**Appendix 2. Clinical Manifestation, Prevention, and Diagnosis of Deep Venous Thrombosis (DVT) / Pulmonary Embolism (PE)**

**1. Clinical manifestation of deep venous thrombosis (DVT) and pulmonary embolism (PE):**

* DVT is a disorder characterized by acute or chronic thrombus (blood clot) formation in deep veins anywhere in the body.
	+ Classic risk factors for DVT are described by Virchow’s triad: [1] alterations in blood flow such as venous stasis; [2] vascular endothelial injury such as surgery or crush injury; and [3] alterations in the constituents of the blood such as inherited or acquired hypercoagulable states.
	+ Common risk factors for DVT include immobility, recent surgery, and acquired hypercoagulability from sepsis, infection, hormone therapy, or malignancy.
	+ Acute DVTs, DVTs in larger caliber veins, and intracardiac thrombi are at higher risk for embolization. Embolization to the venae cavae, right heart, and pulmonary arteries can lead to PE.
* PE is a disorder characterized by embolization of thrombus (blood clot) from the right heart circulation into either or both pulmonary arteries.
	+ Potential complications of PE include pulmonary infarction, acute hypoxia, right heart failure, arrhythmia, or sudden death.

**2. Prevention of DVT:**

* Immobilization and venous stasis may be common in severely ill MDR-TB patients who have difficulty walking and spend most of the day in bed. These patients have a greater risk for DVT and should be given mechanical DVT prophylaxis with graduate **compression stockings** to prevent DVT. **Compression stockings** (at least knee-high, or thigh-high in higher risk patients) should be worn especially while the patient is in bed. If available, **sequential compression devices** may be used in addition to **compression stockings** while the patient is in bed.
* Medical DVT prophylaxis with **enoxaparin (also known as low molecular weight heparin or LMWH)** or **subcutaneous heparin** may be indicated in some patients. Clinicians must weigh the risk of GI bleeding against the benefit of prophylactic anticoagulants. Prophylactic doses of **enoxaparin** or **subcutaneous heparin** are shown in the table below.

**Prophylactic dosing of subcutaneous heparin and enoxaparin for the *prevention* of DVT/PE**

|  |  |  |
| --- | --- | --- |
| **Agent** | **Route** | **Dose** |
| Enoxaparin(low molecular weight heparin, LMWH) | Subcutaneous | 40 mg once daily (Dose at 30 mg once daily if mild renal insufficiency; contraindicated in renal failure;avoid in patients with hepatic impairment) |
| Heparin | Subcutaneous | 5,000 units every 8 hours (No dosage adjustment necessary for renal or hepatic impairment) |

 **3. Diagnosis of DVT:**

* The clinical presentation of DVT includes swelling, pain, tenderness, and/or heaviness of a leg or arm, dilated superficial veins, and bluish discoloration of the skin. Signs and symptoms can be subtle or masked by a larger body habitus or preexisting edema.
* Diagnosis of DVT generally requires a **doppler ultrasound**, the gold standard. If the patient's symptoms strongly suggest a DVT, but the ultrasound is negative, you can consider repeating a **full extremity doppler ultrasound** and/or **central vein doppler ultrasound** targeting the central chest veins or the inferior vena cava as indicated to look for thrombus missed on the initial scan, or repeating the ultrasound several days later.

**4. Diagnosis of PE:**

* The clinical presentation of PE includes hypoxia, sinus tachycardia or other tachyarrhythmia, and right heart strain on ECG on echocardiogram. Definitive diagnosis requires a **CT Pulmonary Angiogram**. A noncontrast CT Chest will not diagnose PE, it must use IV contrast and be protocoled to look specifically for PE to be diagnostic. Use caution in patients with or at risk for acute kidney injury before ordering a CT Pulmonary Angiogram. For these patients, consider prehydration with isotonic saline (0.9% NaCl) and avoid nephrotoxins. For patients on metformin, stop metformin 48 hours before and restart 48 hours after the CT is obtained to avoid inducing lactic acidosis.