

A KNOWN CASE OF VITILIGO VULGARIS WITH VENOUS THROMBOEMBOLISM AND PULMONARY ARTERIAL HYPERTENSION-PATIENT MONITORING AND PATIENT COUNSELING BY CLINICAL PHARMACIST

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ABSTRACT

Pulmonary embolism (PE) is a blockage of the lung's main artery or one of its branches by a substance that has traveled from elsewhere in the body through the bloodstream (embolism). PE results from a deep vein thrombosis (commonly a blood clot in a leg) that breaks off and migrates to the lung, a process termed venous thromboembolism (VTE). Pulmonary arterial hypertension is caused by narrowing or constriction of pulmonary arteries. Vitiligo vulgaris is loss of pigmentation of skin in patches. Present case was admitted with the chief complaint of acute pulmonary thromboembolism with severe pulmonary arterial hypertension and deep vein thrombosis. He was

previously suffering with vitiligo vulgaris. On treatment patient was found to be coherent, showing stable blood pressure, pulse rate and responding to therapy and discharged by giving discharge medication. He was counseled about the disease state and importance of medication adherence.

KEYWORDS: venous thromboembolism, vitiligo vulgaris, pulmonary arterial hypertension, deep vein thrombosis

DEFINITION

Formation of a thrombus within a vein is known as a venous thrombosis. If the thrombus breaks loose and travels through the blood system, it is known as an embolus.^[1] Venous

thrombi is defined as a feature enmeshed erythrocytes, tend to fragment, creating an embolus manifest mainly as DVT and PE.^[1]

Venous thromboembolism

The most common type of VTE is DVT, which occurs predominantly in the large veins in the leg.^[1,2] When part or all of a thrombus breaks away from the blood vessel wall, this thrombus is carried in the direction of blood flow towards the lungs and can block one of the arteries in the lung (pulmonary embolus). Patients with DVT are at risk of PE which can potentially be life-threatening.^[1,2]

Pulmonary arterial hypertension (PAH)

It is a progressive disease caused by narrowing or tightening (constriction) of the pulmonary arteries, which connect the right side of the heart to the lungs. By definition, PAH is characterised by an increase in mean pulmonary arterial pressure (PAP) to ≥ 25 mmHg at rest, and a mean primary capillary wedge pressure of ≤ 15 mmHg.^[3,4]

Vitiligo vulgaris

Vitiligo is a long term skin condition characterized by patches of the skin losing their pigment.^[5] The patches of skin affected become white and usually have sharp margins. The hair from the skin may also becomes white.^[5]

ETIOLOGY

Deep Vein Thrombosis

The frequent causes of DVT are due to augmentation of venous stasis due to immobilization or central venous obstruction. Immobility can be as transient as that occurring during a transcontinental airplane flight or that during an operation under general anesthesia. It can also be extended, as during hospitalization for pelvic, hip, or spinal surgery, or due to stroke or paraplegia. Individuals in these circumstances warrant surveillance, prophylaxis, and treatment if they develop DVT.^[6,7]

Pulmonary Embolism

PE is a potential cardiovascular emergency that occurs when a part of a thrombus, usually dislodged from a DVT (and then called an embolus), passes into the pulmonary circulation, occluding the pulmonary arteries.^[8]

Pulmonary Arterial Hypertension

The exact cause behind the development of Pulmonary arterial hypertension (PAH) remains unknown. PAH is recognized as a complex, multi-factorial condition involving numerous biochemical pathways and different cell types.^[9,10]

Vascular remodeling itself involves every layer of the vessel wall and is characterised by proliferative and obstructive changes involving many cell types, including endothelial cells, smooth muscle cells and fibroblasts.^[9] Inflammatory cells and platelets may also play a significant role in PAH.

