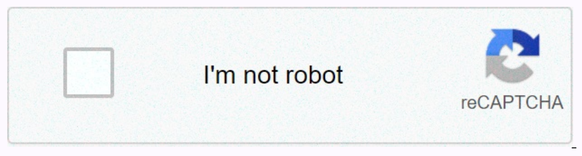

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Pulmonary embolism treatment guidelines canada

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In patients with PE, if concerns about subcutaneous absorption occur, severe renal failure is present, or if thrombolytic therapy is considered, IV UFH is the recommended form of initial anticoagulation. [5] Clinicians often choose to use IV UFH instead of LMWH and fondaparinux in specific clinical circumstances where medical or surgical procedures are likely to be performed and the short half-life of IV UFH allows for temporary cessation of anticoagulation and presumed reduction of bleeding risk during the procedure. Although this approach has limited supporting evidence, it seems to constitute a reasonable practice. The effect of heparin treatment depends on achieving a critical therapeutic level of heparin within the first 24 hours of treatment. The critical therapeutic level of heparin is 1.5 times the baseline control value or the upper limit of the normal range of the activated partial platelet plastin (aPTT). This level of anticoagulation is expected to correspond to a heparin blood level of 0.2-0.4 U/mL of protamine sulfate titration analysis and 0.3-0.6 of the anti-factor X assay. Each laboratory should determine the minimum therapeutic level of heparin, measured by aPTT, to coincide with a heparin blood level of at least 0,2 U/mL for each batch of thromboplastic internal agent used. If IV UFH is selected, an initial bolus of 80 U/kg or 5000 U followed by an infusion of 18 U/kg/h or 1300 U/h should be given, with the aim of rapidly achieving and maintaining aPTT at levels corresponding to therapeutic heparin levels. Fast-dose and supervised regimens of subcutaneous UFH are available and are acceptable alternatives. Current guidelines for patients with acute PE recommend LMWH over IV UFH (Grade 2C) and over SC UFH (Grade 2B). [5] In patients treated with LMWH, once daily regimens are preferred to twice daily regimens (Grade 2C). The choice between fondaparinux and LMWH should be based on local considerations to include cost, availability and familiarity with use. LMWHs have many advantages over UFH. These funds have a greater bioavailability, can be through subcutaneous injections, and have a longer duration of anticoagulant effect. A fixed dose of LMWH can be used, and laboratory monitoring of aPTT is not necessary. Trials comparing LMWH with UFH have shown that LMWH is at least as effective and as safe as UFH. The studies have not pointed to any significant differences in recurrent thromboembdifferent events, major bleeding, or mortality between the 2 types of heparin. LMWH can be safely administered in an outpatient environment. This has led to the development of programs where clinically stable patients with PE are treated at home, to significant cost savings. The ACCP guidelines suggest that patients with low-risk pe who have acceptable circumstances at home are discharged early from the hospital (ie, before the first five days of treatment) (Grade 2B). An international, open-label, randomised study compared outpatient and inpatient treatment (both with LMWH enoxaparin as initial treatment) of low-risk patients with acute PE and concluded that outpatient treatment was noninferior to inpatient treatment. [87] Apixaban, dabigatran, rivaroxaban, and edoxaban are alternatives to warfarin for prophylaxis and treatment of PE. Apixaban, edoxaban and rivaroxaban inhibit factor Xa, while dabigatran is a direct thrombin inhibitor. Rivaroxaban Rivaroxaban (Xarelto) is an oral factor Xa inhibitor approved by the FDA in November 2012 for the treatment of DVT or PE, and to reduce the risk of recurrent DVT and PE after initial treatment. Approval for this indication was based on studies involving a total of 9478 patients with DVT or PE. Participants were randomly assigned to receive rivaroxaban, a combination of enoxaparin and a vitamin K antagonist (VKA) (e.g. warfarin), or placebo. Study endpoints were designed to measure the number of patients who experienced recurrent symptoms of DVT, PE or death after receiving treatment. [88, 89] In addition, results from extended treatment showed a reduced risk of recurrent DVT and PE. Approximately 1.3% in the rivaroxaban group experienced recurrent DVT or PE compared to 7.1% in the placebo group. [90, 91] The results of the Einstein-PE study provide an important advance in the treatment of symptomatic PE. In a prospective open-label study, 4,832 patients were randomised to receive either rivaroxaban or enoxaparin followed by an adjusted dose of vitamin K antagonist for 3, 6 or 12 months. Treatment with a fixeraddos regimen of rivaroxaban was noninferior to standard therapy and had a satisfactory safety profile. [88] Data from a pooled analysis of the EINSTEIN-PE and EINSTEIN DVT studies in the treatment of DVT or pulmonary embolism suggest that rivaroxaban is as effective in preventing VTE recurrence as administration of enoxaparin followed by a vitamin-K antagonist. [92, 93] Rivaroxaban may also be associated with minor bleeding, especially in elderly patients and patients with moderate renal impairment. [92, 93] Rivaroxaban use for VTE prevention in sick medical medical with limited mobility demonstrated noninferiority to enoxaparin in short-term use (10 ± 4 days) and superiority in long-term use (35 ± 4 days) compared to short-term use of enoxaparin followed by placebo. [94] Another study did not show a significant benefit of rivaroxaban over placebo in reducing the compound endpoint of symptomatic VTE or death in medically