Background and Objectives: A feared complication of spinal or epidural anesthesia is the development of epidural or spinal hematoma with subsequent neural element compression. Most available data are derived from the obstetric literature. Little is known about the frequency of hematoma occurrence among patients undergoing orthopedic joint arthroplasty, who are usually elderly and experience significant comorbidities. We sought to study the incidence of clinically significant lesions after spinal and epidural anesthesia and further describe their nature.

Methods: We retrospectively analyzed a database of all patients who underwent total hip or total knee arthroplasty under neuraxial anesthesia at our institution between January 2000 and October 2010. Patients with radiographically confirmed epidural lesions were identified and further analyzed.

Results: A total of 100,027 total knee and hip replacements under neuraxial anesthesia were performed at our institution. Ninety-seven patients were identified with findings of an epidural or gas collection (0.07/1000; 95% confidence interval, 0.77–1.16/1000). Eight patients were identified with findings of an epidural blood or gas collection (0.07/1000; 95% confidence interval, 0.02–0.13/1000). No patients receiving only spinal anesthesia were affected. All patients diagnosed with hematoma took at least 1 drug that potentially impaired coagulation (5 nonsteroidal anti-inflammatory agents, 1 a tricyclic antidepressant, and 1 an antiplatelet drug). No patient incurred persistent nerve damage.

Conclusions: The incidence of epidural/spinal complications found in this consecutive case series is relatively low but higher than previously reported in the nonobstetric population. Further research using large data sets could quantify the significance of some of the potentially contributing factors observed in this study.

An Analysis of the Safety of Epidural and Spinal Neuraxial Anesthesia in More Than 100,000 Consecutive Major Lower Extremity Joint Replacements

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Institutional Anticoagulation Protocol

All patients underwent a standardized anticoagulation protocol, which was developed by the Institutional Quality Assurance Committee on deep venous thrombosis/pulmonary embolism prophylaxis. These consensus guidelines were formulated after review of the literature by the committee. During presurgical assessment, all patients underwent a “risk stratification,” in which patients were classified as “high risk” or “low risk” for thromboembolic events based on our protocol. Comorbidities qualifying a patient as “high risk” were 65 years or older with at least 1 comorbidity (vascular, cardiac, cerebral, or peripheral disease); chronic lung disease; serum creatinine greater than 1.9 mg/dL; diabetes; systemic autoimmune disease; prolonged immobility; stroke/paralysis; prior deep vein thrombosis; family history of vein thromboembolism; cancer; chronic venous insufficiency; obesity (body mass index [BMI], ≥30 kg/m²); cardiac dysfunction; indwelling central venous catheter; nephrotic syndrome; hormonal replacement therapy; intake of estrogen, or selective estrogen receptor modulating drugs; smoking; thrombophilia/hypofibrinolysis or antiphospholipid antibody syndrome; protein S or C deficiency; or antithrombin III deficiency. All other patients were considered “low risk.”

Total Hip Arthroplasty

For unilateral THA, all surgeries were performed under hypotensive epidural anesthesia. For “low-risk” patients undergoing THA, either acetylsalicylic acid (ASA), warfarin, or low-molecular-weight heparin (LMWH) was administered postoperatively at the surgeon’s discretion. “High-risk” patients received warfarin or LMWH at the surgeon’s discretion. All patients underwent intermittent pneumatic compression (IPC) twice a day for 15 minutes if no contraindications were present.

Total Knee Arthroplasty

In unilateral TKA, “low-risk” patients undergoing epidural anesthesia received IPC plus ASA or warfarin or LMWH at the surgeon’s discretion. “High-risk” patients received IPC plus warfarin or LMWH. Postoperatively, pneumatic compressive devices and rapid physiotherapeutic mobilization were applied as tolerated by the individual.