Endothelial dysfunction results in chronically impaired production of vasoactive mediators, such as nitric oxide (NO) and prostacyclin, along with prolonged overexpression of vasoconstrictors, such as endothelin-1 (ET-1), which not only affect vascular tone but also promote vascular remodeling.^[9,11]

Vitiligo vulgaris

It is caused due to progressive decrease of melanocytes. Theories regarding destruction of melanocytes include autoimmune mechanisms, cytotoxic mechanisms, an intrinsic defect of melanocytes, oxidant-antioxidant mechanisms, and neural mechanisms.^[12]

RISK FACTORS

VTE is associated with cancer, trauma and surgery. Idiopathic cases occur where a patient has no clear exposing risk factor (i.e. no triggering event).^[13]

INCIDENCE AND PREVALENCE

Venous thromboembolic disease is a major problem worldwide, causing more than half a million deaths every year in the European Union (EU).^[14]

In 2007, it was estimated that approximately 1.1 million venous thromboembolic events occur each year across the EU encompassing:^[14]

- 1,118,742 total venous thromboembolic events
- 684,019 (61% of total) DVT events
- 434,723 (39% of total) PE events
- 543,454 (49% of total) VTE-related deaths

In the United States (US), the Surgeon General has stated that DVT and PE together affect an estimated 350,000–600,000 people each year,^[15] leading to an estimated 100,000–300,000 deaths annually.^[15,16]

The annual incidence rate of VTE (adjusted for age and sex) has been reported to be 1.17 per 1000 per year over a 25-year period in a US population-based study, or:^[17]

- DVT – 0.48 per 1000 population per year
- PE – 0.69 per 1000 population per year

It has also been noted that VTE is responsible for more than twice the number of deaths than those caused by AIDS, breast cancer, prostate cancer and road traffic accidents combined.^[14]

Although Pulmonary arterial hypertension (PAH) is a rare disease, with an estimated prevalence of 15-50 cases per million.^[18]

Vitiligo occurs worldwide with an overall prevalence of 1%. However, its incidence ranges from 0.1 to > 8.8%.^[19]

PATHOPHYSIOLOGY AND NATURAL HISTORY

Venous thromboembolism

Venous thrombi, composed predominately of red blood cells but also platelets and leukocytes bound together by fibrin, form in sites of vessel damage and areas of stagnant blood flow such as the valve pockets of the deep veins of the calf or thigh. Thrombi either remain in the peripheral veins, where they eventually undergo endogenous fibrinolysis and recanalization, or they embolize to the pulmonary arteries and cause PE.^[20]

Pulmonary arterial hypertension

It is unclear whether the various types of pulmonary arterial hypertension share a common pathogenesis.^[21,22] Three factors are thought to cause the increased pulmonary vascular resistance that characterizes this disease: vasoconstriction, remodeling of the pulmonary vessel wall, and thrombosis in situ.^[22] Advances in our understanding of the molecular mechanisms involved in this disease suggest that endothelial dysfunction plays a key role. Chronically impaired production of vasoactive mediators, such as nitric oxide and prostacyclin, along with prolonged overexpression of vasoconstrictors such as endothelin-1, not only affect vascular tone but also promote vascular remodeling. Thus, these substances represent logical pharmacologic targets.^[23-26]

Vitiligo vulgaris

Vitiligo is a multifactorial polygenic disorder with a complex pathogenesis. It is related to both genetic and nongenetic factors. Although several theories have been proposed about the pathogenesis of vitiligo, the precise cause remains unknown. Generally agreed upon principles are an absence of functional melanocytes in vitiligo skin and a loss of histochemically recognized melanocytes, owing to their destruction. However, the destruction is most likely a slow process resulting in a progressive decrease of melanocytes. Theories regarding destruction of melanocytes include autoimmune mechanisms, cytotoxic mechanisms, an intrinsic defect of melanocytes, oxidant-antioxidant mechanisms, and neural mechanisms.^[12]

SIGNS AND SYMPTOMS**Deep Venous Thrombosis**

History and clinical examination are not reliable ways of diagnosing DVT.^[27] Lower extremity DVT can be symptomatic or asymptomatic. Patients with lower extremity DVT often do not present with erythema, pain, warmth, swelling, or tenderness. Symptomatic patients with proximal DVT may present with lower extremity pain, calf tenderness, and lower extremity swelling.^[28,29] Homans' sign may be demonstrable in DVT. Most of these features lack specificity; hence clinical evaluation usually implies the need for further evaluation. The left leg is the commonest site for venous thrombosis in pregnancy³⁰ and in acute massive venous thrombosis. This may be due to compression of the left iliac vein by the right iliac artery (May-Thurner syndrome).^[31]

Pulmonary Embolism

The most common signs and symptoms of acute PE include dyspnea, tachypnea, and pleuritic chest pain. Other reported findings include apprehension, hemoptysis, cough, syncope, and tachycardia. Fever, gallop, accentuation of the pulmonary closure sound, or an S₃ and/or S₄ rales, and leg erythema or a palpable cord may also be found.^[32]

