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Case Report

Extravasation Injury and Pressure Sore in Brain Damage Patient with Stiffness of the Limbs

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Extravasation injury refers to leakage of corrosive liquids from veins, resulting in tissue damage. The authors report on a case of extravasation injury to the left hand after administration of fluid to the antecubital area in a patient with brain damage. In order to minimize the effects of extravasation injury, rapid diagnosis and management are needed. In patients with stiffness, pressure sores can develop requiring more careful management by the medical staff.

Key Words: Extravasation of diagnostic and therapeutic materials, Vegetative state, Vancomycin, Hand, Pressure ulcer

Extravasation injury refers to the cell injury and tissue destruction caused by the corrosive fluids drained from the vein to the tissues. Its severity is decided by the type, amount, and duration of exposure of the cells and tissue to the fluids. Extravasation can usually be detected early due to its clinical manifestations that include pain, and favorable prognoses have been reported with the use of detoxifying agents.[1] When extravasation is treated in its early stage, its symptoms other than the local swelling are alleviated in a short period. In most patients, adjuvant therapies such as elevation of the lesion and cold or hot compress application are effective for dissipating the fluids. In infant cases, the tissue damage caused by the extravasation of electrolytes or anticancer agents is more serious than that in adult cases due to the thinner diameter of the blood vessels.[2] In the treatment and evaluation of the extravasation injury, pathophysiologic classification is useful, and the mechanisms of the cell-level osmotic destruction, secondary ischemia caused by circulatory disorder, primary cytotoxicity, mechanical pressure, and infection are considered.[3,4]

In patients with brain damage and paralysis or spasticity in their extremities, the detection of extravasation may be challenging, so the administration of the fluids or drugs into the spastic or paralyzed peripheral veins in the extremities requires attention. Despite the importance of this topic, few studies on the extravasation caused by brain damage or spinal cord injury have been reported.

Reported herein is a case of hand damage and finger necrosis in a 49-year-old male patient who had brain damage and spasticity, and who suffered from extravasation caused by injections of Vancomycin and a high volume of fluids due to his pneumonia while he was in a long-term-care hospital.

CASE REPORT

The subject patient underwent neurosurgery a year earlier due to acute extradural hemorrhage and traumatic subarachnoid hemorrhage after a motor vehicle accident. Then he was transferred to a long-term-care hospital due to his vegetative state. While he was injected with a fluid

for pneumonia in the long-term-care hospital, the central vein catheter was blocked, so the fluid and Vancomycin were administered via his forearm. Immediately after the injection, an intermediate or higher level of edema developed in the injection site, and cyanosis occurred on his left hand before he was transferred to the authors' hospital (Fig. 1). Based on the findings a day after the injury, which were obtained upon the arrival of the subject at the authors' hospital, the subject was diagnosed with extravasation injury and related compartment syndrome, so hyaluronidase was hypodermically injected. In the blood tests, there were no specific findings other than a slight increase in the white blood cell count. Due to the spasticity, all the patient's fingers were flexed, and the condition of the third finger was particularly severe. For early treatment, the affected arm was elevated, and a wet dressing with normal saline was applied at least twice a day. To alleviate the hand and finger spasticity, gauze pads were applied while avoiding compression on the areas. The symptoms of edema and distal cyanosis improved over the next five days, but the left third finger showed findings of necrosis in the telomeric site (Fig. 2). The edema in the neighboring fingers improved and de-epithelialization occurred. The partial necrosis in the skin was treated with a dressing, but the necrosis in the third finger deteriorated to show a clear boundary in the mid-hand area after a month. Escharectomy was not performed. Five weeks after the injury, the third finger was amputated at the proximal interphalangeal joint area under partial anesthesia (Fig. 3). Ten days post-operatively, the suture was removed and the wound showed signs of recovery. During the three-month follow-up period, there were no specific findings.

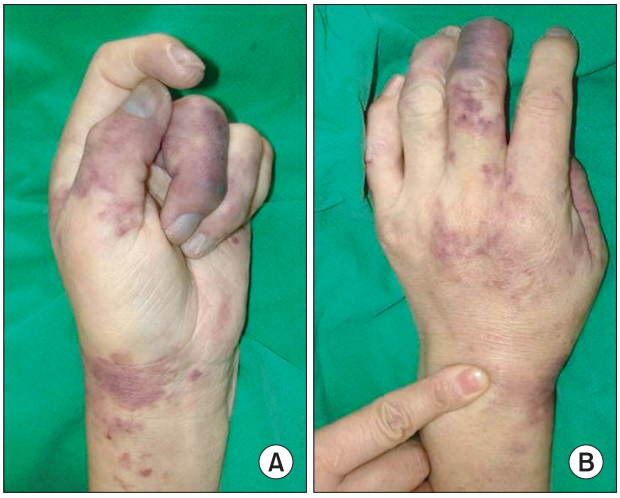

Fig. 1. Preoperative photographic findings show a 49-year-old man with a extravasation injury after one day. (A) Higher level of edema with multifocal ecchymosis was observed on left hand. (B) Edema and cyanosis was dominant on the third finger.

