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Epidural Hematoma Associated with Epidural Anesthesia: Complications of Anticoagulant Therapy

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A recent report[1] described the complication of epidural hematoma associated with epidural anesthesia in two patients. In the first case, the patient received an infusion of urokinase postoperatively. In the second case, the patient received a continuous heparin infusion of 800 U/h. In both cases, the patients complained of back pain postoperatively, and paraplegia developed. The diagnosis of epidural hematoma was made and emergency decompressive laminectomy was performed. Both patients had a complete neurologic recovery. In the following case paraplegia developed in a patient in whom epidural anesthesia and an intraarterial infusion of urokinase were administered intraoperatively, and a continuous infusion of heparin was administered postoperatively. The patient did not recover from his paraplegia.

CASE REPORT

An ASA physical status 3, 69-yr-old man presented with occlusion of the left superficial femoral artery and was scheduled for a left femoral-popliteal artery bypass. His chief complaint was that he could not walk because of leg pain. Physical exam revealed ischemic ulcers of the left heel and first toe. The patient's significant medical history included chronic atrial fibrillation and insulin-dependent diabetes mellitus. Preoperative medications included digoxin, Dyazide, and NPH insulin. The PT was 11.9 s (normal range 10.5–14.5 s) and aPTT was 28.2 s (normal range 25.0–39.0 s). Platelet count was 295,000/mm^3.

The patient was taken to the operating room and placed in the sitting position. Using an 18-G Hustead needle the epidural space was identified atraumatically at the L3–4 interspace using a loss of resistance technique. The catheter was easily threaded to a distance of 12 cm at the skin. Blood was noted in the epidural catheter, however, and the catheter was removed. The procedure was repeated at the L2–3 interspace and the catheter was again threaded without difficulty. Aspiration of the catheter yielded no blood. Anesthesia was induced with 15 ml 2% lidocaine and surgery proceeded. (When additional boluses of bupivacaine were administered later, aspiration of the catheter still yielded no blood.) Ninety minutes later a bolus of 6,300 U heparin was given intravenously at the request of the surgeon. Neither ACT nor aPTT was checked after the heparin was given. The patient was a participant in a blinded study to test the efficacy of intraarterial urokinase. During the procedure the surgeon administered a single bolus of the study drug intraarterially. (When the study code was later broken, this was found to be urokinase.) Five and one-half hours after the epidural catheter was inserted, an intravenous infusion of heparin of 1,000 U/h was begun. The surgery was completed 90 min later. The patient was taken to the PACU and the epidural catheter was removed.

On the first postoperative day, the patient was transferred to the surgical intensive care unit. The ICU nurse and the resident noted that the patient had full movement of both legs. Later that day, the patient was transferred to the floor, still receiving the heparin infusion. The aPTT increased from 74 s to 91 s. On the second postoperative day, the surgeons noted a large hematoma of the left groin. The heparin infusion was discontinued and the patient was scheduled for surgery the next day to drain the hematoma. The next (third postoperative) day the surgery was cancelled because the hematoma was resolving. The aPTT had decreased to 32.5 s. On the fourth postoperative day, when attempting to get the patient out of bed and into a chair, it was noted that the patient could not move his legs. The anesthesia department was consulted. Neurologic exam revealed severely decreased strength in both legs and decreased sensation up to the T10 dermatome. The patient's mental status was impaired (a distinct worsening since the operation). Upon questioning regarding the weakness in his legs he replied that his legs had been weak for several years. He specifically denied any back pain. An MRI was performed, which revealed an epidural hematoma extending from T10–L2. The patient was taken to the operating room that evening and emergency decompressive laminectomy was performed.

Postoperatively there was no improvement in neurologic function over the next several weeks. No repeat diagnostic scans were performed. The patient subsequently expired from complications secondary to infection of the femoral artery graft and sepsis.

Discussion

There are multiple reports of epidural hematomas in the medical literature, some associated with epidural anesthesia,[2–6] some with anticoagulant therapy,[7–9] and some (spontaneous) with neither.[10,11] In most of the cases with complete neurologic recovery, the diagnosis has been made early, with the suspicion based on patients' complaints of back pain and lower extremity weakness. In this case, the patient never complained of either symptom, and because of orders for bed rest related to the surgery and the subsequent groin hematoma, the paraplegia was not recognized.

The physicians caring for such patients postoperatively must seek information regarding these symptoms, even when the patient does not spontaneously complain of such symptoms. It is uncertain what period of time exists for

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a danger of epidural hematoma to occur following epidural anesthesia, and often surgical patients are not seen by anesthesia residents or staff after the first or second postoperative day. Thus we should be certain that those caring for the patient, including both the nursing and surgical staff, are aware of the potential for an epidural hematoma to develop, even after normal movement and sensation return following epidural anesthesia.

The advisability of performing epidural anesthesia in patients in whom anticoagulation is to be used must be examined. New techniques in the management of peripheral vascular disease include infusion of urokinase and the continuous infusion of heparin. Three types of anticoagulant therapy were administered to this patient: an intraoperative injection of urokinase, an intraoperative bolus of 6,300 U heparin, and a continuous infusion of heparin, 1,000 U/h, postoperatively.

Heparin acts by activating anti-thrombin III and suppresses thrombin generation and fibrin formation and prevents the extension of existing thrombi. It does not hasten the resolution of an existing thrombus. Urokinase is an enzyme that activates plasminogen to plasmin and induces systemic fibrinolysis. It is capable of lysing a clot that has already formed. Although the half-life of urokinase is short (less than 30 min), the effects may be significant.