Pulmonary arterial hypertension

Most patients with PAH present with exertional dyspnea, which is indicative of an inability to increase pulmonary blood flow with exercise. Exertional chest pain, syncope, and edema are indications of more severely impaired right heart function.^[33]

Vitiligo vulgaris

Vitiligo lesions are characterized as follows:

- White or hypopigmented
- Usually well demarcated
- Round, oval, or linear in shape
- Borders may be convex
- Range from millimeters to centimeters in size
- Enlarge centrifugally over time at an unpredictable rate

Initial lesions occur most frequently on the hands, forearms, feet, and face, favoring a perioral and periocular distribution^[34]

DIAGNOSIS**Deep Venous Thrombosis**

The clinical examination of DVT is often unreliable; therefore, clinical decision rules (pretest probability scores) based on the patient's signs, symptoms, and risk factors have been developed to stratify patients into low, moderate, or high clinical probability (table 1).^[35-39] This approach helps to improve the effectiveness of diagnosing DVT and to limit the need for additional testing. A clinical prediction score has also been developed for upper extremity DVT using the presence of a pacemaker or a catheter or access device in the internal jugular or subclavian veins, localized pain, unilateral pitting edema, or another diagnosis at least as plausible as independent predictors for DVT.^[40]

Table 1. Pretest Probability of Deep Venous Thrombosis (Wells score)^[41]

Clinical Feature*	Score
Scoring	
Active cancer (treatment ongoing or within previous 6 months of palliative treatment)	1
Paralysis, paresis, or recent plaster immobilization of the lower extremities	1
Recently bedridden for >3 days or major surgery within 4 weeks	1
Localized tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling by >3 cm when compared with the asymptomatic leg (measured 10 cm below tibial tuberosity)	1

Pitting edema (greater in the symptomatic leg)	1
Collateral superficial veins (not varicose)	1
Alternative diagnosis as likely or more likely than that of deep-vein thrombosis	-2
Analysis	
High probability of DVT	≥ 3
Moderate probability of DVT	1 or 2
Low probability of DVT	≤ 0
Modified Score (adds 1 point if there is a previously documented DVT)	
Likely	≥ 2
Unlikely	≤ 1

*In patients with symptoms in both legs, the more symptomatic leg is used.
DVT, deep venous thrombosis

Diagnostic tests

- D-Dimer Testing^[42]
- Duplex Ultrasonography^[43]
- Impedance Plethysmography^[44]
- computed axial tomography venography.^[45]
- magnetic resonance venography (MRV) imaging^[46]

Pulmonary Embolism

Pretest probability scores or clinical decision rules have also been developed to aid in the diagnosis of acute PE.^[47](Table 2). There are a number of clinical decision rules available including the Wells rule and the Geneva score. Both have original and simplified versions.^[48]These clinical decision rules are similar to those employed for DVT; using signs, symptoms, and risk factors to calculate a low, moderate, or high pretest probability score. In a validation study using this approach in combination with a negative D-dimer test, only 0.5% of patients who were thought unlikely to have a PE later developed nonfatal VTE.^[49]

Table 2. Clinical Decision Rules (Pre-test Probability for Pulmonary Embolism)^[50]

Variable	Points
Clinical signs and symptoms of DVT (minimal leg swelling and pain with palpation of the deep veins)	3.0
Alternative diagnosis less likely than PE	3.0
Heart rate >100 bpm	1.5
Immobilization >3 days or surgery in the previous week	1.5
Previous PE or DVT	1.5
Hemoptysis	1.0
Malignancy (receiving treatment or treated in past 6 months or palliative)	1.0

Key: Low probability, <2.0; moderate probability, 2.0-6.0; high probability >6.0.
BPM, beats per minute; DVT, deep venous thrombosis; PE, pulmonary embolism.

