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Pulmonary artery embolism during the course of colitis ulcerosa — the constant diagnostic challenge of invasive fungal infection.

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Sir,

Inflammatory bowel disease (IBD) predisposes one to thrombo-embolic complications [1]. Neither the cause, nor the mechanism behind this phenomenon is clearly defined. Emboli are usually formed of blood clots, but may also be of malignant, bacterial or fungal origin. Here we present the case of a fatal pulmonary artery embolism caused by fungal matter in a patient with ulcerative colitis.

A 55-year-old Caucasian male was admitted to medical ward due to unexpected weakness, abdominal pain and a suspected diagnosis of ulcerative colitis (UC). He had suffered from severe diarrhoea for the previous several months (up to a dozen and more stools per day, some of them with large amounts of blood), with episodes of rectal bleeding between bowel movements, which was assumed to be haemorrhoid bleed. After a few days in a medical ward, an intestinal biopsy was done and a diagnosis of an active stage of ulcerative colitis established. The patient's prior medical history was not significant: he denied suffering from any medical conditions and being on any medication. Although a possible infective cause of diarrhoea was investigated, these tests proved negative. Routine pharmacological management of UC exacerbation was employed. Due to confirmed and significant bleeding from fourth-degree haemorrhoids, a hemorrhoidectomy was scheduled and performed. On the first postoperative day, the patient's condition began to deteriorate.

Based on clinical presentation (abdominal distension, rebound tenderness) and diagnostic tests (abdominal X-ray showed a caecal diameter of 11 cm), a toxic megacolon was suspected. It was decided to proceed with a laparotomy that confirmed this diagnosis. A colectomy with an ileostomy was performed. The following day an upper gastrointestinal bleeding occurred. An emergency gastroscopy was performed, which showed some areas of possible superficial haemorrhage in the fundus. Injections of dilute adrenaline around the areas of haemorrhaging mucosa were used to control the bleeding, with success. The next day patient's condition rapidly changed and he was admitted to an ICU. He was in respiratory failure, had a temperature of 39.5°C, and presented with fluid-unresponsive hypotension. Laboratory tests demonstrated a white cell count of 16 G L^{-1}, C-reactive protein and lactate levels of 178 mg L^{-1} and 20 mg dL^{-1} respectively. He was then intubated and put on mechanical ventilation. Antibiotic therapy was changed to vankomycin, imipenem/cilastatin and voriconazol. An abdominal ultrasound showed an abscess in the lower portion of the wound, which was subsequently drained.

No significant findings were reported in an abdominal computed tomography. Routine intensive care procedures were applied. Apart from inflammation markers being elevated, no other spectacular disturbances in pathology tests were detected. Blood cultures were negative. Subcutaneous enoxaparin sodium was used as antithrombotic prophylaxis during the entire hospital stay. As clotting disturbances were suspected to be likely, some additional tests were done. Low lupus anticoagulant titre was detected, with neither prothrombin, nor factor V Leiden mutations present. The patient's condition was critical but stable, with some minimal improvement observed during the ICU stay. On the 17^{th} day of hospitalization, a sudden cardiac arrest occurred (asystole). After 30 minutes of resuscitation, hemodynamically effective rhythm was restored, inotropes were started to maintain blood pressure (norepinephrine and dobutamine). Although a pulmonary embolism was suspected, the patient's critical condition made his transport to the radiology department hazardous, and thus an angio-CT was not performed. Fibrynolysis was not implemented due to a history of recent surgery and gastrointestinal haemorrhage. Antithrombotic therapy with an appropriate dose of low-molecular-weight heparin (LMWH) was started. Unfortunately, before another 48 hours had passed another cardiac arrest occurred and resuscitation was unsuccessful.

An autopsy revealed major abnormalities in lung tissue consistent with pneumonia. A large thrombus was found in the pulmonary artery, which had been formed by fungal colonies interspersed with fibrinogen. Smaller and more diffuse clots of the same character were noted in the heart cavities.

A history of surgical procedures within the abdominal cavity, perioperative venous thrombosis, vascular damage, as well as immobility itself, place ICU patients into a high-risk group for thrombo-embolic complications [2].

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In ulcerative colitis the average risk is 4%. The coexistence of several risk factors increase this to as much as 20%. It is no less, even when the inflammation is limited to the terminal portion of the colon only. However, Jackson *et al.* suggest that an increased risk of thrombosis in ulcerative colitis is not related to periods of flares and is sustained even during remission [3]. However, the mechanisms of this phenomenon have not been entirely elucidated. It is likely that the prothrombotic activity of plasma is mediated by continuously elevated proinflammatory cytokines. During the course of the disease, both the number and activity of platelets increase, as well as fibrinogen and factors V, VIII, XII and I. Tissue factor and thrombin-antithrombin complex synthesis is similarly promoted. Additionally, the level of tissue plasminogen activator inhibitor (PAI) decreases [3, 4]. According to Jackson *et al.*, during the course of ulcerative colitis, the synthesis of natural anticoagulants, such as protein C, protein S and antithrombin III may be depressed, whereas antiphospholipid antibodies are more likely to be detected [3]. In the case described, neither symptoms nor positive diagnostic results confirming any of the above were noted. Protein C activity was 113%, protein S was normal. Thrombo-embolic complications are not restricted to pulmonary circulation only. There are case reports of hepatic vein thrombosis and Budd-Chiari syndrome in patients with ulcerative colitis [5]. Pulmonary embolism, and in particular the pulmonary artery embolism, is a very serious complication of IBD, with the first case having been described in 1936. Due to the fact that many cases of pulmonary embolism are asymptomatic, it is often identified during an autopsy. For this reason, early diagnosis facilitating the application of the correct and effective treatment is crucial, as up to 25% of all deaths among patients with non-specific colitis is due to thrombo-embolic complications [6].

