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Case Report

Case Report on Supporting Identification and Treatment of the Next Patient with Pulmonary Embolism through AI Risk Assessment Early Warning and VTE Clinical Decision Support System

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Abstract

Venous thromboembolism (VTE) is a grave medical condition comprising deep vein thrombosis (DVT) and pulmonary embolism (PE), both of which pose significant threats to patient health and wellbeing. While DVT primarily affects deep veins, particularly in the lower limbs, PE arises when a blood clot dislodges and travels to the pulmonary artery, potentially leading to fatal consequences. The clinical ramifications of VTE are profound, as it ranks among the leading causes of mortality and morbidity worldwide. PE, a severe complication of VTE, manifests with symptoms such as dyspnea, chest pain and haemoptysis, often resulting in life-threatening situations. Conversely, DVT can lead to chronic pain, swelling and even limb dysfunction in severe cases. Given the gravity of VTE, timely diagnosis and prevention are paramount. Consequently, the exploration of early warning systems and clinical decision support mechanisms for VTE holds immense significance in enhancing patient survival rates and mitigating associated complications. In this context, our critical care department, with support from the Science and Technology Bureau, has developed a VTE clinical decision support system leveraging Artificial Intelligence (AI). The system, currently in the testing phase, aims to provide a secure and reliable platform for early diagnosis and treatment decision-making. By harnessing AI technology, we endeavour to improve the accuracy of VTE risk assessment and streamline clinical management protocols, ultimately advancing patient outcomes. The most important part of this system is to input standardized VTE patients, and this case report is one of the standardized patients that is selected and input for the system to automatically recognize and learn.

Keywords: *Venous Thromboembolism (VTE); Deep Vein Thrombosis (DVT); Pulmonary Embolism (PE); Artificial Intelligence (AI)*

Introduction

Venous thromboembolism (VTE) is a serious medical condition that includes deep vein thrombosis (DVT) and pulmonary embolism (PE). DVT occurs in a vein deep in the body, usually in the lower limbs and when a blood clot breaks off and travels into the pulmonary artery, it can cause PE, which can be fatal [1].

The clinical significance of VTE is significant as it is one of the leading causes of death and disability. PE is one of the most serious complications of VTE, which can lead to difficulty breathing, chest pain and even death. And even smaller PEs can cause serious consequences in a short period. On the other hand, DVT can cause chronic pain, swelling and ulcers in the lower limbs. In severe cases, it may also lead to loss of limb function [2].

Due to the seriousness of VTE, prevention and timely diagnosis are crucial. Therefore, studying VTE risk assessment early warning systems and clinical decision support systems is of great significance to improve the survival rate of VTE patients and reduce the occurrence of related complications.

With the support of the Science and Technology Bureau, our critical care department has developed a VTE clinical decision support system using Artificial intelligence (AI) as a support method. At present, with the joint efforts of all members of the research team, we have initially built a safe and reliable private cloud VTE intelligent decision support platform based on artificial intelligence. The entire platform is in the source code testing stage. The main content of the test is to desensitize the electronic medical records we collected, eliminate sensitive and non-research-related private information, and conduct structured annotation to improve the accuracy of the data source. At present, after the platform has started testing and operation, it is undergoing cross-validation and other processes and is expected to meet the standards of clinical medical interns in our hospital. In other words, the platform itself can not only be analysed through different levels of linguistics such as syntax, semantics and pragmatics but can also be composed of five modules including clinical knowledge base, word segmentation, annotation, named entity recognition and semantic association extraction. Content that can be identified and analysed by AI [3,4].

The most important part of this system is to input standardized VTE patients, and this case report is one of the standardized patients that is selected and input for the system to automatically recognize and learn.

Case Report

Patient description

Patient: Mr A, male, 74 years old, height 172 cm, weight 67.5 kg, BMI 22.8, married.

Main complaint: Coughing blood-streaked phlegm for 4 days and shortness of breath for 10 hours.

History of current illness: The patient began to cough and cough up blood-streaked sputum 4 days ago. He went to the outpatient department of our hospital for treatment and a chest CT showed a consolidation in the lower lobe of the left lung. He was given oral anti-infective treatment with levofloxacin. After getting up at 3 a.m. this morning, he developed shortness of breath, dizziness and discomfort, self-measured blood pressure 100/50 mmhg, no fever, chills, chills, amaurosis, no headache, nausea and vomiting, limb weakness, no black stools, bloody stools, no abdominal pain, abdominal distension diarrhea, no chest tightness, chest pain and other discomforts, so he went to the emergency department of our hospital. Upon diagnosis, chest CT showed embolism in the main trunks of both pulmonary arteries and its multiple branches. The infection lesions in the lower lobe of the left lung have decreased absorption compared with before, and the infection lesions in the lower lobe of the right lung have increased compared with before. A cardiac colour ultrasound examination

showed that the right heart and left atrium were enlarged, and the tricuspid valve had moderate regurgitation. D-dimer 22.49 μg/mL FEU↑; (Radio) troponin T measurement: 0.017 ug/L, blood gas analysis: oxygen partial pressure 41 mmhg, oxygen concentration 29%, after emergency subcutaneous injection of 0.6 ml of Kesai Send it to our department. The patient is currently in good spirits, with normal physical strength, normal appetite, normal sleep, no significant change in weight, normal stool, and normal urination. Admitted to the hospital for further examination and treatment.

