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Spinal or Epidural Haematoma

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1. Introduction

Intravertebral haematomas may occur in the epidural or subdural space. Generally speaking all intravertebral haematomas are also referred as "spinal haematomas". It is an infrequently described complication of neuroaxial anaesthesia techniques. It has been described in the literature in patients with a deranged coagulation profile in the form of systemic diseases (e.g. chronic renal failure, liver failure) or anticoagulant therapy. In this chapter we will discuss spinal haematomas as a devastating complication of the neuroaxial anaesthesia techniques.

2. Etiopathogenesis of spinal haematoma

Spinal haematoma is the accumulation of blood in the potential space between the dura and bone. It can be a complication of neuroaxial anaesthesia techniques, especially in those patients with a deranged coagulation profile due to systemic diseases (e.g. hepatic diseases, renal failure) or anticoagulant therapy. It is more common in the patients treated with anticoagulants, thrombocytopenia, or in patients with alcoholic liver disease.

Advanced liver disease, with associated portal hypertension and hypersplenism, thrombocytopenia, platelet dysfunction, reduced production of clotting factors, increased clotting factor consumption, and increased fibrinolysis may also increase the risk of bleeding. Before neuroaxial anaesthesia is planned, it is very important to detect liver diseases. The laboratory tests needed are; haemoglobin, PT, aPTT, INR, platelet count, platelet function analysis, and fibrinogen level. Dunn et al. reported that in a case with renal disease also causes haemostatic defects due to defects in platelets, subendothelial metabolism, and platelet vessel interactions. The metabolism of antiplatelet drugs and low-molecular-weight heparins is also reduced

during kidney failure. Coagulation profile tests; bleeding time, PT, PTT, must be performed in all patients with renal failure. Grejda and colleagues reported a case with clotting abnormalities, apparently due to chronic renal failure, who developed paraplegia after spinal anaesthesia secondary to spinal hematoma formation [1, 2].

Malnutrition, fat malabsorption, antibiotic usage, and liver disease may be cause for vitamin k deficiency. Vitamin k deficit leads to a reduction of microsomal carboxylase, a liver enzyme dependent on vitamin k activity, which induce lack of converting factors II, VII, IX, and X into their functionally active forms and consequently, a bleeding diathesis. The clinical findings in these patients are melena, hematuria, ecchymoses and haematomas. In the patient at-risk of bleeding it is recommended to give vitamin k supplements hours prior to a procedure [3].

Other considered factors favouring the formation of a spinal haematoma include: trauma, thrombolysis, lumbar puncture, disc herniation and the vertebral procedures, epidural or spinal anaesthesia, coagulopathy or bleeding diathesis, hepatic disease with portal hypertension, and vascular malformations. Less common causes include systemic lupus erythematosus, ankylosing spondylitis, rheumatoid arthritis, Paget's disease in vertebral bones, Valsalva manoeuvre and hypertension. In 40 to 50% of spinal haematomas there was not an apparent underlying cause [4].

The haemorrhage into the spinal canal frequently occurs in the epidural space due to epidural venous plexus rupture (Baston venous plexus), however arterial haemorrhage may also occur [5, 6]. Nevertheless, the main source of bleeding (arterial or venous) is controversial. Spinal haematoma can be seen quickly with arterial bleeding and can lead to neural trauma and ischemia. However, when aetiology is the needle or the epidural catheter, the spinal haematoma may become symptomatic after a few days, so this situation suggests that the cause is not arterial bleeding. Therefore, spinal haematomas are mostly venous because there are no valves in the epidural venous plexus and the pressure in the epidural space is low. Venous plexus blood flow can be reversed with physical activity and the sudden increases in intra-abdominal and intrathoracic pressure (Valsalva manoeuvre). Epidural venous pressure elevation and hemodynamic changes in pregnancy can also cause rupture on the venous vessel walls. Venous bleeding accumulates slowly, but it can theoretically tamponade the epidural space before exceeding the spinal cord perfusion pressure. Thus, the whole clinical situation may not be clearly understood. The amount of blood causing cord ischemia is variable, and it depends on the speed of blood accumulation. Interestingly, most of the blood volume of the haematomas associated with low-molecular-weight heparin (LMWH) is less than the injected blood volume for epidural patch [7].

The region of spinal haematoma is often at cervical and thoracic vertebrae level, spreading throughout the thoracolumbar spine. Most of the spinal haematomas are seen at the dorsal dural sac, because it adheres to the posterior longitudinal ligament at the front of the spinal canal. Posterior or posterolateral thoracic or lumbar regions are often involved. Usually the haematoma is limited to a few vertebral level [4].

3. Frequency

In the current literature, the incidence of spinal haematoma is about 1/150,000 epidural anaesthesia and 1/220,000 for spinal anaesthesia [7]. From 1906 to 1994, there were 61 spinal haematoma cases reported associated with epidural or spinal anaesthesia [5]. 87% of the patients had haemostatic abnormalities, traumatic or difficult needle insertion attempt, and in 33% of these cases had more than one risk factor. What is relevant is that only 38% of patients had partial or complete neurological recovery. A retrospective study from China reviewed medical records from 1954 to 2008 and found an incidence of 2.14/100.000 (95% confidence interval: 0.44–6.25/100.000) of spinal haematomas after neuraxial blockade. The presence of bacterial infection and the need of emergency surgery were found to increase the risk of epidural haematoma. In general, the risk of major bleeding were multifactorial and increased with age (and associated with abnormalities of the spinal cord or vertebral column), the presence of coagulopathy, anticoagulation (especially standard heparin or LMWH), traumatic needle or catheter insertion. There is a correlation between early decompression surgeries with a better neurological recovery. [8] If laminectomy is performed within 8 hours after the onset of neurological dysfunctions, spinal cord ischemia tends to be reversible [9]. Spinal haematomas are responsible for about half of all spinal cord injuries [10].