Diagnostic tests

- D-Dimer Testing for PE^[51]
- Electrocardiography^[52]
- Chest Radiography^[53]
- Arterial Blood Gas Determination^[53]
- Computed Tomographic Pulmonary Angiography^[54]
- Ventilation-Perfusion Scanning^[55]
- Biomarkers (Troponins and Brain Natriuretic peptide)^[56]
- Echocardiography (Transthoracic and Transesophageal)^[57]
- Computed tomography pulmonary angiograph^[58]
- Magnetic Resonance Angiography^[59]

Pulmonary Arterial Hypertension

The diagnosis of PAH can be made on clinical grounds, based on a comprehensive evaluation that includes pulmonary function testing, connective tissue serology, echocardiography, complete cardiac catheterization, \dot{V}/\dot{Q} lung scanning, and/or pulmonary angiography.^[60]

Vitiligo vulgaris

Although the diagnosis of vitiligo generally is made on the basis of clinical findings, biopsy is occasionally helpful for differentiating vitiligo from other hypopigmentary disorders.

Microscopic examination of involved skin shows a complete absence of melanocytes in association with a total loss of epidermal pigmentation. Superficial perivascular and perifollicular lymphocytic infiltrates may be observed at the margin of vitiliginous lesions, consistent with a cell-mediated process destroying melanocytes.

Other documented histologic findings include the following:

- Degenerative changes in keratinocytes and melanocytes in the border lesions and adjacent skin
- Increased numbers of Langerhans cells
- Epidermal vacuolization
- Thickening of the basement membrane

Loss of pigment and melanocytes in the epidermis is highlighted by Fontana-Masson staining and immunohistochemistry testing.^[61,62]

TREATMENT

Venous thromboembolism

The main goals of treatment for DVT include prevention of PE, the PTS, and recurrent thrombosis. Once VTE is suspected, anticoagulation should be started immediately unless there is a contraindication.

MEDICAL MANAGEMENT

Anticoagulation

Initial therapy may include unfractionated heparin (UFH), low-molecular-weight heparin (LMWH), or fondaparinux followed by an oral anticoagulant (vitamin K antagonist [VKA]). Early initiation of a VKA on the first day of parenteral therapy is advised.^[63]

Risk stratification is essential for managing acute PE. The clinical examination (including blood pressure, heart rate, and oxygen saturation) biomarkers (troponin, BNP), and echocardiography to assess the right ventricle and PE size should all be used to assist in the acute management of PE.⁶³ If the patient is normotensive and the right ventricle size and function are normal, standard anticoagulation is advised. If the patient is normotensive, but the right ventricle is abnormal and biomarkers are elevated, treatment is more controversial. For the patient who is hemodynamically unstable, thrombolysis or pulmonary embolectomy should be considered.^[63,64]

New Oral Anticoagulants

Dabigatran (direct thrombin inhibitor) and rivaroxaban (factor Xa inhibitor) have been studied extensively and shown to be non-inferior to VKA for treatment of VTE.^[65] Rivaroxaban has been approved by the FDA for use in the prevention of VTE for the patient undergoing total hip or knee replacement surgery. It has also been approved for the treatment of DVT and PE based on clinical trials. In studies comparing rivaroxaban to enoxaparin and a VKA, rivaroxaban was as effective for treatment of VTE. The drug is given orally once daily and is contraindicated in patients with renal insufficiency. The major side effect observed with rivaroxaban is bleeding, similar to other anticoagulants.^[66]

Thrombolytic Therapy

Thrombolytic therapy for DVT may be beneficial in selected patients, and although it can be administered systemically, local infusion under catheter directed therapy (CDT) is preferred. Both routes carry an increased risk of hemorrhage compared to standard anticoagulation.⁶⁷ Streptokinase, administered as a 250,000 IU loading dose followed by 100,000 IU/hr for 24 hours and tissue plasminogen activator (rtPA) given as a 100-mg infusion over 2 hours are the current agents approved by the FDA. The ACCP guidelines recommend systemic thrombolytic therapy using an agent with a short infusion time in patients who are hemodynamically unstable. Bleeding remains the most serious complication of thrombolytic therapy. Local administration of these agents via catheter-directed therapy is recommended over a pulmonary artery catheter. The risk of intracranial bleeding is 1% to 2%.^[63]

SURGICAL/MECHANICAL INTERVENTION

- Pulmonary Embolectomy^[63]
- Vena Caval Interruption^[68]

COMPRESSION STOCKINGS

Damage to the venous valves from DVT can lead to venous hypertension and result in the development of PTS characterized by edema; skin changes, including increased pigmentation and lipodermatosclerosis; pain; and, in severe cases, venous stasis ulceration. The incidence of PTS has been drastically reduced with the use of compression stockings. Current ACCP guidelines recommend the use of compression stockings at a pressure of 30 mmHg to 40 mmHg

for 2 years following an acute episode of DVT. The American College of Physicians and the American Academy of Family Physicians recommend use for 1 year.^[63,69]

