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Pressure sore as a possible complication of lower central neuraxial blockade

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Introduction

Central neuraxial blockade, such as epidural and spinal anesthesia, is widely used because of its good postoperative course [1]. In central neuraxial blockade, although complications such as respiratory depression, hypotension, motor blocks, and urinary retention are commonly known to occur, pressure sores are not well known but are believed to occur frequently in high-risk patients [2] and to appear on the heel pads [3–5]. We report two young, healthy patients who underwent gynecological surgery with lower central neuraxial blockade and developed pressure sores on the sacral skin during the postoperative period.

Case report

Case 1

A 19-year-old woman, weight 58 kg and height 158 cm, was scheduled for ovarian adnexectomy and omentumectomy for her ovarian tumor. She was otherwise fit and active, except for anemia (hemoglobin 10.6 mg·dl^{-1}). Anesthesia consisted of a combination of general and epidural anesthesia. A lower thoracic (Th11–12) epidural catheter was inserted, and 10 ml of 1% mepivacaine was injected. General anesthesia was induced by intravenous administration of thiopental and vecuronium. General anesthesia was maintained with 0.8% to 2% sevoflurane, nitrous oxide and oxygen (FiO_2 = 0.33), and paralysis with vecuronium. Twenty minutes after the first epidural bolus injection, an epidural infusion of plain 1% mepivacaine was started at a rate of 6 ml·h^{-1}. Throughout the operation, the patient was in the lithotomy position. To prevent pressure sores, the operating table was covered with silicon jelly pads. The operative area was sterilized with 0.2% chlorhexidine gluconate.

Surgery lasted about 4 h and 30 min. The patient was in the lithotomy position for 5 h. During surgery her systolic blood pressure varied between 90 and 110 mmHg, and her general condition was stable. The bladder temperature did not decrease below 36°C. The total blood loss was 320 ml, and we did not give a blood transfusion. At the end of surgery, we checked the condition of the whole body surface of the patient. Although the disinfectant, chlorhexidine gluconate, flowed along her sides a little, there was no change in her sacral skin. Postoperatively, an epidural infusion was started with 0.25% bupivacaine 2 ml·h^{-1} and fentanyl 15 μg·h^{-1}. The patient could move her legs when the continuous epidural infusion was started. She remained hemodynamically stable throughout the first postoperative night. The morning after surgery, 24 hr after the beginning of surgery, a large erythema was discovered on her sacral skin. The erythema was 6 × 4 cm in size and was tinged with violet (Fig. 1). There were no skin lesions on her body except for the sacral area. The patient did not complain of pain on her sacral skin. A dermatologist confirmed the diagnosis of pressure sore. We informed her about the sacral skin lesion, and she consented to treatment with ointments containing disinfectant and anti-decubitus ulcer drugs. All of the erythema had completely disappeared uneventfully by the fourth postoperative week.

Case 2

A 28-year-old woman, weight 48 kg and height 162 cm, had a uterine myoma. She was otherwise fit and active.

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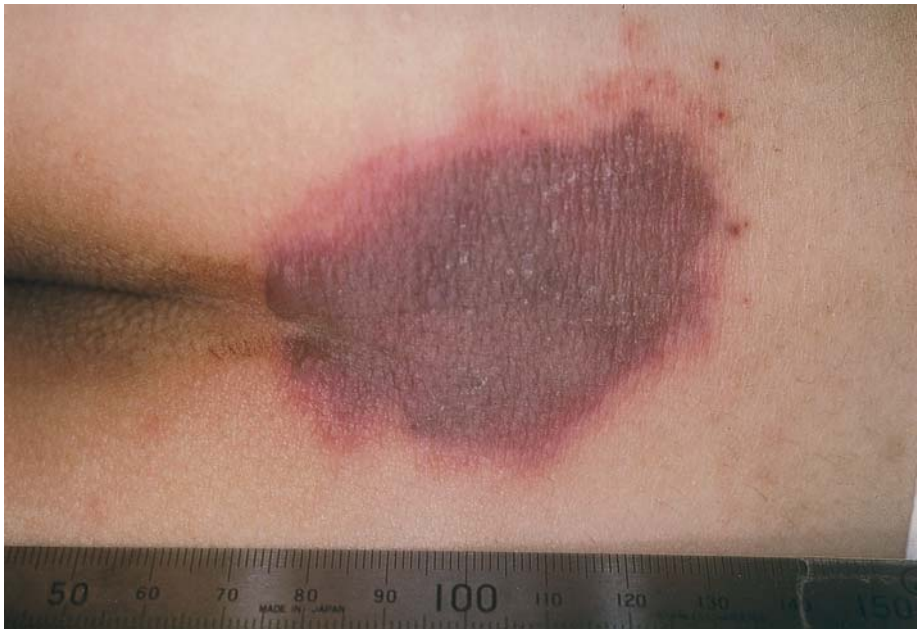

Fig. 1. Pressure sore on the sacral skin in case 1. The photograph was taken the morning after surgery, 24 h after the start of surgery. The right side is rostral and the left side caudal

She was scheduled for a myomectomy. Anesthesia consisted of spinal anesthesia through L3–4. Hyperbaric tetracaine 2.5 ml was injected into the subarachnoid space. The level of spinal anesthesia was above Th8 and high enough to allow operation. Throughout the operation the patient was in the lithotomy position. To prevent pressure sores, the operating table was covered with silicon jelly pads. The operative area was sterilized with 0.2% chlorhexidine gluconate.