The true incidence of neurological dysfunction due to hemorrhagic complications associated with neuroaxial block is not known. Importantly, postoperative numbness or weakness are typically thought to be secondary to the injected local anaesthetic, therefore the diagnosis of cord ischemia may be delayed. On the other hand, patient proper care rarely have the standard level of treatment (1/13 cases), and health care costs are very high. It is impossible to identify the exact risk factors of spinal haematomas from case series. However, the incidence of large surveillance studies (including spinal haematoma) investigated the frequency of high-and low-risk groups are identified. An epidemiological study from Sweden for a 10 years period with 1,260,000 spinal and 450,000 epidural blocks was investigated for serious neurological complications [9]. There were 33 spinal haematoma cases, and from those 24 cases were women and 25 cases were associated with epidural technique. The risk is lower in young women (for epidural analgesia, 1/200,000) than older women (for knee arthroplasty, 1/3,600). Similarly, in women, the risk of spinal haematoma with a hip fracture surgery under spinal anaesthesia (1/22,000) is higher than all spinal anaesthesia (1/480,000) [7, 9].

4. History and physical examination

Needle placement into the spinal or epidural space may damage an epidural vein or artery and causes spinal haematoma formation. Irritation of nerve roots in the epidural space results in acute back pain, and may also cause spinal compression. Impairing vibration, two point discrimination, and position sense are the first clinical findings because the posterior spinal

columns are the first structure to be affected. If the cortical spinal motor tracts are compromised resulting from expanded haematoma, the patient becomes paraplegic. The last clinical findings are pain, temperature changes, and light touch alterations because the anterior lateral spinal thalamic tract is the last structure to be affected [11].

The patient is usually very disturbed, and has often severe, localized and constant low back pain. In addition, a radicular component mimicking disc herniation can be seen. Weakness, drowsiness, urinary or faecal incontinence may be accompanied. In most cases, pain starts spontaneously, but sometimes pain can be associated with minor symptoms such as defecation, lifting, coughing and sneezing. Spinal cord and nerve root dysfunction, depending on the level of the lesion develops quickly and rapidly progression to paraparesis or paraplegia. Low back pain increases with increasing intraspinal pressure manoeuvres that stretching along the spine such as cough, sneeze and percussion. Depending on the size and location of the spinal haematoma the physical findings includes unilateral or bilateral weakness, sensory loss with unilateral or bilateral radicular paresthesia, deep tendon reflexes in the form of various modifications and changes in the bladder and anal sphincter tone.

Lumbar epidural haematoma may mimic an acute disc herniation. Epidural haematoma due to neuroaxial anaesthesia or lumbar puncture may represent new or progressive postoperative neurological symptoms. A time delay in return of loss of sensory or motor (with or without back pain) function after spinal or epidural block, are pathognomonic signals of spinal haematoma, and until proven otherwise, treatment should be considered [4].

5. Diagnosis

Spinal epidural haematoma is usually diagnosed based on the acute neurological deficits, a rapid loss of motor and sensorial function, paraplegia, quadriplegia, or autonomic dysfunction. Quite often patients have acute radiating pain, sensory nerve root or spinal cord compression and focal neurologic deficit. Postoperative epidural haematoma is usually seen in the first 24-48 hours after the neuraxial block. Back pain and lower limb weakness as well as sensory deficit should alert the clinician to the presence of a central compressing lesion. Early clinical signs of pain or focal neurologic deficit are found in the postoperative period. Any new or progressive neurological symptoms or bowel and bladder incontinence require rapid clinical evaluation and diagnostic studies. If a new or progressive neurologic deficit is observed during epidural analgesia infusion, this requires an immediate discontinuation. The epidural catheter is left in place, and no more local anaesthetics are injected because of an early warning signs may be masked by their injection. If an epidural infusion causes the neurological findings, the return of sensory and motor function should be noted when the local anaesthetic effect wear off. On the other hand, speedy radiographic imaging studies and a neurosurgical consultation should be carried out. If there is an acute neurological deficit with low back pain, nerve root and spinal cord compression. An urgent assessment should be made to distinguish situations that mimic spinal haematoma such as epidural abscess, spinal cord disease,

neoplasia, and acute herniated disc. Also, new or progressive neurological manifestations includes muscular or ligamentous injury related to needle placement, postoperative surgical neuropraxia, prolonged or exaggerated neuroaxial block, anterior spinal artery syndrome, and pre-existing undiagnosed neurological disorder need to be discarded.