Pulmonary arterial hypertension

CONVENTIONAL OR SUPPORTIVE THERAPY

A range of treatment approaches have been shown to provide some degree of symptomatic benefit to PAH patients. However, there is no evidence that they have an effect on the disease process or prognosis. Such measures include:

- **Oxygen:** For patients with dyspnoea associated with PAH, supplemental oxygen provides symptomatic relief and improves patient comfort, although there is no consistent evidence supporting any long-term benefit. It is generally considered important to maintain oxygen saturation above 90% at all times, and oxygen may be indicated in some patients^[69-72]
- **Anticoagulants:** clinical data supportive of the use of anticoagulant therapy in PAH are limited; however, improved survival has been reported with oral anticoagulation in patients with idiopathic PAH (IPAH),^[71,72] and because of high risk of in situ thrombosis within the small pulmonary arteries, there is a rationale for the use of oral anticoagulants in PAH patients.^[70]
- **Diuretics:** there are no randomised controlled trials (RCTs) of diuretics in PAH, however clinical experience shows clear symptomatic benefit in fluid-overloaded patients with decompensated right heart failure associated with PAH^[71,72]
- **Calcium channel blockers (CCBs):** A positive vasoreactive response indicating potential suitability for CCB therapy is shown by around 10% of patients,⁷⁰ and approximately 7% of these patients have a sustained response.^[73,74] Patients who respond to vasoreactivity testing and are subsequently treated with CCBs need regular assessment and repeat testing, as the vasoreactivity status may change.^[72,75]

ADVANCED THERAPY (ALSO TERMED PAH-SPECIFIC THERAPY)

PAH-specific therapies have been developed specifically to target one of three major pathways known to be involved in the development of PAH^[74]

- Endothelin receptor antagonists (ERAs)^[74]
- Prostacyclin therapy^[76]
- Phosphodiesterase-5 (PDE-5) inhibitors^[77,78]

Surgical intervention

- Balloon atrial septostomy^[70,73]

Vitiligo vulgaris

Nonsurgical treatments

- Systemic phototherapy: Induces cosmetically satisfactory repigmentation in up to 70% of patients with early or localized disease^[79]
- Laser therapy: Effective on limited, stable patches of vitiligo
- Steroid therapy: Systemic steroids (prednisone) have been used, although prolonged use and their toxicity are undesirable
- Topical therapies
- Depigmentation therapy: If vitiligo is widespread and attempts at repigmentation have not produced satisfactory results, depigmentation may be attempted in selected patients
- Micropigmentation: Tattooing can be used to repigment depigmented skin in dark-skinned individuals^[79,80]
- Surgery
- The basic types of repigmentation surgery are as follow :
- Noncultured epidermal suspensions
- Thin dermoepidermal grafts
- Suction epidermal grafting
- Punch minigrafting
- Cultured epidermis with melanocytes or cultured melanocyte suspensions^[81,82]

CASE STUDY

A patient of 38 years old male was admitted in King George Hospital, Visakhapatnam, Andhra Pradesh, India with a complaint of breathlessness from before night, Pedal edema (bilateral) since 10days.

History of present illness includes patient was apparently normal 10 days ago, complaint started as pedaledema(bilateral) since 10 days, breathlessness from before night. History of paroxysmal nocturnal dyspnea, cough with sputum. No history of chest pain, hemoptysis, palpitations.

There is a past medical history of Pulmonary thromboembolism. Patient is on Tab.acitrom 2mg irregularly, is an anti-coagulant that help to prevent blood clots.

Personal history of the patient includes mixed diet, normal appetite, smoker and alcoholic.

Inj. Elaxim (tenecteplase) was given on the date of admission.

On admission his body temperature was normal, BP 150/70mmHg, PR 80beats/min.

Laboratory investigations included as INR 2.38, Hb 10.0gm%, total leucocyte count 9700cells, differential leukocyte count-polymorphs 57% lymphocytes 39% basophils 4%, ESR 30mm/1st hour, Random blood sugar 147mg%, Serum sodium 141 mmol/lit, Serum potassium 3.25 mmol/lit, Serum creatinine 0.9 mg/dl.