In case described above, a few potential risk factors could have contributed to the progress of fungal infection, such as laparotomy, i.v. steroids, arterial and central venous access. As the clinical presentation of mycosis may be minimal or lacking in a large percentage of patients, the laboratory tests are vital and include a full blood count and white cell differential, CRP and procalcitonin (PCT) determinations. Numerous studies confirm the value of PCT measurements in the diagnosis and management of infectious diseases, of both bacterial and fungal origin, as well as post-operative complications [7]. An increase in PCT (more than hundredfold) was noted 24 hours prior to the occurrence of the fatal pulmonary embolism. It is likely that it was caused by release of the fungal cells into the circulation. Considering that neither PCT nor CRP is specific for this type of infection, some more specific (although not perfect) tests, such as a galactomannan antigen or beta-D-glucan assay could have been performed. However, the latter may return false negative results if prophylactic antifungal therapy is applied (fluconazole and then voriconazole were used). Blood cultures that were sent to exclude possible sepsis were negative for fungi.

The results of the autopsy showed that antifungal treatment had not been effective, despite the appropriate dose and timing. Fungal invasions are often obscure, with minimal or no clinical manifestations, no increase in anti-fungal antibodies and only a low possibility to detect antigens. We believe that in the case described, severe pulmonary embolism was a result of massive fungal invasion superimposed on the chronic fungal infection. Unfortunately, it is not possible to define an exact duration of the latter.

The effective treatment of the underlying inflammatory disorder (IBD), regardless of the method applied, tends to the diminish inflammatory process and decrease the risk of thrombosis, which may be found in both the venous and arterial vasculature [8]. In ulcerative colitis, thromboembolic prophylaxis is largely based on unfractioned heparin or LMWH. It is worth mentioning that both types of heparin exhibit some anti-inflammatory properties, which may potentially influence the course of IBD. Numerous studies confirm that there is no increase in the risk of haemorrhage due to their use [1, 9].

In patients with a high risk of thromboembolic complications, every unusual or unspecific symptom resulting in a compromise of the respiratory system may be indicative of pulmonary embolism. As diagnostic tools are readily available, it appears that it is only the clinician's judgment which facilitates appropriate management. It is our hope that the case described here will draw attention to the multitude of possible causes and clinical presentations of this serious, life-threatening condition.

CONSENT

Written informed consent was obtained from the patient's family for publication of this case and the accompanying images.

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2. The authors declare no conflict of interest.

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Ultrasound and fibreoptic-guided percutaneous tracheostomy in patient with deviated trachea

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Sir,

A 78-year-old female, suffering from bronchial asthma with old pulmonary Koch's disease was admitted with a history of a fall followed by pain in the right hip. She was diagnosed with an inter-trochanteric fracture of the right femur. Her chest x-ray showed right middle zone and lower zone homogenous opacity with tracheal deviation (Fig. 1). She was admitted to the ICU after 4 days of hospital stay following acute breathlessness and arterial blood desaturation. The trachea was intubated and ventilatory support was commenced due to respiratory failure.

Due to predicted prolonged mechanical ventilation support, a percutaneous tracheostomy (PCT) was planned on the 5th day from ICU admission.

The trachea was located being shifted toward right side on palpation. A decision was made to use a USG for accurate localization of trachea and its real time puncture. Following skin disinfection, a vertical incision of 1cm was performed 2 cm lateral to the midline at the level of the 2nd and 3rd tracheal cartilage (Fig. 2). The percutaneous tracheostomy **(**PCT) was performed with the Blue Rhino single dilator technique. The procedure was completed with minimal blood loss and no complications.

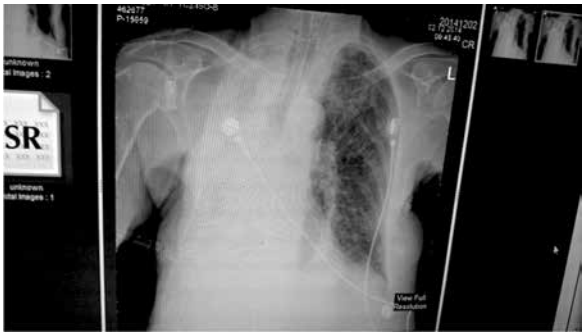

Figure 1. Chest x-ray of the patient showing tracheal deviation toward the right side

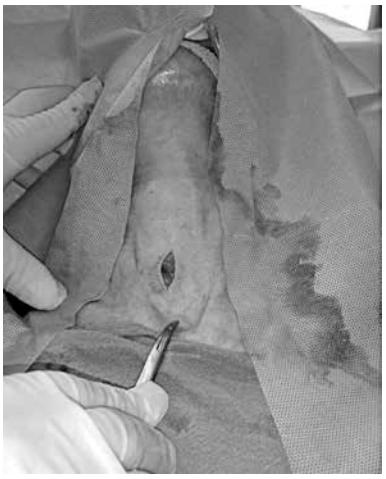

Figure 2. Modified incision for percutaneous tracheostomy 2 cm lateral to midline

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