Complete blood counts including platelets should be done, and the presence of infection should be investigated. Prothrombin time, aPTT, and INR are very useful to study bleeding diathesis

Urgent radiographic diagnostic studies are essential to avoid delay in surgical treatment of spinal haematomas. Magnetic resonance imaging (MRI) is the preferred method due to the rapid and non-invasive technique. MRI can detect presence of the spinal haematoma and location of associated vascular malformation; define the degree of compression of the cord. Also, the age of the haematoma can be diagnosed by MRI. Chronological MRI characteristics of spinal haematoma are similar to intracranial haemorrhage. In hyper acute period (first 6 hours), presence of spinal haematoma is seen isointense in T1-weighted images and mildly hyperintense and heterogeneity in T2-weighted images. In acute period (7-72 hours) hematoma is still isointense on T1-weighted images and begin to hypointense on T2-weighted images. This depends on intracellular deoxyhemoglobine and T2 becomes shorter. With the increase in the concentration of metahemoglobin, haematoma T1 and T2 hyperintense also starts to become homogeneous [4]. (Figure 1).



Figure 1. Sagittal T1 MRI showing an epidural haematoma (h) due to an epidural block in an anticoagulated patient. Courtesy of www.anestesia-dolor.org

Epidural haematoma may be diagnosed by conventional CT, but if the thecal sac or spinal cord haematoma is isodense and if the upper thoracic region image quality is affected by artefacts the result may be falsely negative. Also, CT may not be diagnostic for thoracic spinal level because the resolution is high contrast between the vertebral bone area and lung parenchyma.

Conventional angiography may be required to demonstrate a vascular malformation. Myelography and CT were used in the diagnosis of epidural hematomas, but they are not specific, invasive, and can worsen the clinical condition [4].

6. Precautions, treatment and prognosis

Neuraxial anaesthesia should be avoided in patients who are receiving anticoagulants drugs, patients suspected of bleeding diathesis, thrombolysis or after recent lumbar puncture. Anaesthesiologists should be constantly up to date regarding information on anticoagulation protocols, new anticoagulant medications and the guidelines for regional anaesthesia in this clinical scenario. Antithrombotic therapy in patients receiving neuroaxial block and/or catheter removal needs special timing for the procedure. It should be based on patient regional anaesthesia benefit ratio versus risk of spinal haematoma.

Although some case reports mention that patients with epidural haematoma had been treated successfully with conservative methods, the treatment protocol should be decompressive surgery [12, 13]. Successful non operative treatment has been reported mainly at the level of the cauda equina and in patients with mild neurological symptoms. Most reports of spinal haematomas with neurologic symptoms improved when they were treated with immediate laminectomy; however, the decision of surgery belongs to the neurosurgeon. The most important factors for neurological recovery after a spinal haematoma are preoperative neurological deficit and operative interval. Neurological outcome is related to the time between clinical symptoms and surgical decompression. Early recognition is needed. The clinical symptoms are back pain (radicular), bladder dysfunction and sensory and, more often, motor deficits. These symptoms should initiate immediate further diagnostic efforts. Magnetic resonance imaging is the most appropriate tool. If transport of the patient to a hospital with MRI would prolong the start of surgical therapy considerably, other diagnostic means such as myelography or computed tomography should be considered. Immediate surgical decompression in the case of epidural haematoma is the best way to achieve neurological restitution. Most of the patients with good recovery had less than eight hours delay from the onset of symptoms to surgery [14].

If surgery is delayed, prognosis is poor [15]. For a full neurologic recovery the time interval between the onset of paralysis and surgery should not be more than 8 hours. Neurological improvement without surgery is rare, and consultation for decompression surgery should not be delayed. Overall mortality is 8%. Functional recovery is associated with the duration of symptoms, and the healing is seldom after 72 hours after symptoms have begun [16]. The prognosis for neurological recovery and neurological dysfunction primarily depends

on the duration of the patient's preoperative neurological status. Improvement is due to early diagnosis, hence neurological and neurosurgical consultation should be done as soon as possible. Neurological complications of spinal haematoma include paraplegia, spasticity, neuropathic pain, and urinary and anal sphincter dysfunction.

The clinician performing neuroaxial anaesthesia must be aware of the potential bleeding complication of these procedures. Most cases of spinal haematomas associated with neuroaxial anaesthesia (epidural/spinal) are related to thromboprophylaxis. The American Society of Regional Anesthesia has published guidelines addressing the risk of bleeding and haematomas following neuroaxial techniques in anticoagulated patients, cases receiving antithrombotic or thrombolytic therapy. That will be discussed in detail later in this chapter [10].

Ho et al. [17] summarized in ten steps the safety precautions to minimize the risk of spinal haematoma following epidural catheterization during cardiac surgery:

1. Normalization of coagulation before needle or catheter insertion
2. Avoidance of repeated attempts
3. Postponement of surgery for 24 hours after bloody tap
4. Needle or catheter insertion 1 hour before systemic heparinization
5. Optimization of haemostasis after cardiopulmonary bypass
6. Removal of epidural catheter only after normal haemostasis has been restored postoperatively
7. Close neurologic surveillance
8. Using midline approach technique
9. Administration of saline solution through the needle to distend the epidural space before insertion of the catheter
10. Neuroaxial instrumentation postoperatively only after normalization of coagulation.