Past medical history: Vaccination history: Vaccinations unknown. Allergy history: None. The patient's past physical condition: average health. Disease history: Have a history of hypertension, usually take bisoprolol, amlodipine, telmisartan, hydrochlorothiazide to control blood pressure, systolic blood pressure fluctuates between 150 - 160 mmHg, have a history of diabetes, take empagliflozin, gliclazide to control blood sugar, denying the history of "nephritis", "blood disease", "coronary heart disease", "heart disease", "chronic bronchitis", "cerebral infarction", etc. History of infectious diseases: Denied history of infectious diseases such as hepatitis, tuberculosis, and malaria. Surgical history: Femoral head replacement was performed for left femoral head necrosis. History of trauma: Denies history of trauma. History of blood transfusion: Denied history of blood transfusion. Poisoning history: Denies poisoning history. Long-term medication use: The patient denied any history of long-term medication use. Potentially addictive drugs: None. Medical history related to this disease: None. Others: None.

Personal history: Born in the place of origin, grew up in the place of origin, and has lived in the area for 74 years. The place of origin of the workplace has no history of living in an epidemic area, no history of metallurgical travel, no history of drinking a small amount of alcohol, no history of smoking and no history of exposure to radioactive substances or poisons.

Marital and childbearing history: Marital status: married, childbearing: with children, children in good health.

Family history: There are no cancer patients in the family. No history of infectious diseases or genetics in the family.

Physical examination: consciousness: Clear, body temperature: 36.4°C, pulse: 99 beats/min, blood pressure: 99/65 mmHg (no antihypertensive drugs were taken today), respiration: 25 times/min.

Specialist examination:

1. Lungs: respiratory movement: breathing freely, percussion sounds: voiceless, breath sounds: voiceless breath sounds, rales: no rales were heard.
2. Heart: Heart rate: 99 beats/min, heart rhythm: regular and regular, heart sounds: normal heart sounds, murmurs: no murmurs were heard in the auscultation area of each valve.
3. Blood vessels: Peripheral vascular signs: No Duroziez double murmur or capillary pulsatility.

Laboratory examination: There is no swelling or tenderness in the superficial lymph nodes of the whole body. Laboratory examination: On February 25, 2024, uric acid in our hospital was 8.7 mmol/l, lactic acid in two items of glucose metabolism was 5.4 mmol/l, and B-hydroxybutyric acid was 1.28 mmol/l. Coagulation function Fbg5.78 g/l, APTT29.4s, D dimer 22.49 ug/ml, PCT0.19 ng/ml, CRP162 mg/l, blood routine WBC14.94*10e9/l, N90.6%, HB155 g/l, PLT258*10e9/l, blood gas analysis (oxygen concentration 29%) PH7.44, PCO_2 34.3 mmHg, PO_2 41.3 mmHg. Two items of myocardial injury: troponin T0.14 ug/l, PBNP461pg/ml, liver function, cardiac enzymes and creatinine showed no obvious abnormalities.

Special examination: Pulmonary artery CT in our hospital on February 25, 2024. 1. Embolism of the main trunk of both pulmonary arteries and its multiple branches. 2. The infection lesions in the lower lobe of the left lung have decreased absorption compared with

before, and the infection lesions in the lower lobe of the right lung have increased compared with before. 3. Small calcified nodule in the posterior apical segment of the upper lobe of the left lung, same as before. 4. A small amount of chronic infection/fibrous lesions in the middle lobe of the right lung and the lingual segment of the upper lobe of the left lung, roughly the same as before. 5. A small amount of secretions in the trachea, slightly more than before. 6. The pleura on both sides was slightly thickened, and the small amount of pleural effusion on the left side was reduced than before. 7. The right 2^{nd} anterior rib and the 4^{th} and 7^{th} ribs on the left side are partially distorted. 8. Obvious bone hyperplasia in the thoracic vertebrae and diffuse idiopathic bone hypertrophy has not been eliminated. 9. Aorta and coronary artery sclerosis, same as above. Bedside ultrasound: The main trunks of the common femoral vein, proximal deep femoral vein, superficial femoral vein, popliteal vein, posterior tibial vein, peroneal vein, anterior tibial vein, and intermuscular vein of both lower limbs had smooth blood flow, and no thrombosis was found. There was no dilation in the roots of the double great saphenous veins, and the saphenofemoral vein valves functioned well. There was no dilation in the roots of the small saphenous veins, and the venous valves functioned well. The right heart and left atrium were enlarged, the tricuspid valve had moderate regurgitation, the left ventricular systolic function was normal, and there was no obvious effusion in the pericardial cavity.

Diagnosis on admission: 1. Pulmonary embolism or acute pulmonary heart disease (high risk); 2. Pneumonia; 3. Acute respiratory failure (type I respiratory failure); 4. Hypertension; 5. Type II diabetes.

Diagnose based on [5,6]

Most patients were suspected of having pulmonary embolism due to dyspnea, chest pain, presyncope, syncope, cough, and haemoptysis. The patient in this case had typical cough, haemoptysis and mild dyspnea, but many patients' symptoms are not obvious and lack specificity; therefore, timely laboratory examination is very important:

1. Arterial blood gas analysis: Hypoxemia, hypocapnia, increased alveolar-arterial blood oxygen gradient and respiratory alkalosis may occur. In this case, the blood gas analysis (oxygen concentration 29%) showed PH7.44 and PCO_2 34.3 mmHg, PO_2 41.3 mmHg;
2. Plasma D-dimer: Because tumours, inflammation, haemorrhage, trauma and surgery can all cause elevated levels, the diagnostic value is not high. However, if plasma D-dimer is negative, PTE and DVT can be ruled out, and this patient D dimer 22.49 ug/ml;
3. CT pulmonary angiography: The direct sign is a low-density filling defect in the pulmonary artery, the "track sign" is partially or surrounded by opaque blood flow, or a complete filling defect with no distal blood vessels; the indirect sign is the pulmonary artery sign. Wild wedge-shaped strip-like high-density areas or discoid atelectasis, central pulmonary artery expansion and distal vascular distribution reduction or disappearance, etc. At the same time, the right ventricular shape and ventricular wall thickness can be analysed. The patient's CT showed embolism in the main trunk of both pulmonary arteries and its multiple branches. Care must be taken when evaluating CT examination results in patients with pulmonary embolism because pulmonary artery CT angiography has poor sensitivity in subsegments and below.
4. Others: It also includes electrocardiogram, echocardiogram and chest X-ray. The electrocardiogram shows ST segment depression and T wave inversion in V1-4 and limb leads II, III and AVF and V1 shows a QR shape; the direct sign of echocardiography is thrombus in the proximal pulmonary artery or right ventricular cavity and the indirect sign is right heart disease. Symptoms of overload; chest X-ray shows signs of pulmonary ischemia, such as sparse and slender lung textures, protruding or tumour-like expansion of pulmonary artery segments, widening of the right lower pulmonary artery or truncation sign and right ventricular enlargement sign.

Based on the above reasons, our research team recommends that if the patient is suspected of having a pulmonary embolism, direct CT imaging should be performed to gain time for rescue and treatment. The emergency CT image of this patient is as follows discussed below.

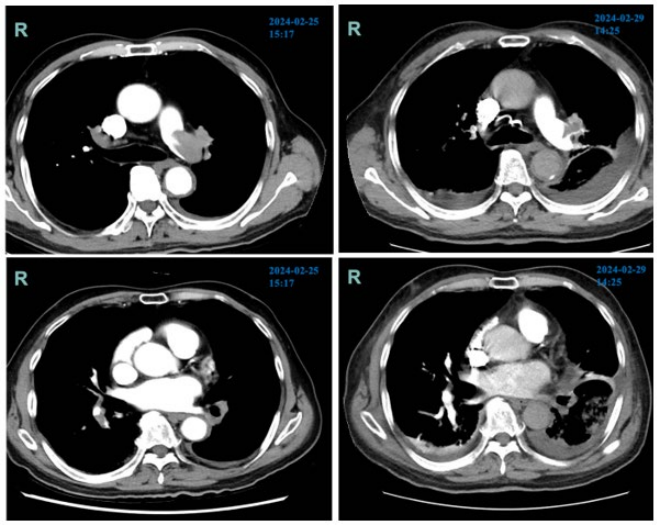

Figure 1: *Blood vessel window (2024-02-25 compare with 2024-02-29).*

The two images on the left depict a filling defect in the left pulmonary trunk and both lower pulmonary arteries, with the lumen nearing occlusion. In contrast, the image on the right illustrates a significant reduction in the filling defect area of the left pulmonary trunk after 4 days of treatment, alongside the disappearance of the filling defect in the right lower pulmonary artery, and the emergence of a small amount of pleural effusion bilaterally.

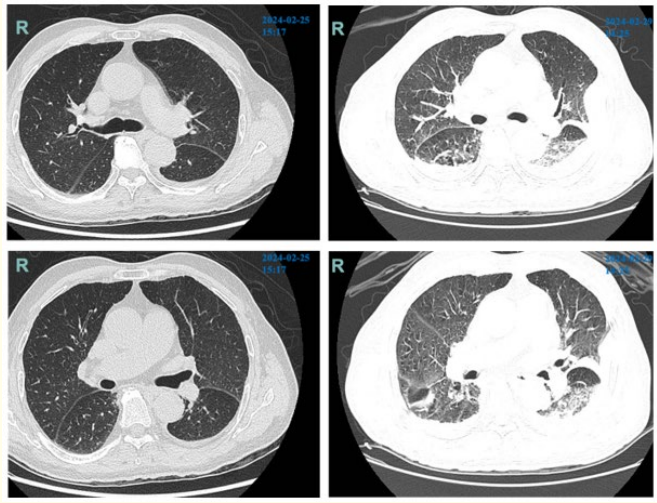

Figure 2: *Lung window (2024-02-25 compare with 2024-02-29).*

This is the lung window of the same case. The left image reveals no abnormalities in either lung before treatment. Conversely, the right image exhibits heightened density in both lower lungs following 4 days of treatment, indicative of inflammation, along with a minor pleural effusion in the bilateral cavities.

2024-02-25, 14:09, (1 hour after admission)

- Symptoms and signs: As previously stated.
- Laboratory tests: As described above.
- Treatment plan: After admission, 50 mg of alteplase was given for thrombolysis, followed by unfractionated heparin for anticoagulation, moxifloxacin for anti-infection, insulin for blood sugar control and other supportive symptomatic treatments.
- Expected results: Improve patient symptoms through thrombolysis, anticoagulation, anti-infection, blood sugar control and other treatments.
- Actual results: The patient's blood oxygen and blood pressure conditions improved, proving that the treatment was symptomatic and effective.

2024-02-26 15:00 (2nd day of admission)