Anesthesia Protocol

Spinal anesthesia alone or combined spinal/epidural anesthesia was performed according to standard practice. For combined spinal/epidural anesthesia, the epidural space was identified between L2 and L5, and at the chosen interspace, a 17-gauge Tuohy needle and the loss of resistance technique either to air or saline was used depending on the practitioners’ preference. Subsequently, a 27-gauge Whitacre needle was passed through the epidural cannula. For spinal anesthesia alone, a 27-gauge pencil-point spinal needle was inserted through an introducer cannula at a level between L2 and L5. After free flowing spinal fluid was obtained, an intrathecal dose of local anesthetic (either bupivacaine or mepivacaine) was injected. After removal of the spinal needle, a closed tip multiflource epidural catheter was threaded and aspirated before use to rule out intrathecal or intravascular placement.

Data Collection

All patients who underwent either TKA or THA under spinal or combined spinal/epidural anesthesia between January 2000 and October 2010 were included in this study. A database query was performed looking at all included patients, identifying those who had a spinal imaging study postoperatively to their joint replacement. Specifically, all patients who had a postoperative epidural hematoma or compressive gas collection confirmed by a musculoskeletal radiologist on either a computed tomographic scan and/or magnetic resonance imaging (MRI) were identified and included in our analysis. This cohort underwent a physical retrospective chart review. The patients’ hospital charts, office notes, and imaging studies were reviewed. Patients who underwent TKA or THA with general anesthesia were excluded from this study.

Associated factors for compressive lesion formation were identified via a comprehensive literature review and the presence and absence of individual factors was documented for each patient.

When patients were suspected of developing an epidural hematoma, a musculoskeletal radiologist quantified location, size, and extent of the blood and/or gas collection on the imaging studies. The patient’s hospital chart, office notes, and imaging studies were studied for any of the following potential risk factors: patient demographics, record of previous epidural and/or spinal anesthesia, coagulation disorder, liver disease, regular or excessive alcohol consumption, medications, severe degenerative disc disease, scoliosis, preoperative and postoperative platelet count, international normalized ratio (INR), intraoperative heparin (administered during hip arthroplasty; 1000 U of heparin after the insertion of the acetabular component and before kinking of the large vessels), duration of postoperative indwelling catheter, and length of hospitalization. Furthermore, we recorded the postoperative course as well as any sensory and motor deficit according to the muscle group and Medical Research Council scale. The day of postoperative diagnosis was recorded and the management strategies reviewed.
at the time of catheter removal was within reference range according to our institutional standard value (INR of 1.8–2.5) in all cases. All patients had either unilateral or bilateral motor deficits of the lower extremity on examination. The presenting complaint in 4/7 patients was unrelenting, nonpositional, acute onset back pain. The other 3/7 presented with major neurologic motor deficits as their primary complaint. No patients experienced bowel or bladder dysfunction. Two of the 3 patients presenting with severe sensory-motor deficits required emergent surgical decompression of the spinal canal. Both patients were diagnosed with epidural hematoma and regained motor function after surgical intervention. The average hospitalization of all patients was 14.5 days (±11.9 days).

Upon analysis of the location and extent of each epidural gas and fluid collection, it seemed that the gas collections were more cranially located than the fluid collections. The fluid collections seemed to be evenly distributed around the level L1–L2. In general, the extension of collection did not vary between fluid or gas collection. Detailed analysis of location of the epidural and spinal hematoma and/or gas collection is displayed in Figure 1.

**Possible Contributing Factors**

Of the 7 reviewed patients, only 1 was classified as “low risk” for thromboembolic events by the risk stratification model of the Institutional Quality Assurance Committee, the other 6 as “high risk.” Four patients had undergone prior spinal and/or epidural anesthesia without any complications. All patients who experienced a hematoma took at least 1 drug that increased the likelihood for bleeding (5 were taking nonsteroidal anti-inflammatory agents, 1 a tricyclic antidepressant, and 1 an antiplatelet drug). Additionally, 1 patient had hereditary thrombocytopenia and 2 admitted to regular excessive alcohol consumption. The platelet count was reduced in 1 patient on the day of surgery and on the day of removal of the epidural catheter (70,000/μL), respectively. All patients with epidural hematoma had preoperative hypertension. Scoliosis, possibly causing difficulties during needle/catheter placement, was found to be present in 5 patients. Table 1 summarizes other potential contributing factors.