Raj and colleagues [18] have developed a bleeding risk score, which estimated based on the potential hazards of bleeding, associated with specific anticoagulants and bleeding disorders. In this scoring system each factor count as one point; the target structure is near a major vascular or neurological structure or is in a confined space. The other factors are the calibre of the needle, the use of fluoroscopy and contrast media, and the use of aspiration are factors that influence the risk and recognition of bleeding, and a "single shot" procedure. The clinician should be made a decision to cancel or carry out the procedure according to the bleeding risk score (Table 1) and overall risk stratification (Table 2)

Risk factors associated with neuroaxial technique	Score
• Proximity to significant vascular structures	1
• Proximity to significant neurological structures	1
• Target in a confined space	1
• Use of a sharp, rather than blunt needle to reach target	1
• Multiple passages	1
• Contrast not used, if applicable	1
• Fluoroscopy not used, if applicable	1
• Aspiration not performed or presence of blood at needle hub	1
• Needle size larger than 20 gauge	1
• Continuous, not single shot procedure	1

Table 1. Bleeding risk score during neuroaxial blocks (adapted from reference 18)

Overall score	0-4	5-6	7-10
Overall risk stratification	Low	Medium	High

Table 2. Overall risk stratification according to the bleeding risk score

Antithrombotic therapy

In 2008 the American College of Chest Physicians (ACCP), 8th Antithrombotic and Thrombolytic Therapy Conference issued a statement [19]. Recommendations from this report are summarized in Table 3.

These recommendations have brought new challenges for the management of patients with neuraxial block. In general, the long-range high degree thromboprophylaxis is recommended. Acceptable alternative guide to ACCP is Surgical Care Improvement Project (SCIP; www.qualitynet.org). In addition, American Academy of Orthopaedic Surgeons (AAOS) has published a guide in 2007 for surgical bleeding associated with thromboprophylaxis for deep vein thrombosis in patients undergoing hip surgery to prevent pulmonary embolism (www.aaos.org/guidelines.pdf). In general, the AAOS guideline is more conservative and recommends routine mechanical prophylaxis and aggressive chemoprophylaxis in high-risk patients [10].

Understanding of mechanism of blood coagulation, pharmacological properties and clinical studies of anticoagulation and antiplatelet medication reduced the risk of spinal haematoma in neuroaxial blocks.

Risk level	DVT risk without thromboprophylaxis (%)*	Recommended thromboprophylaxis options
Low risk • Minor surgery in mobile patients • Medical patients who are fully mobile	<10	Not specific thromboprophylaxis Early and aggressive ambulation
Middle risk • Most of gynecological or urological patients • Internal medicine patients: in bed, middle VTE risk+high bleeding risk	10–40	DMWH (on recommended doses), LDUH or fondaparinux Mechanical thromboprophylaxis
High risk • Hip and knee arthroplasty, hip surgery • Major trauma, spinal cord injury, high VTE risk + high bleeding risk	40–80	DMWH (on recommended doses), fondaparinux, oral vitamin K antagonist (INR 2–3) Mechanical thromboprophylaxis

* Asymptomatic DVT rates with objective diagnostic screening in patients not receiving thromboprophylaxis.

† mechanical thromboprophylaxis: intermittent pneumatics pressure, venous foot pump, and/or Anti-embolism Stockings, Low molecular weight heparin (LMWH), LDUH: low dose unfractionated heparin, INR, international normalized ratio, VTE: venous thromboemboli

Table 3. The risk of thromboembolism and recommended thromboprophylaxis for patients in hospital stay [19].

7. Thrombolytic and fibrinolytic therapy

Thrombolytic agents actively dissolve the fibrin clots. Exogenous plasminogen activators (streptokinase and urokinase) are drugs used to dissolve thrombus, and also affect circulating plasminogen that is decreasing the plasminogen and fibrin levels. Recombinant tissue-type plasminogen activator (rt-PA) is more fibrin selective and has less impact on the level of circulating plasminogen. Clot lysis leads to increase the fibrin degradation products, they also inhibit platelet aggregation and has anticoagulant effect. About one day after the administration of thrombolytic agents the haemostasis is altered. Fibrinogen is the latest improved factor. In addition to fibrinolytic agent, these patients often receive intravenous heparin and clopidogrel or aspirin in order to keep 1.5-2 times higher than the normal aPTT level. There are spinal haematomas case reports in patients taking thrombolytic agents while had an epidural catheter placed [7, 9].

The contraindication guidelines for thrombolytic agents suggests that if these drugs are taken, a needle insertion attempt should not be done during the following 10 days. Although there is no precise data for a neuroaxial block attempt after stopping the drugs [7, 9].

8. Management of regional anaesthesia in patients receiving thrombolytic and fibrinolytic therapy [20].

- Patients receiving fibrinolytic or thrombolytic drugs should be considered if spinal or epidural anaesthesia are thought to be applied.
- Patients receiving fibrinolytic or thrombolytic therapy, there is no suggestion a definite time for the withdrawal of the epidural catheter. Fibrinogen level measurement may be useful to assess residual thrombolytic effect.

8.1. Oral anticoagulants

Patients have different sensitivities to anticoagulants. In very sensitive patients the effect of anticoagulants drugs is more potent and longer after their discontinuation. Prothrombin time (PT) is up to 20% higher with a single dose of 3 to 5 mg of warfarin. The patients with resistance to these drugs have shorter duration of anticoagulation. Factors that increase the sensitivity to heparin and warfarin include general medical condition, diet, > 65 years old, female gender, excessive surgical blood loss, liver, or heart, or kidney diseases [7].

