Massive pulmonary thromboembolism during arthroscopic surgery: the important roles of chest compression and endotracheal intubation.

Guoqiang Chen, Qin Xia, Liwei Liu, Cheng Li, Shukun Fu*

Department of Anesthesiology, School of Medicine, Tongji University, Shanghai Tenth People’s Hospital, Shanghai, PR China

Abstract

A case of acute massive pulmonary embolism event happened during fracture fixation in our department recently. Shortly after surgery has begun, vital signs of the patient fluctuated emergently, his consciousness was lost, blood pressure and heart rate was once lost in monitor. We immediately started cardiopulmonary resuscitation plans, including setting up advanced life support, chest compressions and cryogenic brain protection strategy, opening center vein to establish a blood pressure, and monitoring central venous pressure and arterial blood pressure. When trying to maintain the vital signs stable, we improved the relevant inspection at the same time. Through a multidisciplinary collaborative diagnosis, acute massive pulmonary embolism was diagnosed. Heparin was given for anticoagulation, and recombinant tissue plasminogen activator was used for thrombolysis treatment. After vital signs were stable, the patient was transferred to the intensive care unit. Then, the disease progressed into multiple organs failure. After treatment for nearly a month, the patient was discharged, without any sequela or complications.

Keywords: Thromboembolism, Arthroscopic surgery, Chest compression, Endotracheal.

Introduction

Pulmonary Thromboembolism is a common complication of Deep Venous Thrombosis (DVT), they are collectively known as Venous Thromboembolism (VTE). In recent years, though the awareness of Pulmonary Embolism (PE) increases, still there is an existence of misdiagnosis, missed diagnosis or not timely diagnosis, as well as non-standard thrombolysis and anticoagulation treatment in clinical practice.

Epidemiology

Acute PE is the most severe clinical manifestation of VTE. Most existing epidemiology researches study risk factors and the natural course of VTE as a whole. The incidence of it was 100-200/100,000 [1,2].

There can be no symptoms in PE. Sometimes it was diagnosed accidentally. Initial manifestation in some PE patients is sudden death. So it is difficult to obtain accurate PE epidemiological data. According to estimates from epidemiological model, in the six countries of the European Union with a population of 454.4 million in 2004, PE caused death to more than 317,000 cases. Among them, the sudden fatal PE accounted for 34%, of whom 59% failed to diagnose before death, only 7% of early death cases were diagnosed before death. The risk of PE is associated with increasing age. In people aged more than 40, for every 10 y increase, incidence of PE increases about 1 time.

In items for prevention and control of pulmonary embolism in China from 1997-2008, PE patients from more than 60 tertiary hospitals registered. There were a total of 18,206 cases in 16,792,182 cases of hospitalized patients diagnosed with PE. The incidence was 0.1% [3].

Susceptible factors

Susceptible factors of VTE include the patient’s own factors (mostly permanent factors) and environmental factors (mostly temporary factors). Six weeks to three months of temporary and reversible risk factors (such as surgery, trauma, braking, pregnancy, oral contraceptive or hormone replacement therapy, etc.) may induce VTE, but in the cases of lack of any known risk factors, PE can also occur.

Major trauma, surgery, lower limb fracture, joint replacement and spinal cord injury are the strong inducing factors of VTE; tumor, pregnancy, oral contraceptives, hormone replacement therapy, and center venepuncture are also recognized susceptible factors of VTE. With the deepening of research, new susceptible factors have been found: VTE, as a part of the cardiovascular disease, shares common risk factors (such as smoking, obesity, hyperlipidaemia, hypertension and diabetes) with artery diseases, especially atherosclerosis. Incidence of VTE is much higher in patients with a disease history of myocardial infarction within 3 months, or hospitalized because of heart failure, atrial fibrillation, or atrial flutter. In vitro
fertilization further increases the risk of VTE related to pregnancy, especially in the early 3 months. Infection is one of the main inducing factors of in-hospital VTE, and blood transfusion and erythropoiesis factors can also increase the risk of VTE. Common susceptible factors are shown in Table 1 [4].

