Deep Venous Thrombosis leading to Pulmonary Embolism: A Cause for Acute Respiratory Distress

Dr. Pooja Gupta¹, Dr. Kanika Kinra², Dr Manoj Gupta³, Dr Mahesh Gupta⁴

¹MD (Medicine) Senior Consultant Sgl Charitable Hospital Jalandhar, Punjab
²DNB (Medicine) Std. SGL Charitable Hospital Jalandhar, Punjab
³MS, M.Ch (Urology) Senior Consultant Sgl Charitable Hospital Jalandhar, Punjab
⁴MS, DNAS, FMAS Professor & Head Department Of Surgery
Rama Medical College & Hospital, Mandhana, Kanpur, U.P

Corresponding Author: Dr. Pooja Gupta

Abstract: Venous thrombosis, comprising deep vein thrombosis (DVT) and pulmonary embolism (PE), occurs with an incidence of approximately 1 per 1000 annually in adult populations. We report a similar case of DVT & PE and conclude that timely intervention of DVT with anticoagulants is essential to reduce the incidence of pulmonary embolism and its associated mortality.

Keywords: Deep vein thrombosis, Pulmonary embolism, venous thrombo-embolism

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I. Introduction

Venous thrombosis is comprised of deep vein thrombosis (DVT) and pulmonary embolism (PE) and it occurs with an incidence of approximately 1 per 1000 per year. It is slightly higher in men than women. Acute pulmonary embolism (APE) is the most serious clinical presentation of venous thrombo-embolism (VTE). The common cause of sudden death (SD) in deep venous thrombosis (DVT) is fatal pulmonary embolism (PE). The other major outcomes of venous thrombosis are recurrence, post-thrombotic syndrome and major bleeding due to anticoagulation. We are presenting one such case of deep venous thrombosis who had pulmonary embolism and presented with acute respiratory distress.

II. Case Report

48 year male with history of diabetes and hypertension for the last 2 years presented with sudden episode of severe breathlessness. He had left leg pain and swelling since 4 days and it was not associated with chest pain, fever or vomiting. On examination left lower limb temperature was raised and there was a localized swelling. A provisional diagnosis of deep venous thrombosis and pulmonary embolism was made on clinical grounds which was followed by investigations including complete blood counts, renal function tests, liver function tests. These investigations were within the normal range. Colour Doppler for both lower limbs showed popliteal vein thrombosis(Fig-1). Chest X-Ray showed only hilar prominence. Electrocardiogram(ECG) revealed typical changes of S wave in lead 1 and Q wave in lead 3 along with T wave inversion in lead 3 (Fig 2). Echocardiography showed dilated right atria and right ventricle with right ventricle free wall hypokinesia and ejection fraction of 68% with moderate TR (Fig 3&4). Patient was managed by thrombolysis with streptokinase initially and he recovered well with the conservative treatment.

III. Discussion

Deep vein thrombosis (DVT) is the formation of a blood clot within a deep vein predominantly in the legs. Detachment of a clot that travels to the lungs may cause pulmonary embolism (PE) which is a potentially life-threatening complication. Collectively DVT and PE constitute a single disease process termed as venous thromboembolism (VTE). Risk factors that increase the risk of VTE include deficiencies of protein C, protein S, and anti-thrombin and mutations in the factor V and prothrombin genes. Very few cases of DVT with multiple high risk factors have been reported. Non-cardiogenic pulmonary edema or Acute Respiratory Distress Syndrome occurs in a wide variety of clinical settings, such as hypovolemic shock, major trauma, and septicemia. However it was described as a complication of classic PE by Windebank and Moran in 1973. Electrocardiographic changes in acute cor pulmonale (acute pressure and volume overload of the right ventricle because of pulmonary hypertension) are prominent S wave in lead I, Q wave and inverted T wave in lead III.
This finding is present in 15% to 25% of patients diagnosed with pulmonary embolism. In addition to this, 'McConnell sign' (normally contracting right ventricular apex associated to a severe hypokinesia of the mid-free wall) on echocardiography is a distinctive feature of acute pulmonary embolism. These findings were also evident in our patient suggesting acute pulmonary embolism.

IV. Conclusion

Early diagnosis and treatment of DVT with anticoagulants has been demonstrated to reduce the incidence of pulmonary embolism and its associated mortality along with relief of acute symptoms in the leg. However failure to diagnose and treat DVT can lead to postphlebitic syndrome, chronic pulmonary thromboembolic disease, and pulmonary hypertension.

References