Anaesthetic management of patients receiving preoperative warfarin depends on the dosage and the treatment time. In patients with chronic oral anticoagulation, PT and INR levels take 3-5 days to return to normal values after anticoagulants discontinuation. Theoretically, PT and INR will be affected more factor VII activity (factor VII half-life of 6-8 hours), the time to return to normal PT and INR, factor II and X levels may not be sufficient for haemostasis. If INR is within the normal range, vitamin k dependent factors are typically normal. Thus, the coagulation should returns to normal range before the neuraxial block [10]. Although after warfarin discontinuation PT/INR are back to normal, the residual (subclinical) warfarin anticoagulant effect may be seen postoperatively [9]. Depending on the time of warfarin initiation factor half-life: Factor VII: 6-8 hours, Factor IX: 24 hours, Factor X: 25-60 hours, Factor II: 50-80 hours.

The correlation between vitamin k dependent coagulation factors and INR should be known for the proper management of regional anaesthesia. In patients with congenital factors II, IX and X deficits, activity levels of each factor for hemostasis should be normal or at least reach to 40%. If any clotting factor level are 20-40% below of their normal values, bleeding may occur. Factor VII and X activities are sensitive to PT and INR, but factor II activity is less sensitive to them. Because factor VII have a relatively short half-life, PT and INR may increased in 24-36 hours.

When factor VII activity is approximately 55% the INR can be longer (INR > 1.2). If INR is 1.5 the activity of factor VII is about 40%. If INR is <1.5 the haemostasis should be normal. However, prolonged PT/INR with factor VII activity may range from normal to very low levels. Neuraxial catheterization is safe with normal PT/INR, but with prolonged PT/INR is difficult to interpret for VII factor activity, and as much as 10% of the epidural catheters are withdrawn early and unnecessary [6]. Adequate anaesthesia management of patients receiving warfarin has to be based on the proper knowledge of the anticoagulant pharmacology, vitamin-K-dependent factors levels, and in the experience of reported cases of spinal haematoma [10].

Proper time for withdrawal of the neuroaxial catheter is controversial. Nearly 6,000 patients who received preoperative oral anticoagulants with spinal or epidural catheters is examined with four studies [7, 21]. This study showed that responses of patients with warfarin have been highly variable. Up to 48 hours after initiation of treatment PT may not be prolonged, but even after a single dose, PT prolongation occurred in significant number of patients. Larger doses (> 5 mg of warfarin) increases these findings. To avoid excessive PT prolongation, you should assess daily levels [10].

8.2. Regional anaesthesia management for the patients treated with anticoagulants [20].

- Oral anticoagulation is stopped before neuraxial block, and normalization of PT is verified.
- PT and INR are monitored daily.
- When the vitamin-K dependent factors are adequate (INR <1.5), neuraxial catheters can be pulled out.
- There is no definite recommendation when INR values are >1.5-<3.0 regarding the withdrawal of neuraxial catheters. Neurological condition should be carefully evaluated until INR stabilized, and the neuraxial catheters should be cautiously withdrawn.
- If INR > 3, warfarin should be avoided. There is no definite recommendation regarding the withdrawal of neuraxial catheters (eg. partial or complete recovery of the anticoagulant effect of warfarin, or it can be interrupt until recovery of spontaneous haemostasis).

8.3. Intravenous and subcutaneous standard (unfractionated) heparin

In high risk patients (acute thromboembolism), full systemic heparinization is given until a normal aPTT level is doubled. However, in vascular interventions, intravenous mid-level dosage of heparin (~5000 units) is given intraoperatively. Systemic heparinization in patients with spinal or epidural catheters were found to be safe in more than 4000 patients. Spinal haematomas were detected in 2% of these patients after diagnostic lumbar procedures and heparinization. In patients with anticoagulation within concomitant use of aspirin or anticoagulant therapy, after traumatic needle attempt or after a week of this attempt are defined as risk factors for spinal haematoma [7].

In general, a large number studies and clinical experience of regional anaesthesia techniques do not predict the risk of spinal haematoma during the use of systemic heparinization. However, they have stated that such events are not rare as previously thought, and if there is a suspicion for spinal haematoma diagnostic processes should be done as early as possible [10].

In patients receiving high-dose intraoperative systemic heparin, particularly in cardiac surgery. Epidural and spinal anaesthesia and analgesia has gained popularity and has not been reported cases of spinal haematomas. The possibility of epidural haematoma in these patients is for epidural anaesthesia 1/1528, and for spinal anaesthesia 1/3610 [9].

In a study conducted on 9000 patients that received subcutaneous heparin and applied spinal or epidural anaesthesia no spinal haematoma was reported. In patients receiving low-dose

heparin, only four spinal haematoma has been reported after neuraxial block, epidural anaesthesia technique was applied in three of them [9]. Neuraxial block security for these patients is not known [19].