### Table 1. Susceptible factors of VTE.

<table>
<thead>
<tr>
<th>Strong susceptibility factors (OR&gt;10)</th>
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<tr>
<td>Lower limb fracture</td>
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<tr>
<td>Hospitalization due to heart failure, atrial fibrillation or atrial flutter within 3 months</td>
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<td>Hip or knee arthroplasty</td>
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<td>Severe trauma</td>
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<td>Myocardial infarction within 3 months</td>
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<td>Previous VTE history</td>
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<td>Spinal cord injury</td>
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<tr>
<th>Medium susceptibility factors (OR 2-9)</th>
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<tbody>
<tr>
<td>Knee arthroscopic surgery</td>
</tr>
<tr>
<td>Autoimmune disease</td>
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<tr>
<td>Blood transfusion</td>
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<td>Central venous catheter</td>
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<tr>
<td>Chemotherapy</td>
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<tr>
<td>Chronic heart failure or respiratory failure</td>
</tr>
<tr>
<td>Application of erythropoiesis factors</td>
</tr>
<tr>
<td>Hormone replacement therapy</td>
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<tr>
<td><em>In vitro</em> fertilization</td>
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<tr>
<td>Infection (respiratory system, urinary system infection or HIV infection)</td>
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<tr>
<td>Inflammatory bowel disease</td>
</tr>
<tr>
<td>Tumor</td>
</tr>
<tr>
<td>Oral contraceptives</td>
</tr>
<tr>
<td>Stroke paralysis</td>
</tr>
<tr>
<td>Postpartum</td>
</tr>
<tr>
<td>Superficial vein thrombosis</td>
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<tr>
<td>Inherited thrombophilia</td>
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<tr>
<th>Weak susceptible factors (OR&lt;2)</th>
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<tbody>
<tr>
<td>Stay in bed&gt;3 d</td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Sedentary condition (such as car or aircraft travel for a long time)</td>
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<tr>
<td>Age increase</td>
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<tr>
<td>Laparoscopic surgery (such as laparoscopic cholecystectomy)</td>
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<tr>
<td>Obesity</td>
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Note: OR=Odds Ratio, Relative Risk

This pulmonary thromboembolism patient was with multiple risk factors (such as lower limbs surgery, a knee operation, obesity, sedentary, age increase, etc.), and the onset was severe, with the first symptom of shock, so that it was unable to accurately diagnose the cause at the first time. Corresponding symptomatic treatment and life support were the only treatment. Then, with the improvement of relevant inspection and multidisciplinary assist diagnosis, etiology was clear, precise anticoagulation thrombolysis treatment was implemented; the patient was gradually smooth and steady. Since the patient experienced sharp fluctuations in circulation and pulmonary embolism, after transferred to the ICU for further treatment, multiple organs had varying degrees of damage; especially kidney function suffered the worst damage. After treatment and haemodialysis for a month, the patient was discharged, without any complications. We now report the pulmonary embolism case as follows.

### Case Report

A 40 y old male patient, injured by car accident, was caused impact trauma of lower limb leading to closed injury of right knee joint, who was then hospitalized with right knee pain and motion limitation. The MRI revealed the right lateral tibial plateau fracture; possibility of right knee lateral collateral ligament injury; right knee effusion. There was nothing unusual in personal history, obstetrical history and family history of this patient. The preoperative examination had nothing special.

The patient was treated with celecoxib to kill pain, lower limb plaster immobilization, without low molecular weight heparin. The patient was distributed to operating room 6 d after trauma, and spinal anesthesia was delivered. In the middle of surgery, the patient experienced sudden chest tightness, sharp chest pain and irritability. The blood oxygen saturation (SPO$_2$) and heart rate dropped, non-invasive blood pressure (NIBP) was 65/40 mmHg. Ephedrine 15 mg, atropine 0.5 mg and methylprednisolone 160 mg were injected immediately, the patient breathed on his own with tidal volume 400-500 ml, and the lungs bilateral breath sounds were clear and symmetrical. Since the SPO$_2$ continued to decline, endotracheal intubation was applied to this patient, but the SPO$_2$ was still only 70-80%. The ETCO$_2$ maintained at 0-5 mmHg, with the position of endotracheal tube has been confirmed. At this moment, the SPO$_2$ decreased to 60-70%, NIBP was unavailable and heart rate decreased to 20-30 bpm with ECG in third degree atrioventricular block. Epinephrine 1 mg, heparin sodium 100 mg were injected intravenously, and defibrillation was applied to the patient. Then, the heart rate had returned to normal rhythm and increased to 160 bpm with SPO$_2$ and NIBP still unavailable. After a few min, the heart rate gradually decreased. The ECG was in profound bradycardia, 20-30 bpm
and ventricular escape beats were seen. Atropine 1 mg was injected but had no effect; subsequent epinephrine 2 mg was given to him with chest compression applied. After about 30 times chest compressions, the ETCO$_2$ suddenly increased from 0-5 mmHg to 30 mmHg and the SPO$_2$ gradually recovered to 100%. The 12-lead ECG was in sinus rhythm with SIQIIITIII present, and a complete right branch block was diagnosed. Bedside chest radiograph showed nothing different from the preoperative one. Blood gas analysis revealed profound respiratory acidosis and metabolic acidosis (Table 2). The D-dimer value was extremely high (Table 3).