- Symptoms and signs: The patient's cough and haemoptysis symptoms were significantly relieved, dyspnea symptoms disappeared, and occasional palpitations occurred. SPO_2 90% (low flow oxygen)
- Laboratory tests: White blood cell count 14.83x10^9/L↑; neutrophil percentage 89.8%↑; lymphocyte percentage 5.8%↓; blood gas analysis: corrected oxygen partial pressure 61.1 mmHg↓; corrected pH 7.470↑; corrected carbon dioxide partial pressure 32.7 mmHg↓; oxygen concentration 82.0%; oxygenation index 76.0 mmHg↓; prothrombin ratio 1.22↑; activated partial thromboplastin time 36.6 seconds↑; fibrinogen content 5.59 g/L↑; prothrombin activity 67.6%↓; prothrombin international ratio 1.22↑; D-dimer 17.18 μg/mL FEU↑; high-sensitivity troponin T0.159 μg/L↑; amino-terminal B-type natriuretic peptide 984 pg/ml↑; male tumour-related Antigen (serum): carcinoembryonic antigen 6.95 ng/ml↑; neuron-specific enolase 21.20 ng/ml↑; cytokeratin 19 fragment 5.56 ng/ml↑; *Mycoplasma pneumoniae* IgG antibody 43.3 AU/ml↑; protein C49.8% (70-140), protein S 58.9% (75-130), lupus-like anticoagulant screening: preliminary screening test (LA1) 66.2 seconds (31-44), confirmatory test (LA2) 50.4 seconds (30-38), lupus primary screening/lupus confirmation (LA1/LA2) 1.31 (0.8-1.2). There were no obvious abnormalities in cardiac enzymes, blood homocysteine, rheumatoid factor, anti-CCP antibodies, five items of thyroid function, 17 items of ENA antibody spectrum, 5 items of vasculitis, eight items of infection, and two items of autoantibodies. Antithrombin III activity 54.6%↓; albumin 32.1 g/L↓; complement 40.47 g/L↑; dynamic electrocardiogram showed sinus rhythm, frequent atrial premature beats, some with short bursts of atrial tachycardia, and some with doublet law. The colour ultrasound examination of the deep veins of the right lower limb showed: mild tricuspid regurgitation, low-normal left ventricular systolic function, no obvious effusion in the pericardial cavity, common femoral veins, proximal deep femoral veins, superficial femoral veins, The main trunks of the popliteal vein, posterior tibial vein, peroneal vein, anterior tibial vein, and intermuscular vein had smooth blood flow, and no thrombosis was found. There was no dilation of the roots of the double great saphenous veins. The saphenofemoral vein valve function was good; there was no dilation of the roots of the small saphenous veins, the venous valve function is good.
- Treatment plan: Maintain the original treatment plan.
- Expected results: Further improvement of patient symptoms, especially symptoms of hypoxia and dyspnea.
- Actual results: The patient's dyspnea symptoms disappeared, and laboratory tests showed that hypoxia was relieved; prothrombin ratio increased, antithrombin III activity decreased; D-dimer increased; deep vein colour ultrasound examination showed blood flow in most blood vessels Patent, left ventricular systolic function is at a low normal value.

2024-02-27 15:50 (3rd day of admission)

- Symptoms and signs: The patient's symptoms such as dyspnea, coughing and haemoptysis disappeared, but he began to have repeated fevers with a body temperature between 38.5 degrees and 39.1 degrees. No abnormalities were found on physical examination. SPO_2 90% (low flow oxygen).

- Treatment plan: Maintain the original treatment plan and change the type of antibiotics tomorrow.
- Expected results: Further improvement of patient symptoms.
- Actual results: The patient developed symptoms of infection, and it is unknown whether it was a hospital-acquired infection.

2024-02-28 15:30 (4th day of admission)

- Symptoms and signs: The patient still has repeated fevers, with a body temperature between 38.3 degrees and 39.2 degrees. No abnormalities were found on physical examination. SPO_2 95% (low flow oxygen).
- Treatment plan: Maintain the original treatment plan, switch to antibiotics and use Terzhixin to increase anti-infective treatment.
- Expected results: Strengthen infection treatment based on the original treatment plan and clarify the cause of infection.
- Actual results: The cause of infection is unknown and the symptoms of infection are not controlled as expected.

2024-02-29 17:37 (5th day of admission)

- Symptoms and signs: The patient occasionally coughs and still has repeated fever, with a body temperature between 38.1 degrees and 39.1 degrees. No obvious abnormalities were found on physical examination. SPO_2 95% (low flow oxygen).
- Laboratory tests: Pulmonary artery contrast-enhanced CTA and whole-abdominal and head CT were performed: 1. Embolism in the main trunks of both pulmonary arteries and its multiple branches has decreased compared with before and is now dominated by the left lower pulmonary artery. 2. Infection in the lower lobes of both lungs, with more lesions than before. 3. Small calcified nodule in the posterior apical segment of the upper lobe of the left lung, same as before. 4. A small amount of chronic infection/fibrous lesions in the middle lobe of the right lung and the lingual segment of the upper lobe of the left lung, roughly the same as before. 5. There is a small amount of secretion in the trachea, which is less than before. 6. The bilateral pleura was slightly thickened, and the small amount of pleural effusion on both sides increased compared with before. There was also partial encapsulated pleural effusion on the left side, and partial lung insufficiency was found in both lungs.
- Treatment plan: 1. Maintain the original treatment plan, and increase anti-infective treatment with antibiotics. 2. The left side of the chest was punctured and a catheter was inserted for drainage. The results of pleural effusion (pleural effusion) were as follows: red colour; mononuclear cell proportion 43%; Lifan test positive (+)↑; lobulated nuclear cell proportion 57%; transparency and turbidity; coagulation no block; red blood cell count 92000 10^6/L; white blood cell count 3231 10^6/L; four tumour-related antigens (pleural effusion): carcinoembryonic antigen 2.88 ng/ml; alpha-fetoprotein 0.91 ng/ml; carbohydrate antigen 125439.00 U/ml; carbohydrate antigen 19 - 95.55 U/ml, lactate dehydrogenase 663 U/L; chlorine 111.2 mmol/L; adenosine deaminase 9.6 U/L; glucose 7.02 mmol/L; total protein 30.2 g/L. The experimental results suggest that the pleural effusion is mainly exudate.
- Expected results: Strengthen infection treatment based on the original treatment plan and clarify the cause of infection.
- Actual results: The cause of infection is unknown and the relationship between the occurrence of pleural effusion and infection cannot be determined.