**Epidemiology**

This is the largest study of post-TKA and THA patients to review the consequences of various anticoagulation strategies used with concomitant spinal and/or epidural anesthesia. Of all of the patients undergoing spinal-only anesthesia (without indwelling epidural catheter), there were no instances of symptomatic epidural hematoma and/or compressive gas (n = 37,171). Among patients undergoing combined spinal and epidural anesthesia with indwelling epidural catheter, there were 8 identified symptomatic epidural gas collections and/or hematomas identified, yielding an occurrence rate of 1:7,857. Two of the 8 patients identified had severe neurologic deficits requiring emergency spinal decompression. There were no long-term sequelae in any of the cases.

After extensive review of office charts, hospital medical records, and imaging studies, we analyzed factors that have been associated with the formation of an epidural lesion as previously reported in the literature, including prior regional anesthesia, thrombocytopenia, hypertension, alcohol use, use of antiplatelet drugs, anticoagulation drugs, and the presence of scoliosis. Interestingly, the cause of neurologic symptoms in 3 patients was attributable to the presence of epidural gas collections only.

**Antiplatelet and Anticoagulation Drugs**

All 4 epidural hematoma patients were taking a variety of antiplatelet drugs in the preoperative period (Table 1). According to institutional guidelines, non-ASA antiplatelet therapies should be stopped 7 days before surgery and regional anesthesia. In accordance with American Society of Regional Anesthesia guidelines, cardioprotective ASA should not discontinued in the preoperative period. Furthermore, for spinal-epidural anesthesia, a platelet count above 80,000/μL has been regarded as relatively safe by the Hospital for Special Surgery Quality Assurance committee. In our study, 1 of 4 patients had an abnormally low platelet count on the day of surgery and the day of removal of the catheter (patient 6 in Table 1, 70,000/μL). However, epidural catheterization and catheter removal in this patient despite of contraindications was done after careful consideration of risks and benefits and does not represent common practice at this institution. All patients in the epidural hematoma group were taking ASA. Chronic ASA administration is known to increase bleeding risk but the benefit may outweigh this small risk. Other drugs that have been shown to interact with platelet aggregation and were found in our study population included tricyclic antidepressants (nortriptyline) (1 of 4), nonsteroidal anti-inflammatory drugs (ibuprofen) (2 of 4) and clopidogrel (1 of 4). The patient taking the latter discontinued clopidogrel only 4 days before surgery as opposed to the recommended period of 5 to 7 days. Additionally, venous thrombotic and pulmonary embolism prophylaxis started immediately after surgery could further increase the risk of epidural hematoma (details depicted in Table 1).

Published studies have suggested an increased risk of epidural hematoma development after epidural anesthesia with concomitant administration of LMWH. Conversely, Horlocker et al. found that preoperative antiplatelet therapy does not correlate with an increased risk of neurologic deficits due to spinal hematoma development; however, the authors advocate great caution with the use of any combination of anticoagulant. In our cohort, all epidural hematoma patients underwent postoperative anticoagulation with warfarin but also had a history of previous antiplatelet drug exposure. Furthermore, it is worth mentioning that no ethnic correlation was found among those patients who received warfarin and developed an epidural hematoma. No patients with hematoma received LMWH; however, reports of spontaneous spinal-epidural hematoma have been published.

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TABLE 1. A Depiction of Each Patient’s Demographics, Possible Contributing Factors, Diagnosis, Treatment, and Hospitalization

<table>
<thead>
<tr>
<th>Patients</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
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<td>Primary THR</td>
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<td>Epidural hematoma collection</td>
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</tbody>
</table>

ASA indicates acetylsalicylic acid; NSAIDs, nonsteroidal anti-inflammatory drugs; TCAs, tricyclic antidepressants.