**Table 2. Dynamics parameters of blood gas analysis during the surgery.**

<table>
<thead>
<tr>
<th>Time (h)</th>
<th>pH</th>
<th>PaCO$_2$ (mmHg)</th>
<th>PaO$_2$ (mmHg)</th>
<th>BE (mmol/L)</th>
<th>HCO$_3$ (mmol/L)</th>
<th>SPO$_2$ (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.529861</td>
<td>6.801</td>
<td>110.5</td>
<td>224</td>
<td>-17</td>
<td>17.2</td>
<td>99</td>
</tr>
<tr>
<td>0.577778</td>
<td>6.881</td>
<td>100.4</td>
<td>106</td>
<td>-14</td>
<td>18.8</td>
<td>91</td>
</tr>
<tr>
<td>0.600694</td>
<td>6.892</td>
<td>103</td>
<td>92</td>
<td>-13</td>
<td>19.8</td>
<td>87</td>
</tr>
<tr>
<td>0.658333</td>
<td>7.014</td>
<td>90.8</td>
<td>60</td>
<td>-8</td>
<td>23.1</td>
<td>74</td>
</tr>
</tbody>
</table>

**Table 3. Dynamics parameters of coagulation function during the surgery.**

<table>
<thead>
<tr>
<th>Time (h)</th>
<th>D-dimer (µg/ml)</th>
<th>INR</th>
<th>APTT (s)</th>
<th>PT (s)</th>
<th>Fibrinogen (g/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.647222</td>
<td>&gt;10</td>
<td>1.54</td>
<td>&gt;120</td>
<td>18</td>
<td>2.9</td>
</tr>
<tr>
<td>0.69236</td>
<td>&gt;10</td>
<td>3.65</td>
<td>&gt;120</td>
<td>43.3</td>
<td>&lt;0.5</td>
</tr>
</tbody>
</table>

The patient was diagnosed as massive pulmonary embolism, given recombinant Tissue Plasminogen Activator (rTPA) 100 mg intravenously for thrombolysis, and NaHCO$_3$ 150 ml was infused twice for correcting acidosis. When the plasma pH ascended to 7.014, the infusing of NaHCO$_3$ had been terminated, in order to avoid intracellular acidosis. Due to hemodynamic instability, epinephrine was given several times repeatedly. The patient was unconscious with dilatation of pupils and disappearance of pupillary reflex. Ice cap was given for cooling, and mannitol for dehydration treatment. The SPO$_2$ recovered to 100% after chest compressions, then gradually decreased, finally stabilized at about 88%, and the Arterial Blood Pressure (ABP) stabilized at about 100/65 mmHg with dopamine continuous infusion. When the ABP was stabilized, furosemide 40 mg was administrated immediately, but it was not until rTPA infused over, the urine came out. The patient was transferred to ICU for further treatment. He developed acute multiple organ failure after admission to the ICU (Figures 1A-1E). The echocardiography showed dilated right atrium and right ventricle, tricuspid valve with moderate regurgitation, suggesting pulmonary hypertension, diastolic retrogression of left ventricular. After 2 months treatment, the patient was discharged with no sequela left. The surgery was successful without any sequela.

**Figure 1.** (A) The dynamic change of ALT within 11 d after operation (peaked at the second day, and then decreased till normal); (B) The dynamic change of NT-proBNP within 12 d after operation (peaked at the seventh day, and then decreased); (C) The dynamic change of hs-TNT within 12 d after operation (peaked at the second day, and then decreased till normal); (D) The dynamic change of Creatinine within 40 d after operation (peaked at the eighth day, and then decreased); (E) The dynamic change of D-dimer after operation (decreased rapidly after three days). P.O.D: Post Operative Day.