8.4. Regional anaesthesia management in patients receiving unfractionated heparin [20].

Regional anaesthesia and intravenous heparin for vascular surgery can be considered under the following conditions:

- After needle or catheter attempt intravenous heparin is delayed by 1 hour.
- Prolonged anticoagulation increases the risk of spinal haematoma, especially in combination with other anticoagulants or thrombolytic agents. If systematic anticoagulation is given when the patient has an epidural catheter, its withdrawal has to be delayed 2-4 hours after discontinuation of heparin, and after the coagulation status is evaluated.
- The catheter is withdrawn one hour before administration of heparin.
- If total daily dose < 10 000 units, there is no contraindication to neuraxial techniques if subcutaneous standard heparin is given. The risk of spinal haematoma with higher doses are uncertain; neurological follow-up is done on an individual basis and is closely evaluated.
- In patients receiving subcutaneous heparin > 5 days serial platelet counts should be performed.

8.5. Low-Molecular-Weight Heparin (LMWH)

Enoxaparin was the first approved LMWH by the FDA in 1993. Between 1993-1997, 30 cases of spinal haematoma were seen in patients receiving LMWH and undergoing spinal or epidural anaesthesia. In 1997, the FDA has started to investigate these cases. In addition, all LMWH and heparinoid manufacturers were warned [9].

Anesthesia and Anticoagulation Neuroaxial consensus conference (1998) described 45 spinal haematoma cases associated with LMWH, in 40 of these patients neuraxial anaesthesia was implicated. Severe radicular back pain was not manifest symptoms, but most patients had incipient numbness, weakness, and bowel or bladder dysfunction. Median time between the treatment start with LMWH and neurological dysfunction development was 3 days, and the mean time between onset of symptoms and laminectomy was more than 24 hours. Bad or good neurological recovery were seen in less than 1/3 patients.

The risk of spinal haematoma due to LMWH, neuraxial techniques, and the prevalence of reported cases, with continuous epidural anaesthesia was reported in approximately 1/3000, with spinal anaesthesia 1:40,000. However, the cases are probably more. About 60 cases have been reported between the years of 1993-1998 by the FDA. The Second Consensus Conference 1998-2002, reported 13 spinal haematoma cases related to neuraxial block. In addition to LMWH, 5 patients received ketorolac, one patient was taking ibuprofen, and one patient received intravenous unfractionated heparin. Spinal anaesthesia in 3 cases, and 10 patients underwent epidural anaesthesia while receiving LMWH. Thus, the reported characteristics of

patients are supported previously recommendations that epidural catheter withdrawal before starting LMWH thromboprophylaxis and other antiplatelet or anticoagulant medication [9]. (Table 4).

Patients factors	Female gender
	Elderly
	Ankylosing spondylitis or spinal stenosis
	Renal failure
Anaesthesia factors	Traumatic needle/catheter placement
	More risk in epidural compared with spinal technique
	Epidural catheter placement during LMWH therapy
LMWH dosage factors	Early preoperative (or intraoperative) LMWH therapy
	Early postoperative LMWH therapy
	Concomitant antiplatelet or anticoagulant medications
	Twice daily LMWH administration

Table 4. Patient, anaesthetic, and LMWH dosing variables associated with spinal haematoma [7]

The effect of renal function cannot be completely evaluated. The anticoagulant effect increased in serious renal failure and half-life is prolonged from 4-6 hours to 16 hours [22].

Efforts to determine the indications for LMWH are continuing. LMWH is not a good choice for patients receiving chronic warfarin therapy, and is indicated for pregnant women, in patients with prosthetic heart valve and atrial fibrillation, and in patients with a history and existing hypercoagulable states. LMWH dose for the treatment of DVT is higher than dosage of prophylaxis. The needle must be inserted at least 24 hours after last dose [9].

8.5.1. Regional anaesthesia management in patients receiving LMWH [20]

Perioperative management of patients receiving LMWH requires coordination and communication. In addition, even if there is a protocol the patient dose may not be closely followed. LMWH is not recommended with other antiplatelet or oral anticoagulant drugs.

Preoperative LMWH

- Neuraxial techniques should be applied at least 10-12 hours after thromboprophylaxis dose and 24 hours after high therapeutic dose of the LMWH.

Postoperative LMWH

- Two doses per day, the first dose of LMWH should be given at least 24 hours after the operation, regardless of the anaesthetic technique, and only if there is adequate hemostasis.
- Remove the catheter before starting LMWH thromboprophylaxis.

- The first dose of LMWH should be given at least 2 hours after withdrawal of catheter, and after 24 hours insertion of needle or catheter.
- The interval between the insertion of needle or catheter with the first dose of LMWH should be 6-8 hours. The other dose should not be given within 24 hours after the first dose.

Antiplatelet medications

Antiplatelet drugs includes the nonsteroidal anti-inflammatory drugs (NSAIDs), thienopyridine, and glycoprotein IIb/IIIa inhibitors, seldom used as agents for primary thromboprophylaxis. It is important to note the pharmacologic differences among the drugs with antiplatelet effects. Many orthopaedic patients are chronic NSAIDs (aspirin, ibuprofen, ketorolac, and naproxen) users. Three of the 61 reported patients who developed spinal haematoma after spinal or epidural anaesthesia were taking antiplatelet therapy. Many studies of these drugs showed that they are relatively safe for neuraxial block in obstetric, surgical, or pain clinic patients [10, 23]. Clinician also should be alert in heparinized patients who also take antiplatelet agents as possible increased risk of spinal haematoma. Ticlopidine and clopidogrel are from thienopyridine group, and are also platelet aggregation inhibitors. These agents inhibit the platelet-fibrinogen binding and then impair platelet-platelet interaction [9]. This effect is irreversible during the platelets life time. Platelet dysfunction lasts 5-7 days for clopidogrel and 10-14 days after ticlopidine. Clopidogrel in completely normal clotting dose range need not secure due to the block. Prasugrel is a new thienopyridine drug and inhibits platelets faster and sustained. It is used only for percutaneous coronary intervention in acute coronary syndromes in the United States of America.