The effect of these drugs on the development of epidural hematomas has not been fully investigated. In 1981 Rao[12] performed a prospective study in 4,011 patients anticoagulated after insertion of an epidural catheter. In four patients, blood was freely aspirated following insertion of the needle into the epidural space. In these four patients, surgery was cancelled and rescheduled for the following day under general anesthesia. He reported no epidural hematomas leading to spinal cord compression. Odoom[13] reviewed 1,000 cases of epidural anesthesia for vascular surgery in patients receiving preoperative oral anticoagulation therapy and an intraoperative bolus and a continuous intraoperative infusion of heparin. He reported no neurologic complications related to epidural hematoma and concluded that epidural anesthesia could be safely used in patients receiving anticoagulant therapy. On the other hand, Kwitka[14] recently recommended that use of an epidural catheter should be avoided if postoperative anticoagulation with heparin is planned.

Regarding urokinase, significant hemorrhage has been reported in 45% of patients undergoing fibrinolytic therapy.[15] Contraindications to fibrinolytic therapy include active bleeding from an inaccessible site and recent surgery or biopsy in a relatively inaccessible area. Even if there is no active bleeding noted at the time the epidural catheter is inserted, subsequent bleeding in the epidural space will be difficult to recognize early, and requires surgery (decompressive laminectomy) for definitive treatment.

We would recommend the following:

1. If a continuous infusion of heparin is planned and if a traumatic tap occurs on the first attempt at placement of the catheter, the case should be cancelled and surgery postponed for 24 h, as per Rao's study (although Dickman reported a case of epidural hematoma associated with continuous infusion of heparin, with no evidence of a traumatic tap.)
2. If urokinase with heparin (single bolus or infusion) is to be used, we recommend that epidural anesthesia be avoided. Fibrinolytic therapy followed by heparin infusion also must be considered hazardous, since such a combination could lead to prolonged bleeding. Our patient had this combination as treatment.
3. Regarding use of epidural anesthesia in patients receiving fibrinolytic agents alone; the medical literature suggests that this is relatively contraindicated. Dangers of bleeding into a closed space are very high. Additionally, Dickman reported an epidural hematoma in a patient receiving urokinase therapy, without a traumatic tap. No formal study has been done to assess the safety of regional anesthesia in patients receiving fibrinolytic agents.

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Segmental Reflex Sympathetic Dystrophy Involving the Thumb: A Rare Complication of a Varicella Zoster Infection

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The reflex sympathetic dystrophy syndrome (RSDS) as a complication of varicella zoster was first described by Sudek in 1901.[1] In the past 90 yr a small number of reports have associated a varicella zoster infection as the precipitating factor in RSDS.[2–5]

The following case report describes a rare manifestation of segmental RSDS associated with a varicella zoster infection. To the author's knowledge, this is the first published paper of a segmental RSDS precipitated by varicella zoster infection.

CASE REPORT

The patient was an 84-year-old white woman who was referred to the pain clinic because of persistent burning pain in the left hand and forearm.

Approximately 5 months previously she had been treated for typical symptoms of varicella zoster involving C6, C7, and C8, on the left. The vesicles were noted along distribution of the left radial nerve of the arm and hand. She also had burning pain over the dorsum of the hand, lower arm, and upper arm in the area supplied by the radial nerve. Some pain was located in the nail bed of the thumb. Oral acyclovir, 800 mg, five times daily was prescribed for 7 days.

During the subsequent 3 weeks the vesicles and rash in the upper arm, forearm, dorsum of the hand, and the first three digits subsided without complicating bacterial infection. She continued to complain of mild burning pain and hyperalgesia in the sites of the healing lesions.

At this time, the patient's attention was focused on the insidious onset of an intense, burning pain in the left thumb. The persistent, progressively severe pain required hydrocodone for relief. Initially the pain affected the phalangeal and metacarpophalangeal joints. The periarticular pain was associated with swelling, redness, and stiffness in the joints. Function and mobility of the thumb became progressively limited. Concomitantly edema, cold and mechanical allodynia, and a cyanotic flush appeared. These symptoms affected the thumb to the level of the metacarpophalangeal joint on the dorsal surface and most of the thenar surface. She could not tolerate a loose-fitting glove because of the allodynia. A cold wind or immersion of the hand in cold water exacerbated the burning pain in the thumb.

The initial examination revealed an alert but depressed elderly woman in severe pain. She held her left arm in her right hand avoiding any contact with the examination table. She also complained bitterly of the intense, burning pain in the left thumb and resisted any passive movement of the hand. There was slight edema of the forefinger, however, the periarticular areas were not tender and mobility appeared normal. The third, fourth, and fifth fingers were mobile and had normal grip strength compared to the right hand. The thumb nail was large, thickened, cracked, and had irregular longitudinal ridges. She was unable to trim the nail because of the intense pain precipitated by the attempt. The skin of the thumb was thin, warm, moist, cyanotic, edematous, and smooth. An alcohol sponge placed on the thumb caused exacerbation of the pain. Light touch of the entire thumb surface produced intense pain. She could be induced to flex the thumb about 1 cm with considerable pain.

An x-ray film of the left hand revealed irregular periarticular patchy areas of osteoporosis and resorption of trabecular bone in the metaphysis of the proximal and distal phalanx of the thumb. The radiologist noted other areas of minimal osteoporosis expected at her age; however, the marked changes of the thumb bones "were consistent with a diagnosis of RSDS."

Thermography studies confirmed the vasodilation in the skin of the thumb. In general, the temperatures of the skin on the left hand ranged from 30.2° C to 32.0° C in contrast to the range of 28.4–29.3° C on the right.

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