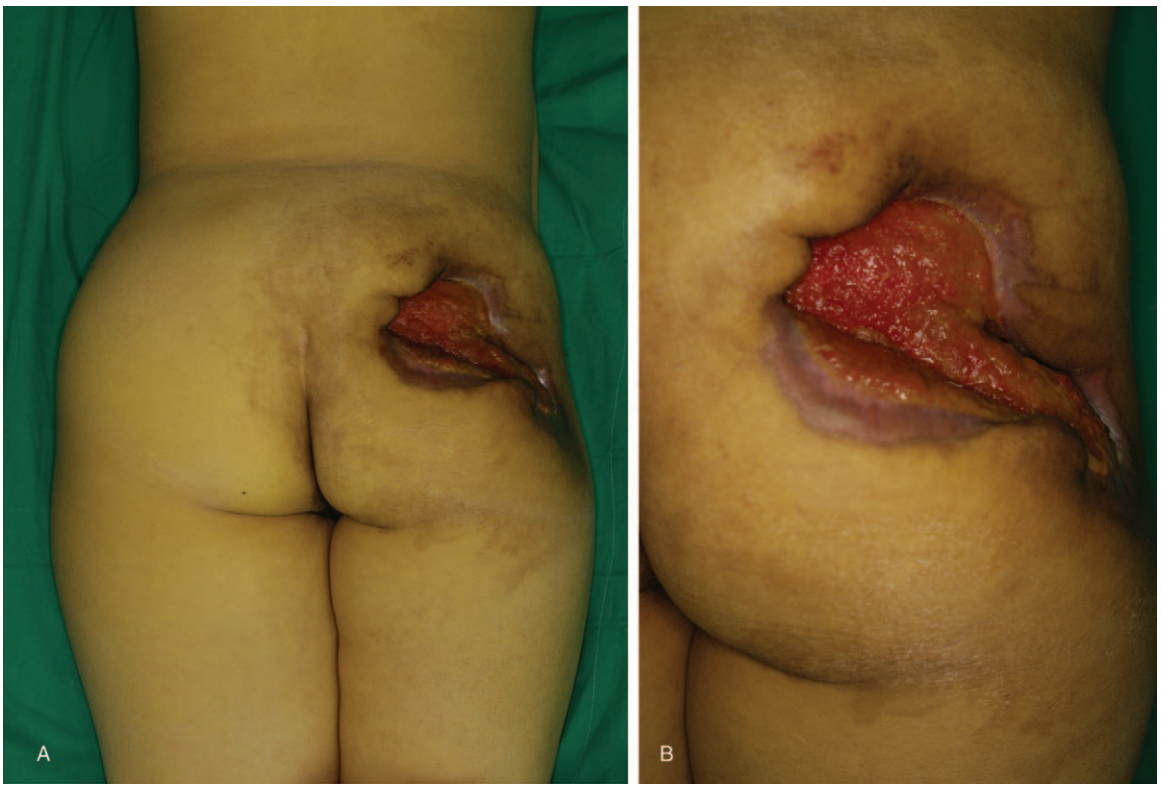

Figure 5. Photographs of the right buttock 3 months after the initial visit (A, B). The lesion exhibited moderate improvement after frequent dressing changes.

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