2024-03-01 15:30 (6th day of admission)

- Symptoms and signs: The patient's fever symptoms gradually disappeared and the body temperature was controlled between 36.7 degrees and 37.1 degrees. There was no obvious cough and sputum and no chest tightness, chest pain and haemoptysis. No obvious abnormalities were found on physical examination. SPO_2 95% (low flow oxygen).
- Laboratory tests: Lung CT scan and compare the image with first day of admission.
- Treatment plan: Maintain original treatment plan.

- Expected results: Infection control.
- Actual results: The patient's fever symptoms improved and his infection symptoms were significantly improved.

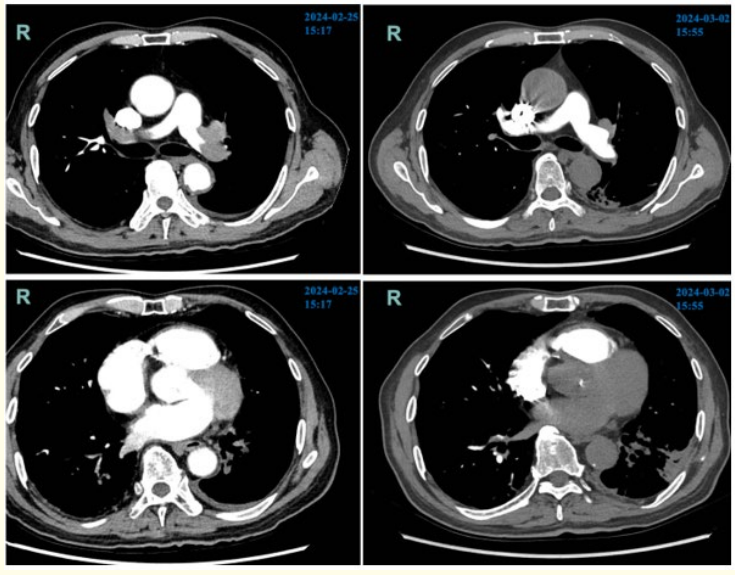

Figure 3: *Blood vessel window (2024-02-25 Compare with 2024-02-29).*

The left image reveals filling defects in both bilateral pulmonary trunks and inferior pulmonary arteries. In contrast, the right image, taken after 1 month of treatment, shows the disappearance of the filling defects in the bilateral pulmonary trunks, a reduction in the extent of the filling defects in the bilateral inferior pulmonary arteries, and the emergence of a wedge-shaped increased density shadow beneath the pleura of the lower lobe of the left lung, indicative of pulmonary infarction, along with a minor left-sided chest effusion.

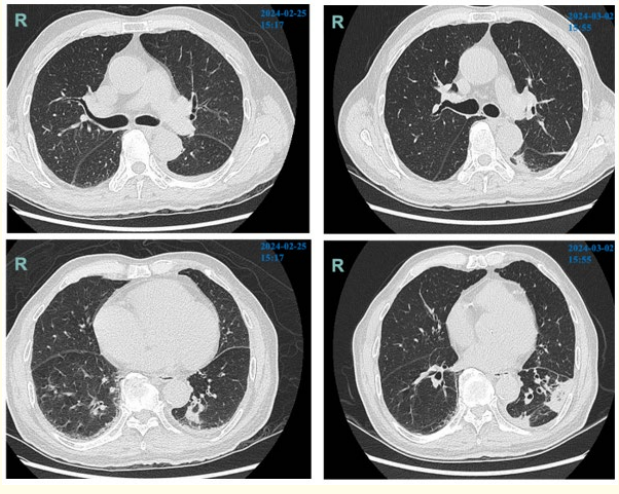

Figure 4: *Lung window (2024-02-25 Compare with 2024-02-29).*

This is the lung window of the same case. The left image exhibits no abnormalities in either lung before treatment. In contrast, the right image displays a small-scale subpleural pulmonary infarction in the lower lobe of the left lung and a minor effusion in the left pleural cavity, observed one month after treatment.

2024-03-02 16:50 (7th day of admission)

- Symptoms and signs: The patient's symptoms improved significantly, and no obvious abnormalities were found in the physical examination. SPO_2 95% (low flow oxygen).
- Laboratory tests: None.
- Treatment plan: The patient was switched to Kesai anticoagulant therapy, and the remaining treatment options were the same as before.
- Expected results: Control infection and patient is ready to stop the anticoagulant therapy.
- Actual results: Compared to admission, the patient's cough, haemoptysis, and dyspnea symptoms have largely subsided, with no anomalies detected during the physical examination. Additionally, compared to the lung CT scan conducted on February 25th, there is significant absorption of pleural effusion and favourable resolution of lung infection and pulmonary embolism indicators. Consequently, the patient can be transferred to the general ward for treatment.

2024-03-07 17:57 (12th day after admission)

- Symptoms and signs: The patient's symptoms such as cough and fever have disappeared, and no obvious abnormalities were found in the physical examination. SPO_2 95% (low flow oxygen).
- Laboratory tests: Precursor B-type natriuretic peptide (PRO-BNP) determination (serum): high-sensitivity troponin T0.020 μg/L↑; amino-terminal B-type natriuretic peptide 836 pg/ml↑; fibrinogen content 5.78 g/L↑; D-dimer 2.01 μg/mL FEU↑; rapid CRP77.87 mg/L↑; haemoglobin concentration 127 g/L↓; white blood cell count 9.29x10^9/L; platelet count 461x10^9/L↑; medium. The percentage of granulocytes was 78.8%↑; blood gas analysis: corrected pH 7.495↑; oxygenation index 358.7 mmHg↓; corrected oxygen partial pressure 70.9 mmHg↓; measured oxygen partial pressure 75.3 mmHg↓.
- Treatment plan: The patient is ready to be discharged from the hospital. The medicines he brings are (Provincial Asmei) compound methoxyphenamine capsules 2.0 tablets P.O. tid (7 days); (Andash) dapagliflozin metformin sustained-release tablets (I) 1.0 tablets P.O. qd (7 days); (Wenkean) acarbose tablets 50.0 mg P.O tid (7 days); (Shutanqing) Ambroxol dispersible tablets 30.0 mg P.O tid (7 days); Sitafloxacin tablets 100.0 mg P.O qd (7 days); Edoxaban 60 mg qd (self-prepared).