ill patients at increased risk of VTE after discharge; However, there were few events and the primary safety outcome, major bleeding, did not significantly increase with treatment. [95] Apixaban Apixaban was approved for treatment by PE in August 2014. The approval for the treatment of PE and prevention of recurrence was based on the results of AMPLIFY (Apixaban for the initial management of pulmonary embolism and deep-vein thrombosis as First-Line Therapy) and AMPLIFY-EXT studies, in which apixaban therapy was compared with enoxaparin and warfarin treatment. The AMPLIFY study showed that, in comparison to the standard anticoagulation regimen, apixaban therapy resulted in a 16% reduction in the risk of a compound endpoint that included recurrent symptomatic vein thromboembolism (VTE) or VTE-associated deaths. [96, 97] This advance thus offers the prospect of a safe and effective regimen of anticoagulation for patients with the benefits of simplicity and cost-effectiveness in comparison to current management strategies. Dabigatran Dabigatran (Pradaxa) was approved by the FDA in 2014 for the treatment of DVT and PE and reduces venous thromboembolic recurrence. In the RE-COVER and RE-COVER 2 studies, patients with DVT and PE who had received initial parenteral anticoagulation (e.g., IV heparin, SC LMWH) were randomised for 5-10 days to warfarin or dabigatran. These two studies showed dabigatran was noninferior to warfarin to reduce DVT and PE and was associated with lower bleeding rates. [98, 99] Edoxaban Edoxaban (Savaysa) was approved by the FDA in January 2015 for the treatment of DVT and PE in patients who have initially been treated with a parenteral anticoagulant for 5-10 days. The approval was based on the Hokusai-VTE study, which involved 3,319 patients with PE. Of these patients, 938 had right ventricular dysfunction, which was assessed by measuring N-terminal pro-brain natriuretic peptide levels. The share of recurrent VTE in this subgroup was 3.3% in the Edoxaban group and 6.2% in the warfarin group. Edoxaban was noninferior to high-quality standard warfarin therapy and caused significantly less bleeding in a wide range of patients with VTE, including those with severe pulmonary embolism. [100] Betrixaban Betrixaban, a factor Xa inhibitor, was approved by the FDA in June 2017. It is indicated for prophylaxis of VTE in adults hospitalized for acute medical disease who are at risk of thromboembolic complications due to moderate or severe limited mobility and other risk factors that may cause VTE. Approval of betrixaban was based on phase 3 data randomised, double-blind, multinational clinical trials compared betrixaban (35-42 days) with short-term enoxaparin (6-14 days) for VTE in 7513 acutely hospitalised patients with VTE risk factors. Patients in the betrixaban group took an initial dose of 160 mg perorelle on day 1, followed by 80 mg once daily for 35-42 days, and received a placebo injection once daily for 6-14 days. Patients in the enoxaparin group received 40 mg of subcutaneously once daily for 6-4 days and took oral placebo once daily for 35-42 days. Efficacy was measured in 7441 patients using a composite score consisting of the presence of asymptomatic or symptomatic proximal deep vein thrombosis, nonfatal pulmonary embolism, stroke, or VTE-related deaths. Betrixaban showed significant reductions in VTE events compared to enoxaparin. [101, 102] In patients with acute PE, fondaparinux as initial treatment is favored over IV UFH and over SC UFH. [5] The choice between fondaparinux and LMWH should be based on local considerations to include cost, availability and familiarity with use. Fondaparinux is a synthetic polysaccharide derived from the antitro-binding region of heparin. Fondaparinux catalysis factor Xa inactivation by antithrombin without inhibiting thrombin. Once daily fondaparinux was found to have similar rates of recurrent PE, bleeding, and death to IV UFH, according to a randomized open-label study of 2,213 patients with symptomatic pulmonary embolism. [103] In general, the use of LMWH or fondaparinux is recommended in front of IV UFH and SC UFH. This is due to a more predictable bioavailability, faster instiva effect, and the advantage of not typically needing to monitor anticoagulants effect. However, if uncertainty arises as to the correctness of the dose, factor Xa levels may be monitored to determine the effect. A vitamin K antagonist like warfarin should be started on the same day as anticoagulants therapy in patients with acute PE. [5] Parenteral anticoagulation and warfarin should be continued together for at least five days and until INR is 2.0. The anticoagulation effect of warfarin is mediated by the inhibition of vitamin K-dependent factors, which are II, VII, IX, and X. The peak effect does not occur until 36-72 hours after drug administration, and the dosage is difficult to titrate. A prothrombin time ratio is expressed as an INR and monitored to assess the appropriateness of warfarin therapy. The recommended therapeutic range for venous thromboembolism is an INR of 2-3. This level of anticoagulation significantly reduces the risk of bleeding without losing effectiveness. Initially, INR measurements are performed daily; once the patient has stabilised at a specific dose of warfarin, the INR determinations can be performed every 1-2 weeks or at longer intervals. A patient with a first thromboembolism event that occurs when adjusting reversible risk factors, such as surgery, or trauma, should receive warfarin treatment for at least 3 months. No difference in relapse rates was observed in any of 2 studies comparing 3 