Thrombocytopenia

Similar in mechanism to antiplatelet therapies, an acquired deficiency in platelets may increase the risk of bleeding. Patient 6 carried the diagnosis of idiopathic thrombocytopenia, an autoimmune disease in which patients produce antiplatelet antibodies, lowering platelet counts and increasing bleeding time. It seems plausible that this patient population may be at increased risk for epidural hematoma development.23

Hypertension

All patients included in this study with spinal compressive lesion carried the diagnosis of hypertension. This increased risk may be a result of the increased pressure in the epidural vessels. A recent case series on spontaneous epidural hematoma formation identified hypertension as a risk factor.24 This association can be especially meaningful in light of the high prevalence of this condition. Although approximately one third of the general United States population carries a diagnosis of hypertension, orthopedic patients are affected significantly more frequently, with rates exceeding 50%.25,26

Epidural Gas Collection

Three patients had an epidural gas collection and 1 patient had a combined hematoma and gas collection. Of those 4 patients, all had degenerative spinal conditions, which might predispose the patient to a higher risk of epidural gas collection and development of symptoms. Five patients were found to have scoliosis. Although speculative, it is possible that anesthesiologists might find it more challenging to identify the epidural space under those conditions, leading to repeated attempts and injection of air using the loss of resistance technique. Accordingly, one of the major risk factors identified by Horlocker et al4 for spinal hematoma was a technically difficult needle placement. We believe accurate documentation of the procedure in the anesthesia records of the patients could be essential to identify patients with an increased risk of compressive spinal lesions (ie, number of needle passes). Alternatively, very small volumes of epidural gas may collect from the tubing during infusion via a pain pump postoperatively.

Treatment

All patients presented with either unilateral or bilateral lower extremity motor deficits; 3/7 presented with severe deficits (less than 3/5 motor strength in a major motor group) and 4/7 presented with relentless, nonpositional new onset back pain. Of the 3 patients who presented with severe deficits, 1 patient began to improve spontaneously after imaging and reexamination. Two of 7 patients had persistent or worsening deficits and required urgent decompression and evacuation of hematoma from the spinal canal. The other 5 patients were carefully observed. All patients had complete resolution of pain symptoms and deficits by the 6-week postoperative visit. Timing for surgical intervention is highly critical and in case of such rare events, we suggest that a multidisciplinary team, consisting of the surgeon, a radiologist, and neurologist should make treatment decisions jointly. As our study population shows, not all patients have neurologic deficits. However, in case of motor function loss, surgical intervention is recommended.

Limitations

There are several limitations to this study. All possible contributing factors were not statistically analyzed. After the
incidence of this complication was established, a multidisciplinary board was formed to establish contributing risk factors. These risk factors were analyzed in those patients with spinal lesions only and found in a relatively high percentage and discussed with the current literature in mind. Due to the large study population, collection of all contributing factors in all patients was not possible. Therefore, all mentioned factors must be regarded only as possible contributors. As a next step, power analysis should be performed and each contributing factor evaluated prospectively in registries.

Another limitation is that this study lacks a cohort for spontaneous hematoma in patients undergoing total joint replacement with the same anticoagulation protocols and general anesthesia. A control group within our patient population was not analyzed, as the search strategy consisted of identifying only those with the complication.

Although our warfarin-based anticoagulation protocol has not changed significantly during the past decade, the analysis of contributing factors such as dosage or timing of catheter removal may have been warranted.

In conclusion, the incidence of epidural/spinal complications found in this consecutive case series is relatively low but higher than previously reported. Regional spinal anesthesia protocols combined with contemporary arthroplasty anticoagulation strategies have greatly reduced the morbidity of surgery and improved patient outcomes and safety, and thus the risks must be weighed against benefits afforded by this approach. Increased attention to the possible contributing factors highlighted in this article should further alert medical care teams to the possibility of an increase in risk, and the presence of an epidural compressive lesion when encountering a THA or TKA postoperative patient with neurologic deficit and/or new onset back pain. Prompt evaluation and surgical intervention, where necessary, can reduce the risk of long-term sequelae from this rare complication.

REFERENCES