Laboratory examination change chart

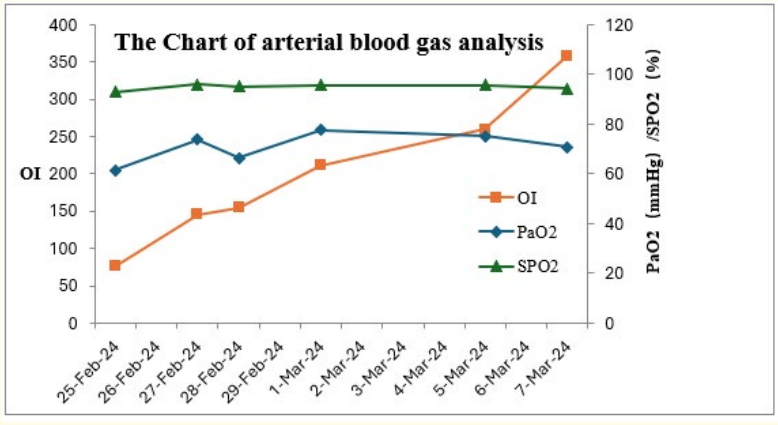

Figure 5: *The chart of arterial blood gas analysis.*

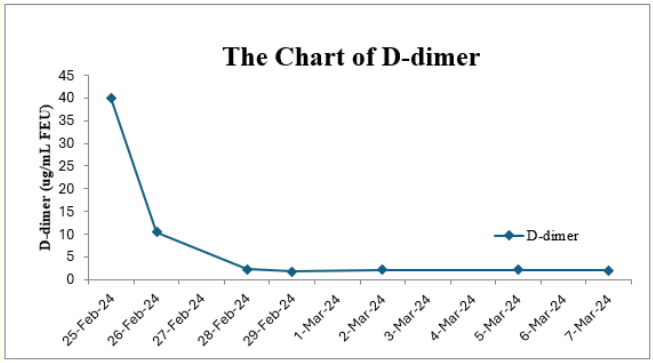

Figure 6: *The chart of D-dimer.*

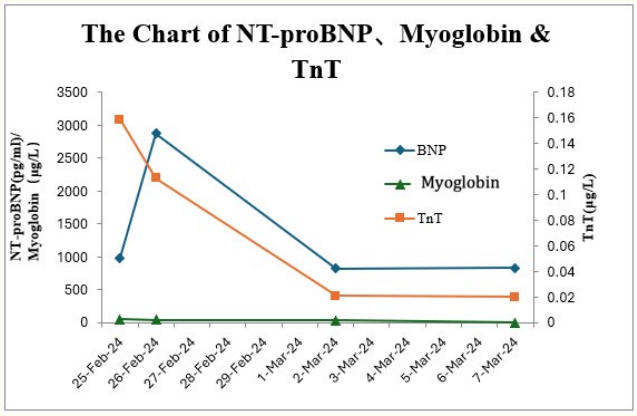

Figure 7: *The chart of NT-proBNP, myoglobin and TnT.*

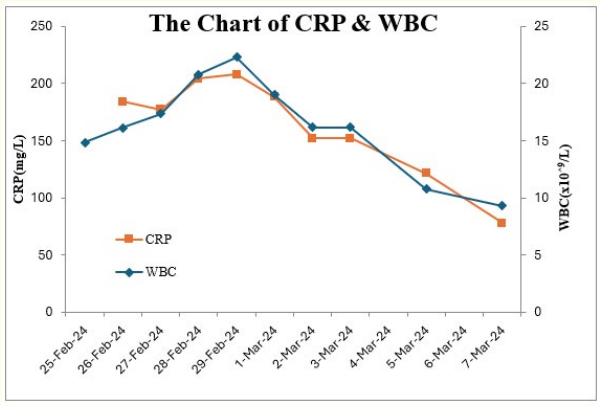

Figure 8: *The chart of CRP and WBC.*

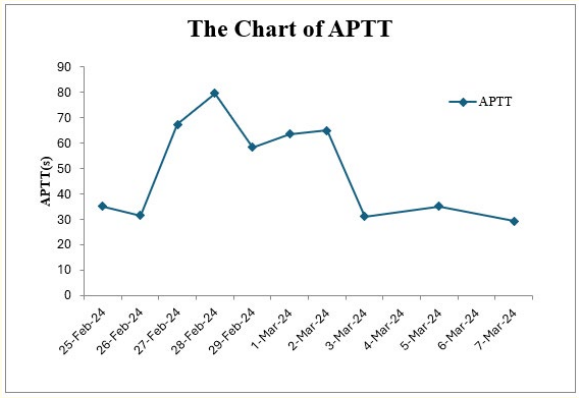

Figure 9: *The chart of APTT.*

Discussion

VTE (Venous thromboembolism) is a serious medical condition that includes deep vein thrombosis (DVT) and pulmonary embolism (PE), which is caused by obstruction of the pulmonary artery by endogenous or exogenous emboli, causing pulmonary circulation and right heart dysfunction. Clinical syndromes include pulmonary thromboembolism, amniotic fluid embolism, fat embolism, air embolism, tumour embolism, etc [1].