The surgery lasted for 1 h and 40 min. The patient was in the lithotomy position for 2 hours. During surgery her systolic blood pressure varied between 110 and 140 mmHg and her general condition was stable.

Immediately after the operation, a member of the medical staff noticed erythema on her sacral skin. There was no disinfectant (chlorhexidine gluconate) on her erythema. A dermatologist confirmed the diagnosis of pressure sore. She complained of abdominal pain but not of sacral pain. We informed her about the sacral skin lesion, and she consented to treatment for it. Because she took little interest in the sacral skin lesion, we interviewed her in detail. Our interview revealed that she had experienced similar erythema after horseback riding for a short time. Therefore, she was not surprised at her sacral erythema. Treatment consisted of ointments containing disinfectant and anti-decubitus ulcer drugs. These treatments had effective. All of the sore had completely disappeared uneventfully by the third postoperative week.

Discussion

A pressure sore is a localized area of tissue necrosis that tends to occur when soft tissue is compressed between a bony prominence and an external surface for a prolonged period [6]. The National Pressure Ulcer Advisory Panel proposed the following staging definitions of pressure sores [6]: Stage I, nonblanchable erythema of intact skin, considered the heralding lesion of skin ulceration. Stage II, partial-thickness skin loss that involves the epidermis or dermis (or both). The ulcer is superficial and manifests clinically as an abrasion, blister, or shallow crater. Stage III, full-thickness skin loss and damage or necrosis of subcutaneous tissue that may extend to, but not through, the underlying fascia. The ulcer manifests clinically as a deep crater, with or without undermining of adjacent tissue. Stage IV, full-thickness skin loss associated with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures, such as tendons or joint capsules.

In our report, the following points were common to both patients: erythemas were diagnosed as pressure sores (stage I) by dermatologists; the operations were gynecologic procedures in the lower abdomen; lower central neuraxial blockade (spinal anesthesia or lower thoracic epidural anesthesia) was used; and there were no major preoperative complications.

Pressure sore is not widely indicated as a complication of central neuraxial blockade. There have been a few reports of pressure sores as a complication of anesthesia, most of them on the heel pads [3–5]. The erythema occurred on the sacral skin in our patients, and there may be some question whether the erythemas

were really pressure sores. The erythemas, however, were diagnosed by dermatologists as pressure sores, and we were confident that they were not simple burns due to the electrocauterizer or chemical burns from the disinfectant.

Pressure sores are usually seen in patients who have been lying in bed for a long time. Interestingly, pressure sores developing under anesthesia were not reported in our hospital when anesthesia consisted of a combination of general anesthesia and cervical/upper thoracic epidural anesthesia or simply general anesthesia, even during cardiac surgery or neurosurgery, which generally lasts longer. Therefore, we considered the possibility that lower central neuraxial blockade was a factor.

Mein et al. reported the relationship between skin temperature response and pressure load under spinal anesthesia [7]. According to their report, it was commonly accepted that central neural blockade improved regional blood flow; however, the recovery of response after removal of the pressure load was significantly delayed under spinal anesthesia with respect to skin temperature. They speculated that decubitus ulcer would be easily formed under spinal anesthesia.

Central nerve blockade blocks sympathetic nerves and results in distension of the vascular and lymphatic systems. Although central nervous blockade has been shown both theoretically and experimentally to improve regional blood flow, it is not clear whether regional blood flow changes against pressure load under spinal anesthesia. If change in skin temperature is used as an index of change in cutaneous blood flow, the region under central nervous blockade may suffer from low blood flow.

However, pressure sores do not occur in all cases in which lower central neuraxial blockade is used. The risk of pressure sores increases in elderly, unconscious, emaciated, paralyzed, and bedridden patients [8–10]. We do not have a definitive answer yet, and further study is needed to elucidate the relationship between pressure load and regional blood flow. Interestingly, a detailed postoperative interview revealed that patient 2 had experienced similar erythema after horseback riding for a short time. There are no reports discussing the relationships between pressure sores and constitution. However, there may be patients in whom pressure sores easily develop because of their constitution. It is unclear whether the addition of fentanyl to the postoperative epidural infusion contributed to the development of the pressure sore in case 1 [3]. There are some reports of pressure sores in patients under lower central neuraxial blockade [3–5,11,12]. Although all the patients in these reports were in the supine position, the patients in our study were in the lithotomy position, which may account for the difference in where the pressure sores occurred.

The cutaneous condition should be carefully observed during the postoperative period, even in young, healthy patients. The best way to prevent pressure sores is to be alert to their development [13]. During postoperative rounds the presence of pressure sores should be checked for thoroughly, including on the sacral skin, especially in cases in which lower central neuraxial blockade has been used.

In conclusion, we reported two patients with pressure sores on the sacral skin, which are thought to have resulted from lower central neuraxial blockade.

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