**Discussion**

Venous Thromboembolism (VTE) is responsible for the hospitalization of 250,000 Americans annually and represents a significant risk for morbidity and mortality [5]. PE history, superior vena cava syndrome, Deep Vein Thrombosis (DVT) history or family history of DVT, major operations, immobilization for more than 30 d, general anesthesia, malignancy, central venous catheterization, major injuries, foreign body, polycythemia, thrombocytosis, cor pulmonale, congestive cardiac failure, thoracic outlet syndrome, coagulation disorders and usage of oral contraception pills are considered predisposing factors of PE [6-11]. Patients with immobilized lower limb trauma should take measures to prevent PE, like smoking cessation, low molecular weight heparin, venous filter implantation and early surgery.

In PE, pulmonary arterial pressure is increased following thrombotic obstruction. Vasoconstriction, pulmonary hypertension, right-sided heart failure, and bronchoconstriction may be seen in the advanced stage [12]. Guidelines identify massive PE as acute PE with sustained hypotension (systolic blood pressure<90 mm Hg for at least 15
Compared to other biochemical parameters, the renal function
not stabilize at approximately 88%.

was damaged so badly and persistently due to the hypoxia,
early stage might be more helpful.

treatment of missive PE. The usage of haemodialysis in the
hypotension, acidosis and the usage of large dose epinephrine
that the protection of renal function was very important in the
treatment of missive PE was rarely mentioned.

Our experience with the presented case suggested endotracheal
intubation and chest compression played important roles in the
rescue of patient with massive PE diagnosed.

Endotracheal intubation and the use of muscle relaxant could
ensure the pulmonary ventilation to greatest extent which is
extremely important in the missive PE. Endotracheal intubation
established the advanced airway for the possible consequent
cardiopulmonary resuscitation.

The monitor of ETCO\textsubscript{2} could not only confirm the position of
endotracheal tube but also estimate the treatment effect of
missive PE. In this patient, the ETCO\textsubscript{2} jumped from 0-5
mmHg to 30 mmHg strongly suggested that the effective
alveolare ventilation had been extremely improved. Moreover,
monitor of ETCO\textsubscript{2} could evaluate the quality of possible
cardiopulmonary resuscitation, which might help the
physicians to determine whether more efficient
cardiopulmonary resuscitation was needed or the spontaneous
circulation had returned [14].

The implementation of chest compression might cause the life-
threatening blood clots to change position or fragment into
small pieces, which resulting the pulmonary hemodynamic
improvement might play a crucial role in this case of missive
PE. Chest compression is easy to implement and effects fast,
which is matching the character of emergency rescue for
missive PE.

However, we did not deny the importance of anticoagulation and
fibrinolysis. Actually, therapeutic anticoagulation was
given to the patient during the diagnostic workup to avoid the
blood clots growing up. Fibrinolysis was given to him even
though the surgery was in progress, because of the
hemodynamic instability. Moreover, the implementation of
chest compression might cause the life-threating blood clots
to fragment into small pieces, but according to the continuous
results of arterial blood gas analysis, the SPO\textsubscript{2} recovered to
100% after chest compressions, then gradually decreased,
suggesting the smaller blood clots might entry and block
pulmonary small arteries. So without fibrinolysis, the blood
clots could not be eliminated completely and the SPO\textsubscript{2} could
not stabilize at approximately 88%.

Compared to other biochemical parameters, the renal function
was damaged so badly and persistently due to the hypoxia,
hyptension, acidosis and the usage of large dose epinephrine
that the artificial kidney dialysis had to be applied. It suggested
that the protection of renal function was very important in the
treatment of missive PE. The usage of haemodialysis in the
early stage might be more helpful.

In conclusion, our experience with the presented case suggests
that endotracheal intubation with monitor of ETCO\textsubscript{2} and chest
compression should be implemented as soon as possible when
the life-threatening missive PE diagnosed. More efforts should
be spent on the protection of renal function in the treatment of
missive PE. However, more prospective studies are needed.

Acknowledgment

This work was supported in part by Youth Project of Shanghai
Health and Family Plan Committee (20144Y0150).

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*Correspondence to
Shukun Fu
Department of Anesthesiology
School of Medicine
Tongji University
Shanghai Tenth People’s Hospital
Shanghai
PR China