When the pulmonary vascular bed area is reduced by 40 - 50%, pulmonary artery pressure increases and the heart index decreases; when the pulmonary vascular bed area is reduced by 50 - 70%, persistent pulmonary hypertension may occur; when it exceeds 85%, sudden death may occur. Due to the seriousness of VTE, prevention and timely diagnosis are crucial. The AI-assisted intelligent decision support platform can provide a lot of help for early diagnosis and treatment decisions [7,8].

In the diagnosis of this disease, diabetes, smoking, obesity, hyperlipidaemia, pregnancy, tumours, chemotherapy and the use of erythropoiesis-stimulating factors, respiratory, urinary tract and HIV infections, blood transfusions and laparoscopy are high-risk groups, but not necessarily It is a necessary condition for the onset of disease [1].

The patient reported in this case had a typical onset of pulmonary embolism, with the triad of pulmonary embolism symptoms, namely dyspnea, chest pain and haemoptysis. However, generally speaking, the symptoms of many patients with pulmonary embolism are not obvious and lack specificity; the severity of their symptoms depends on the size and number of the emboli, the location of the embolism and whether the patient has underlying diseases of the heart, lungs and other organs. Most patients are suspected of having pulmonary embolism due to dyspnea, chest pain, presyncope, syncope, cough, and haemoptysis [9].

Typical signs include increased respiratory rate (> 20 times/min), increased heart rate (> 90 times/min), decreased blood pressure, and cyanosis. Jugular vein filling or abnormal pulsation, liver enlargement, hepatojugular reflux sign and lower limb oedema, one thigh or calf circumference is more than 1 cm larger than the opposite side, or lower limb varicose veins, hyperactive or split-second heart sound in the pulmonary valve area, systolic murmur, etc. can be heard in the tricuspid valve area. However, it may also be ignored or misdiagnosed because the signs are not obvious [10,11].

Therefore, clinically, if the patient has an obvious triad of pulmonary embolism, or if the patient is suspected of having pulmonary embolism, emergency lung CT can be considered, and pulmonary angiography can be performed directly if possible, because pulmonary

angiography is the gold standard for diagnosis, and direct signs include Contrast medium filling defect in the pulmonary artery, with or without blood flow obstruction of the "track sign"; indirect signs include slow flow of contrast medium in the pulmonary artery, local hypoperfusion, and delayed venous return.

However, pulmonary angiography also has its limitations because pulmonary angiography has poor sensitivity in subsegments and below. If the patient's disease is considered to be in the subsegment and below, radionuclide lung ventilation and perfusion scanning can be considered. The typical sign is a segmentally distributed perfusion defect that does not match the ventilation imaging. The sensitivity was 92% and the specificity was 87%. It has special significance, especially for sub-segments and below. However, any factors that cause impairment of pulmonary blood flow or ventilation, such as pneumonia, tumours, chronic obstructive pulmonary disease, etc., can cause local ventilation and blood flow imbalance. This examination alone can easily lead to misdiagnosis [12,13].

Therefore, our research group recommends three emergency tests for pulmonary embolism: arterial blood gas analysis, plasma D-dimer, and pulmonary angiography; if it is considered or there are symptoms, signs, or laboratory tests suggest that the pulmonary embolism is located at or below the subsegment, plus a radionuclide lung ventilation and perfusion scan.

Its diagnosis and evaluation can be based on "Wells score -original and simplified".

	Points 'original' Wells	Points 'simple' Wells
Clinical signs of DVT	3	1
Heart rate > 100/min	1.5	1
Recent surgery or immobilization	1.5	1
Previous PE or DVT	1.5	1
Hemoptysis	1	1
Malignancy	1	1
Alternative diagnosis less likely than PE	3	1
Cut-off for PE unlikely	≤4	≤1

PE = pulmonary embolism; DVT = deep venous thrombosis.

Figure 10: *"Wells score -original and simplified". Image adapted from Wells., et al. and Gibson., et al [14].*

In terms of treatment, the following initial risk stratification and diagnosis and treatment strategies can be selected [15].

The purpose of treatment for pulmonary embolism is to dissolve the thrombus as soon as possible, clear the blood vessels, reduce vascular endothelial damage and reduce the occurrence of chronic thromboembolic pulmonary hypertension [15-18].

Treatment in the acute phase is divided into three parts, 1. Hemodynamic and respiratory support; 2. Anticoagulation: The purpose of anticoagulation therapy for patients with pulmonary embolism is to prevent early death and recurrence of VTE. Divided into parenteral anticoagulation and oral anticoagulation. 3. Thrombolytic therapy: Thrombolytic therapy can quickly dissolve thrombus, restore lung tissue perfusion, reverse right heart failure, increase pulmonary capillary capacity and reduce mortality and recurrence rates.

Hemodynamic and respiratory support: Insufficient cardiac output caused by acute right heart failure is the primary cause of death in patients with acute pulmonary embolism. Active volume expansion is not only useless but may worsen right heart disease due to excessive mechanical stretch or reflex inhibition of myocardial contractility. Cardiac Function. For patients with acute pulmonary embolism with low cardiac index and normal blood pressure, moderate fluid shock can help increase cardiac output.

What should be noted here is that oxygen inhalation can relieve hypoxia and hypocapnia. However, positive end-expiratory pressure during mechanical ventilation can reduce venous return and worsen pulmonary function in patients with acute pulmonary embolisms who are hemodynamically unstable.