Finally diagnosed as Acute Pulmonary thromboembolism with Severe Pulmonary arterial hypertension and Deep vein thrombosis.

THERAPY

DAY	TREATMENT
On day 1	Inj. Elaxim (tenecteplase) 40mg Inj. Lasix (furosemide) 1amp IV BD Tab. Pulmoday (sildenafil) 20mg TID Tab. Enam (enalapril) 2.5 mg OD
On day 2	Inj. Clexane (enoxaparin sodium) SC BD Tab. Aspirin 75mg OD Tab. Pulmoday 20mg TID Tab. Enam 2.5mg OD Inj. Lasix 1amp IV BD Tab. Pantop (pantoprazole) 40mg OD Tab. Acitrom (acenocoumarol) 2mg OD
On day 3	Inj. Clexane (enoxaparin sodium) SC BD Tab. Aspirin 75mg OD Tab. Pulmoday 20mg TID Inj. Lasix 1amp IV BD Tab. Pantop (pantoprazole) 40mg OD Tab. Acitrom (acenocoumarol) 2mg OD
On day 4	Along with the above drugs Tab. losar (losartan) 25mg OD is added
On day 5	Inj. Clexane SC BD Tab. Pulmoday 20mg TID Inj. Lasix 1amp IV BD Tab. Pantop 40mg OD Tab. Acitrom 2mg OD Tab. losar 25mg OD
On day 6	Inj. Clexane SC BD Tab. Pulmoday 20mg TID Inj. Lasix 1amp IV BD Tab. Pantop 40mg OD Tab. Acitrom 2mg OD Heparin 5000 IU IV QID
On day 7	Inj. Clexane SC BD Tab. Pulmoday 20mg TID Tab. Pantop 40mg OD

	Tab. Acitrom 2mg OD
On day 8	Inj. Clexane SC BD Tab. Pulmoday 20mg TID Tab. Pantop 40mg OD Tab. Acitrom 2mg OD Asthalin(salbutamol) nebulisation stat Tab. Thyroxine 200mg OD
On day 9	Inj. Clexane SC BD Tab. Pulmoday 20mg TID Tab. Pantop 40mg OD Tab. Acitrom 2mg OD Tab.CPM(chlorpheniramine) Liquid paraffin BD
On day 10	Inj. Clexane SC BD Tab. Pulmoday 20mg TID Tab. Pantop 40mg OD Tab. Acitrom 2mg OD
On day 11	Tab. Pulmoday 20mg TID Tab. Pantop 40mg OD Tab. Acitrom 2mg OD
On day 12	Tab. Pulmoday 20mg TID Tab. Pantop 40mg OD
On day 13	Tab. Pulmoday 20mg TID Tab. Pantop 40mg OD Tab. Acitrom 2mg OD
On day 14,15, 16, 17 and 18	Tab. Pulmoday 20mg TID Tab. Pantop 40mg OD

After admission the patient was first given with Inj.Elaxim 40mg, a tissue plasminogen activator given as a thrombolytic drug; Inj.lasix 1amp IV BD, is a loop diuretic used to treat fluid build-up; Tab.Pulmoday 20mg TID, is a Phosphodiesterase Type 5 Inhibitor used to treat pulmonary arterial hypertension; Tab.Enam 2.5mg OD, is a ACE inhibitor given to control blood pressure.

On 1st day BP was monitored frequently. Results shown as 120/80mmHg at 2:30 PM, 110/80 mmHg at 4:30 PM, 110/80 mmHg at 8:30 PM,110/80 mmHg at 10:00 PM.

On 2nd day BP was 110/80mmHg, PR 91beats/min. The patient was given with Inj.Clexane 0.6cc SC BD, given as anti-coagulant; Tab.Aspirin 75mg OD, is a platelet aggregation inhibitor given to treat thromboembolic disorder; Tab.Pulmoday 20mg TID, Tab.Enam 2.5mg OD, Inj.Lasix 1 amp IV BD, Tab.Pantop 40mg OD, is a proton pump inhibitor used to inhibit excess gastric acid secretion; Tab.Acitrom 2mg OD, is an anti-coagulant that help to prevent blood clots.

On 3rd day BP was 110/70mmHg, PR 90beats/min. Patient was conscious so given with Inj.Clexane, Tab.Aspirin, Tab.Pulmoday, Inj.Lasix, Tab.Pantop, Tab.Acitron. Tab.Enam was excluded as BP was low.