DISCUSSION

In accordance with the developments with potent agents and with the increase in the use of conservative chemotherapies and infusion pumps, the risk of toxic

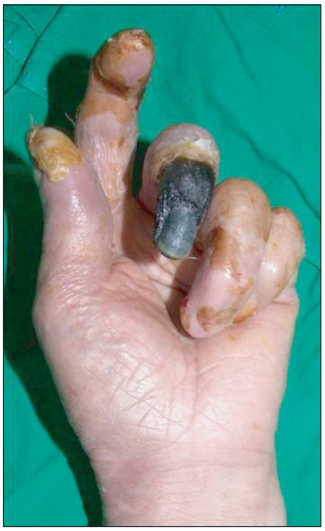

Fig. 2. Preoperative photographic findings show skin necrosis on 3rd finger.

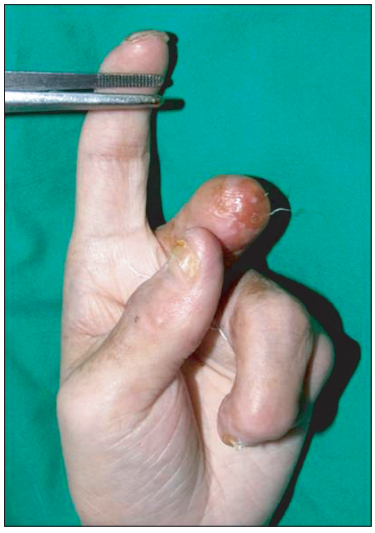

Fig. 3. Photograph shows postoperative finding after 3 months.

materials leakage out of the vein into the surrounding tissues is increasing.[5]

In all extravasation cases, edema develops regardless of the type of material that leaked; but when a normal saline or glucose solution leaks, the edema can be removed through adjuvant therapy unless a secondary infection develops.[3] However, when the tissue is damaged by anticancer agents or electrolytes, the boundary of the affected tissues becomes evident within 5 to 7 days, after which a crust is formed. Without secondary bacterial infection, the crust remains dry for several months or longer.[1]

The mechanism of the extravasation that causes tissue injury is not clearly understood yet, but the following five factors are considered to be involved in it:[2] the osmotic injury caused by the hypertonic solution that contains calcium ions, potassium ions and urea; the secondary ischemic injury caused by a circulatory disorder; and the direct injury on the cells caused by toxic materials such as anticancer agents. Mechanical compression and infection were also considered factors.[3-7]

To prevent skin necrosis caused by extravasation, medication, debridement and irrigation of the affected area have been suggested. In addition to the general and preventive measures, Lee et al.[3] suggested early administration of an appropriate volume of hyaluronidase solution to reduce the tissue injury caused by the calcium gluconate extravasation, using the effects of diffusion, dilution and cell preservation. Hyaluronidase is used to treat the extravasation of nafcillin, 12% dextrose, bicarbonate and aminophylline. Vincristine and vinblastine are known to be effective for the skin necrosis caused by the extravasation of calcium chloride and hyperalimentation fluid. In this study, hyaluronidase was used for three days, but the skin necrosis in the third finger was not prevented. This might have been due to the too small dose or to the abnormal blood circulation in the spasticity patient.

According to Seul et al.,[4] hydrocortisone succinate inhibited tissue injuries caused by adriamycin, which is a representative anticancer agent; and the earlier the administration was, the better the prevention of the tissue necrosis was. Adrenocortical agents are known to facilitate decorin formation, which in turn inhibits the formation of type 1 collagen to form a loose collagen matrix.[8] As shown, adrenocortical agents inhibit tissue necrosis caused by the extravasation of anticancer agents due to their anti-inflammatory effects and their facilitation of drug diffusion and absorption. However, in this study, steroid agents were not used because the subject patient had symptoms of infection due to pneumonia.

Spasticity develops when the upper motor neurons that control the spinal cord for the movement of the extremities are injured and their inhibitory effects decrease. Spasticity can prevent the development of complications such as muscular atrophy, osteoporosis and thrombosis, but it can harden articulation and cause pain. In this study, the patient had spasticity after he experienced brain damage; and when he was injected with antibiotics, a type of compartment syndrome developed due to the intermediate level of the extravasation injury. In this study, despite the administration of the appropriate treatment for extravasation injury, finger necrosis developed because of the micro-circulation disorder caused by the spasticity. The third-finger necrosis in the subject might have occurred because the finger was long enough to induce a compression ulcer in the palmar area, the third-finger spasticity might have caused poor blood circulation; and the third finger being the longest and the thickest among the fingers, it might have caused ischemia. In patients with hand or finger spasticity, their pain perception capacity is poor, so attention is required in venous injection. It is recommended that the injection be made on the arm or leg without spasticity; and if the injection in the affected extremities is inevitable, the proximal area rather than the telomeric site should be selected to minimize the risk of micro-circulation disorder developing. In cases of finger spasticity, there is a risk of compression ulcer, so careful attention may be needed.

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