Anticoagulation: The purpose of anticoagulation therapy for patients with pulmonary embolism is to prevent early death and recurrence of VTE. Therefore, low-molecular-weight heparin and fondaparinux are superior to unfractionated heparin for initial anticoagulation, with a low risk of major bleeding and heparin-induced thrombocytopenia. Unfractionated heparin has the advantages of a short half-life, easy monitoring of anticoagulation, and can be quickly neutralized by protamine. It is recommended for patients who intend to be directly reperfused, as well as patients with severe renal insufficiency or severe obesity.

If unfractionated heparin is used: First give a loading dose of 2000 - 5000 IU or 80 IU/kg intravenously, followed by a continuous intravenous infusion of 18 IU/kg/h. Anticoagulation must be sufficient, otherwise it will seriously affect the efficacy and increase the recurrence rate of thrombosis. Measure APTT every 4 - 6 hours for the first 24 hours to adjust the dose of unfractionated heparin, and measure APTT again 3 hours after each dose adjustment to reach and maintain it at 1.5 - 2.5 times the normal value as soon as possible. Check platelets every 3 - 5 days.

If low molecular weight heparin is administered based on body weight, monitoring is generally not required. Use fondaparinux 2.5 mg subcutaneously once daily and no monitoring is required.

In addition to oral medications, oral anticoagulants should be given as early as possible, preferably with parenteral anticoagulants same day. Vitamin K antagonists include warfarin, nitrobenzyl acetone coumarin, phenprocoumon, phenindione, etc. Warfarin dose is 1 - 3 mg, when INR reaches 2 - 3, and maintain discontinue parenteral anticoagulants after 2 days or more. Non-vitamin K antagonists such as dabigatran, rivaroxaban, apixaban, and edoxaban can also be used.

Method for adjusting the dose of unfractionated heparin based on partial thromboplastin time (APTT).

Thrombolytic therapy: Thrombolytic therapy can quickly dissolve thrombus, restore lung tissue perfusion, reverse right heart failure, increase pulmonary capillary capacity and reduce mortality and recurrence rates.

The first is urokinase 20000 IU·kg^{-1}·2h^{-1}·intravenous infusion. And, followed by an intravenous injection of rt-PA50-100 mg for 2 hours. There is also r-PA (recombinant human tissue plasminogen kinase derivative) 18 mg dissolved in normal saline for intravenous bolus injection >2 min, and repeated bolus injection of 18 mg after 30 min.

It is necessary to accurately grasp the time window for thrombolysis: because the lung tissue is rich in oxygen supply and has triple oxygen supply of pulmonary arteries and veins, bronchial arteries and veins, and intra-alveolar ventilation, the incidence of pulmonary infarction is low. Thrombolytic therapy is most effective when started within 48 hours of the onset of acute pulmonary embolism. Thrombolytic therapy is still effective within 6 - 14 days for symptomatic patients with acute pulmonary embolism [19,20].

During the treatment of this patient in our ward, the treatment window was selected. Therefore, this case fully demonstrates the importance of AI technology in realizing intelligent risk assessment early warning and VTE clinical decision support systems. VTE (venous thromboembolism) seriously affects patient survival rate, so a VTE clinical decision support system using AI as a support method is proposed. By establishing a standardized private cloud platform to process and analyse VTE patient data, the system can automatically identify and learn standardized patient information to provide support for early diagnosis and treatment. This system not only helps improve survival rates but also reduces complications, emphasizing the importance of prevention and timely diagnosis for VTE patients.

From the above discussion, it can be seen that the AI-assisted intelligent decision support platform has good application prospects in VTE management [21].

Patients with pulmonary embolism must undergo strict risk assessment. For patients with acute pulmonary embolism accompanied by shock or sustained hypotension, hemodynamic and respiratory support should be provided. Direct reperfusion therapy is the best choice. For patients with contraindications to thrombolysis or failure of thrombolysis and hemodynamic instability, surgical thrombectomy is feasible. Of course, percutaneous catheter intervention is also possible. Routine systemic thrombolysis is not recommended for patients without shock or hypotension. Subcutaneous injection of low molecular weight heparin or fondaparinux is the best choice.

Finally, in terms of the duration of anticoagulant treatment, the experience of my research group is long-term anticoagulant drug selection: most patients can use warfarin for a long time, and long-term use of low molecular weight heparin is safer and more effective for tumour patients. The new oral anticoagulants dabigatran, rivaroxaban and apixaban are effective in long-term anticoagulation in the treatment of VTE and are safer than warfarin. After the patient was discharged from the hospital with medicine, he received regular follow-up visits and outpatient re-examination.

The above is a typical diagnosis and treatment process of a standardized pulmonary embolism patient in my research group, which has been entered into the platform as a VTE patient for AI learning. After the overall big data continues to be collected, sorted and analysed, the VTE intelligent risk warning system, clinical record post-structuring system, AI-VTE risk control analysis platform and patient VTE independent risk management platform can be directly integrated into one.

Conclusion

In conclusion, VTE poses a formidable challenge in contemporary healthcare, necessitating concerted efforts to enhance diagnostic accuracy and therapeutic efficacy. Our research underscores the critical role of AI in augmenting clinical decision-making processes for VTE management. Through the development of an AI-assisted intelligent decision support platform, we aim to empower healthcare professionals with tools for early risk assessment and intervention. The ongoing refinement and validation of this platform signify a pivotal step towards optimizing VTE care delivery. Moreover, our findings underscore the importance of rigorous risk assessment and personalized treatment strategies in improving patient outcomes.

Based on this, more standardized patients will be selected and inputted to initiate and activate the AI-assisted intelligent decision support platform.

And looking ahead, our research integrate of AI technologies into VTE management holds promise for revolutionizing clinical practice and ushering in an era of precision medicine.

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