2D echo on 3rd day shown Interventricular septal end diastole and end systole(IVSD)-1.4cm, left ventricle(LV)-4.5x3.1cm, left ventricular posterior wall end diastole and end systole(LVPWD)-1.2cm, ejection fraction(EF)-60%, fractional shortening(FS)-31%

On 4th day patient complains of dry cough. His PR was 90beats/min, BP 130/40 mmHg so given with Inj.Clexane, Tab.Aspirin, Tab.Pulmoday, Inj.Lasix, Tab.Pantop, Tab.Acitrom. In addition Tab.Losar 25mg OD is added as BP was increased and to decrease risk of stroke.

On 5th day BP was 130/40mmHg, PR 82beats/min, coagulation profile shown factor V deficiency so Tab.Aspirin was stopped and remaining Inj.Clexane, Tab.Pulmoday, Inj.Lasix, Tab.Pantop, Tab.Acitrom and Tab.Losar was continued.

On 6th day BP was 130/80 mmHg, PR 90beats/min. Inj.Clexane, Tab.Pulmoday, Inj.Lasix, Tab.Pantop, Tab.Acitrom were continued in addition Heparin 5000 IU IV QID is given. Color Doppler test was performed on 6th day whose findings were:

- Color Doppler findings of arterial system of both lower limbs:
 - ✓ Flow visible and spectral pattern was observed.
 - ✓ No findings of stress or thickening

Impression-Normal study

- Color Doppler test of venous system of both lower limbs:

Right limb:

- ✓ Sapheno femoral junction(SFJ) competent.
- ✓ Splenoporto venography(SPV) thrombosed.
- ✓ Thrombosis noted in lower Superficial femoral vein(SFV) and Pulmonary vein(PV).

Impression-Acute anterior in SFV and PVs

Left limb:

- ✓ Sapheno femoral junction(SFJ) and Saphenopopliteal junction(SPJ) are thrombosed.
- ✓ Subcentre on drug noted.
- ✓ Thrombosis noted common femoral vein(CFV), SFV and PV.
- ✓ No flow detected.

Impression: Sub acute Deep vein thrombosis involving above mentioned ones.

On 7th day BP was 130/80mmHg, PR 88beats/min regular. So Inj.Clexane, Tab.Pulmoday, Tab.Pantop, Tab.Acitrom continued excluding Inj.Lasix and Heparin.

On 8th day BP was 140/80mmHg, PR 88beats/min regular, Bilateral occasional wheeze was seen. Inj.Clexane, Tab.Pulmoday, Tab.Pantop, Tab.Acitrom along with Asthalin nebulisation stat, is a bronchodilator given to decrease breathlessness; Tab.Thyroxine 200mg OD. Advised for T₃,T₄ and TSH examination. Patient complains of itching all over the body so sent for examination to dermatology ward.

On 9th day patient was conscious and coherent, PR 78beats/min, BP 120/80mmHg. Dermatological examination shown depigmented skin over extremities and to oral mucosa, dense over the lower limbs. He was a known case of Vitiligo vulgaris, took some homeopathic treatment. So now given with Tab.CPM OD, is an anti-histamine given to reduce itching; and Liquid paraffin BD to treat Vitiligo vulgaris. Along with these two drugs Inj.Clexane, Tab.Pulmoday, Tab.Pantop, Tab.Acitron was continued excluding Asthalin nebulisation and Tab.Thyroxine as thyroid levels were found normal.

On 10th day patient was conscious and coherent, PR was 82beats/min, BP 150/100mmHg. Inj.Clexane, Tab.Pulmoday, Tab.Pantop, Tab.Acitron was continued. PT/INR value shown >100 and temperature was above 100°F so advised to stop Inj.Clexane 0.6cc and Tab.Acitrom.

On 11th day BP was 140/90mmHg, PR 72beats/min. So given with Tab.Pulmoday, Tab.Pantop, Tab.Acitrom excluding Inj.Clexane.

On 12th day patient was conscious and coherent, PR 72beats/min, BP 140/90mmHg. Tab.Pulmoday, Tab.Pantop, excluding Tab.Acitrom. Advised for 2D echo.