Platelet glycoprotein IIb/IIIa receptor antagonists (abciximab, eptifibatide and tirofiban) impair platelet aggregation, platelet-fibrinogen binding and platelet-platelet interaction. Time to normal platelet aggregation following discontinuation of therapy ranges from 8 hours (eptifibatide, tirofiban) to 24 to 48 hours (abciximab). During therapy with GP IIb/IIIa antagonists, labeling precautions recommend that puncture of noncompressible sites and "epidural" procedures be avoided [9].

8.5.2. Regional anaesthesia management in patients receiving antiplatelet drugs [20].

- If antiplatelet drugs are taken together with other anticoagulants, bleeding risk is high.
- NSAIDs alone does not a significant risk factor for spinal haematoma in epidural or spinal anaesthesia.
- Platelet function should be return to normal before neuroaxial block in patients receiving ticlopidine, clopidogrel, and platelet GP IIb/IIIa receptor antagonists. Platelet aggregation was returning to normal after discontinuation of the drug: 14 days for ticlopidine; 5-7 days for clopidogrel; 7-10 days for prasugrel. The effects of GP IIb/IIIa inhibitors are terminated between 8 hours (eptifibatide and tirofiban) with 48 hours (abciximab).

8.6. Herbal medications

The use of herbal products in surgical patients is frequent, and sometimes the patients also may disguise their use of them. Polypharmacy and physiological changes lead to morbidity and mortality in the perioperative period. The bleeding may be seen due to garlic, ginkgo, ginseng and ginseng-warfarin interaction. These commercial products are not yet sufficiently under control, therefore, unexpected and adverse reactions may be seen, and in particular, and the anaesthesiologist should be familiar the effects of these agents [9].

Herbal treatment and regional anaesthesia management [20].

- Herbal remedies does not pose any additional risk for spinal haematoma in epidural or spinal anaesthesia. There is not a fully accepted test to assess the adequacy of haemostasis for the herbal products. Inquiry and evaluation should be performed preoperatively. There is no data evaluating the combination of herbal therapy with other anticoagulants. However, an increased risk of bleeding with the drugs that effect on hemostasis system.

8.7. Fondaparinux

Fondaparinux is a synthetic pentasaccharide, the FDA approved in 2001, makes antithrombotic effect by the inhibition of factor Xa. Plasma half-life of 21 hours, 6 hours after the operation is given once daily. Spinal haematomas have not been reported with its use, but it must be used very carefully. The actual risk of spinal haematoma to fondaparinux is unknown.

Fondaparinux and regional anaesthesia management [20].

- Neuraxial techniques should be applied carefully until an adequate clinical information is obtained (single pass through with a needle, the use of atraumatic needle, avoidance from neuraxial catheter). If the precautions are not possible, another method of prevention should be considered.

8.8. Dabigatran

Dabigatran etexilate reversibly inhibits clot bound thrombin. It is a prodrug, gastrointestinal absorption and bioavailability is about 5%. After being absorbed, esterases returned it the active metabolite, dabigatran. A single dose of dabigatran has a half-life of 8 hours, with multiple doses is 17 hours. Daily dose of the drug is suitable. It is contraindicated in patients with renal failure, because 80 % of the drug is excreted unchanged from the kidney. Spinal haematomas are not reported but currently data are insufficient. It is currently used only in non-valvular atrial fibrillation [7].

Management of regional anaesthesia in patients receiving dabigatran [20].

- Dabigatran should be discontinued 7 days before the neuraxial block, because of the long half-life, and irreversible effect. Neuraxial catheters should be withdrawn at least 6 hours prior to initiation of treatment with dabigatran [22].

8.9. Rivaroxaban

Rivaroxaban is a potent, selective and reversible, orally active factor Xa inhibitor. Oral bioavailability is approximately 80%. Maximum inhibitory effect is seen in 1-4 hours and the inhibition lasts 12 hours. Antithrombotic effect is measured by PT, aPTT, and Heptest. It is excreted through kidney and intestine, therefore in patients with renal failure is contraindicated. The half-life is 9 hours, but in the elderly last up to 13 hours. Clinical studies showed that rivaroxaban (5-40 mg a day, the first dose 6-8 hours after surgery) a similar effect with enoxaparin (40 mg given 12 hours before surgery). Although spinal haematoma is not reported, it must be use cautiously because of the longer half-life [10].

Management of regional anaesthesia in patients receiving rivaroxaban [20].

- According to the European guidelines, neuroaxial block can be applied after 22 to 26 hours of discontinuation of rivaroxaban. If there is renal failure this interval will be longer. Neuraxial catheters are contraindicated. Postoperative rivaroxaban treatment should be started at least 4-6 hours after the spinal block [22].