compared to 6 months of anticoagulants in patients with idiopathic (or unprovoked) first events. [104, 105] The current recommendation is anticoagulation for at least 3 months in these patients; the need to extend the duration of anticoagulation should be reassessed at this time. The current ACCP guidelines recommend that all patients with unprovoked PE receive three months of anticoagulation treatment for a shorter duration of treatment and have an assessment of the risk-benefit ratio for prolonged treatment at the end of three months (Grade 1B). [5] Patients with a first episode of venous thromboembolism and with a low or moderate risk of bleeding should have prolonged anticoagulants (grade 2B). Patients with a first episode of venous thromboembolism with a high risk of bleeding should have treatment limited to three months (Grade 1B). In patients with a second unprovoked episode of venous thromboembolism and low or moderate risk of bleeding, prolonged anticoagulation therapy (grades 1B and 2B, respectively) is recommended. In patients with a second episode of venous thromboembolism and high risk of bleeding, three months of anticoagulation is preferable to prolonged anticoagulation (Grade 2B). Patients who have PE and pre-existing irreversible risk factors, such as deficiency of antithrombin III, protein S and C, factor V Leiden mutation, or the presence of antiphospholipid antibodies, should be placed on long-term anticoagulation. Patients who have PE in collaboration with an active neoplasm present challenges for long-term management due to their increased continued risk of recurrent VTE and PE. The ninth edition of ACCP guidelines recommends that such patients receive extended anticoagulation as opposed to three months of treatment if they are low or moderate at risk of bleeding complications (Grade 1B). [5] If patients with active neoplasm are at high risk of bleeding, it is still suggested that they receive extended therapy, although the supporting evidence is less conclusive (Grade 2B). For the treatment of PE in cancer patients, LMWH is instead recommended for a vitamin K antagonist such as warfarin (grade 2B). However, some cancer patients choose not to receive long-term treatment with LMWH due to the need for daily injections and treatment costs. If cancer patients with PE choose not to receive treatment with LMWH, a vitamin K antagonist is preferred as warfarin over dabigatran or rivaroxaban (grade 2C). Heparin-induced thrombocytopenia (HIT) is a transient prothrombotic disorder initiated by heparin. The main features of HIT are (1) thrombocytopenia resulting from immunoglobulin G-mediated platelet activation and (2) in vivo thrombus ingeneration and increased risk of venous and arterial thrombosis. The highest frequency of HIT, 5%, has been reported in surgery patients receiving up to 2 weeks of molested heparin. HIT occurred in approximately 0.5% of patients receiving LMWH after orthopaedic surgery for up to 2 weeks. HIT can manifest clinically as an extension of the thrombus or the formation of new arterial thrombosis. HIT should be suspected whenever the patient's platelet count drops to less than 100,000/µL or less than 50% of the base value, generally after 5-15 days of heparin treatment. For patients receiving heparin where the risk of HIT is thought to be greater than 1%, guidelines suggest that platelet counts are obtained every two or three days from day 4 to day 14 of therapy, or until heparin is stopped (Grade 2C). [5] The definitive diagnosis is made by performing a platelet activation factor analysis. The treatment of patients who develop HIT is to stop all heparin products, including catheter flushes and heparin coated catheters, and to initiate an alternative, nonheparin anticoagulanti, even when thrombosis is not clinically obvious. In patients with HIT with or without thrombosis, the use of lepirudin, argatroban or danaparoid is preferable to the continued use of heparin, LMWH, or either the initiation or continuation of a vitamin K antagonist (Grade 1C). [5] If a vitamin K antagonist has already been started when HIT is diagnosed, guidelines recommend that it be discontinued and that vitamin K be administered (Grade 2C). [5] Once HIT has been confirmed, vitamin K antagonists should not be started until the number of platelets has recovered to at least 150 x 109/L (Grade 1C), it should be started at low doses (i.e. 5 mg of warfarin), and it should be given simultaneously with a nonheparin anticoagulant for at least five days and until INR is within the target range (Grade 1C). [5] In patients with renal failure who have HIT and thrombosis, argatroban is preferable to other non-heparin anticoagulants (grade 2C). [5] Few patients with venous thromboembolism require large doses of heparin to achieve an optimally activated partial thromboplastin time (aPTT). The patients who need them have increased plasma concentrations of factor VIII and heparin-binding proteins. Increased factor VIII concentration causes dissociation between aPTT and plasma heparin values. APTT is suboptimal, but patients have adequate heparin levels at protamine titration. This usually occurs in patients with a concomitant inflammatory disease. Monitoring of the results of antifactor Xa assay in this situation is safe and effective and results in less escalation of the heparin dose when compared to monitoring with aPTT. Whenever a therapeutic level of aPTT cannot be achieved with large doses of UFH, either the determination of plasma heparin concentration or therapy with LMWH should be instituted. Set up.

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