On 13th day patient was conscious and coherent, PR 74beats/min, BP 140/110mmHg. Tab.Pulmoday, Tab.Pantop, continued along with Tab. Acitrom 2mg OD. 2D echo findings shown Interventricular septal end diastole and end systole (IVSD)-1.7cm, left ventricle-4.0*2.5cm, left ventricular posterior wall end diastole and end systole (LVPWD)-1.3cm, ejection fraction (EF)-67%, fractional shortening (FS)-36%

On 14th day patient was conscious and coherent, PR 82beats/min, BP 150/100mmHg.

On 15th day patient was conscious and coherent, PR 78beats/min, BP 150/100 mm Hg.

On 16th day patient was conscious and coherent, PR 78beats/min, BP 140/100mmHg.

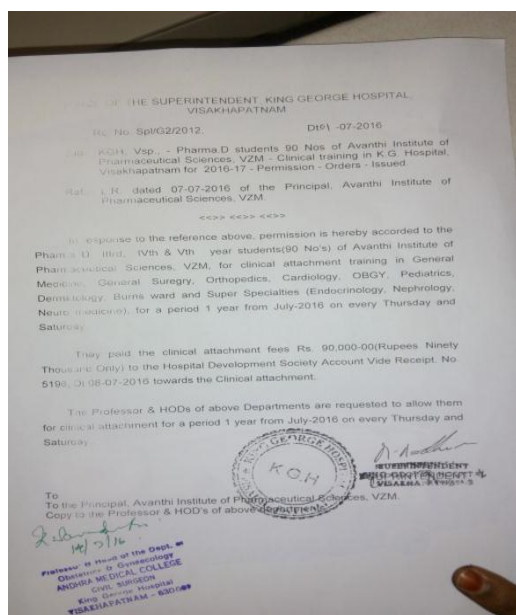
On 17th day patient was conscious and coherent and temperature was 98.6⁰F,BP 130/90mmHg, PR 72beats/min..

On 18th day patient was conscious and coherent, PR 82beats/min, BP 130/90mmHg.

Above five days Tab.Pulmoday, Tab.Pantop were continued. As patient was stable, he was discharged.

Discharge medication :

- Tab.Acitrom - 2mg OD
- Tab.Losartan - 50mg BD
- Tab.Pulmoday - 20mg TID
- Tab.CPM - 4mg OD
- Liquid paraffin - BD
- Tab.Pantop - 40mg BD



AUTHORIZATION FOR PUBLICATION OF CASE STUDY

I, P. Sai nadh give K.Phanimala,V.Uma Sankar, S.Dhana lakshmi,V.Pavani and Avanthi Institute of Pharmaceutical Sciences, permission to publish, reproduce, and distribute, the attached Case Study, regarding A known case of Vitiligo vulgaris with Venous Thromboembolism and Pulmonary arterial hypertension-Patient monitoring and patient counseling by Clinical Pharmacist. I am aware that the Case Study does NOT mention my name or address, but it does reflect my medical care, gender, age and medical history.

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Patient's Name: <u>P. Sai nadh</u>	
Patient's Address: <u>Pilla vavi street, Anakapalli, Visakhapatnam</u>	
Patient/Representative's Signature: <u>[Signature]</u>	Date: <u>25/12/2016</u>

CONCLUSION

Pulmonary embolism is a blockage in one of the pulmonary arteries in lungs. In most cases, pulmonary embolism is caused by blood clots that travel to the lungs from the legs or, rarely, other parts of the body (deep vein thrombosis). Pulmonary embolism can also lead to pulmonary hypertension, a condition in which the blood pressure in lungs and in the right side of the heart is too high. The present case is a typical hypertensive case with PTE and DVT. The patient has a co-morbidity of Vitiligo vulgaris. But the effective treatment with Tab.Acitrom, Tab.Losartan, Tab.Pulmoday - 20mg TID and Tab.CPM patient showed positive prognosis after a long duration of therapy for 18 days. As a clinical pharmacist we have done the TDM and drug utilisation review for the patient for the occurrence of any drug related side effects or drug interactions and for positive prognosis of the patient with the collaborative efforts of physician,clinical pharmacist and nursing staff. Patient exhibited positive prognosis and discharged with medication Tab.Acitrom - 2mg OD, Tab.Losartan - 50mg BD, Tab.Pulmoday - 20mg TID, Tab.CPM - 4mg OD,Liquid paraffin – BD,Tab.Pantop - 40mg BD. Patient was counselled about the disease, proper use of medicatios,diet to be taken and life style modifications.

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