8.10. Peripheral nerve blocks and plexus blocks

It is known that the most important serious complication of neuraxial blocks is spinal haematoma, but the risk is not identified for plexus and peripheral blocks. Few serious complications have been reported. In patients received antiplatelet or anticoagulant agents, major bleeding is reported after lumbar sympathetic block, or psoas compartment block. Neurological damage has not been reported. The Neuroaxial Anesthesia and Anticoagulation Consensus Statement are also used for the peripheral and plexus blocks [9, 10]

9. Diagnosis and treatment

In the differential diagnosis of postoperative new or progressive neurologic symptoms, surgical neuropraxia, prolonged or exaggerated neuraxial block, anterior spinal artery syndrome, epidural abscess, recurrence and existing undiagnosed neurological condition, neurological disorders and spinal haematoma should be considered. Immediate post-operative onset of symptoms is rare. Spinal haematomas rarely can be seen as "prolonged" in the form of blocks [5, 6]. The time between the start of thromboprophylaxis with the entry of the needle is important for neurological dysfunction. Complete paralysis develops within 10-15 hours after the start of neurological deficits. Clinical assessment should be focused on the recognition of reversible or treatable causes. Thus, if any new or progressive neurological symptoms are seen during epidural analgesia, infusion must be promptly stopped (catheter is left) and the local anaesthetic effect and of volume effect is ruled out. If the neurological deficit is due to the local anaesthetic and/or volume effects, the deficit is often return quickly, and it should be noted. Neurological recovery is due to early diagnosis and intervention, radiographic imaging, preferably MRI should be done as soon as possible. In terms of the need for emergency surgery, neurosurgery consultation should be requested immediately. Inter-

estingly, all spinal haematomas do not require emergency surgery, spontaneous healing have also been reported [5, 6]. However, the decision of an emergent surgery or observation belongs to the neurosurgeon. Neurological outcome for most patients are worse in all series. In addition, if there is more than 8 hours after onset of the symptoms, a full recovery usually has not been realized. Generally, bleeding after peripheral techniques is less common than neuraxial haematoma, and often appears as hypovolemia not neural deficit. The decide for surgery or observation for neuraxial haematoma or bleeding is based on the presence and severity of neural deficit.

10. Summary and conclusion

Spinal hematoma is a haemorrhage in the spinal or epidural space that develops with forming a heterogeneous group of disorders. Haematoma can be acute, chronic, spontaneous, traumatic or iatrogenic. It is especially related to medication or disease associated with coagulopathy. MRI is a special importance in diagnosis. Delay of surgery may rapidly worsen the clinical outcome, so the surgery should be done urgently.

Spinal haematomas are rare and potentially reversible spinal cord compression. Early diagnosis is essential for a full recovery. Spinal haematomas can occur in the absence of identifiable risk factors. The clinician should be alert for the new neurological signs. Spinal cord and root compression are potentially reversible. If the treatment is done quickly healing is complete. Continuous surveillance of risk identification, assessment and training up to date information for the physician should be done constantly for spinal and epidural blocks. The introduction of new anticoagulants and antiplatelet agents, and the complex balance between thromboembolic events and hemorrhagic complications with regional anaesthesia or analgesia requirement requires an evaluation of the indications for patient. Thus, the antithrombotic therapy in patients receiving spinal or epidural anaesthesia or analgesia, timing of catheter removal should be evaluated basis on the patient's situations. If there is an unacceptable risk, alternative anaesthesia or analgesia techniques should be considered. The patient's coagulation status should be optimized and the level of anticoagulation should be carefully monitored during epidural catheterization. If there is a significant increase in the risk of spinal haematoma, the catheter should not be removed. Identification of risk factors and the publication of the guidelines does not eliminate the complication of spinal haematoma. It is reported that spinal haematoma may develop in patients treated in accordance with the guidelines [24, 25]. United States [10] Europe [26] and the Nordic countries [27, 28] have published guidelines. Closely monitored of the patient to detect early neurological dysfunction is very important to recognize and attempt to fast decompression. Not only try to prevent of spinal haematoma, but also should be focused to make the best of the neurological consequences [7, 10].

Summary of the clinical key points

1. The clinician should have a high index of suspicion at all times in any patient who has undergone spinal anaesthesia and who exhibits any sign or symptom of a neuraxial haematoma

2. Adequate monitoring, follow-up, and immediately treatment are essential in patients on anticoagulants who are receiving neuraxial blocks
3. Early recognition of epidural haematoma
4. Physical examination: vibration and position ability in the lower extremities. Pain, temperature, and light touch (last sensory modalities), Assessing rectal tone.
5. If a new or progressive neurologic deficit are observed during epidural analgesia infusion, it requires immediate discontinuation and the catheter is left in place
6. Urgent radiographic diagnostic studies: MRI (more sensitive and preferred method), Conventional CT, conventional angiography, myelography and CT
7. Differential diagnosis: Epidural abscess, spinal cord disease, neoplasia, muscular or ligamentous injury related to needle placement, postoperative surgical neuropraxia, prolonged or exaggerated neuroaxial block, anterior spinal artery syndrome, and pre-existing undiagnosed neurological disorder
8. Emergency neurosurgical evaluation for surgical decompression.

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