False Pulmonary Embolism on Computed Tomography Angiography in Two Patients With Thoracic Anatomic Distortion☆

Falso tromboembolismo pulmonar en la angiografía por tomografía computarizada en dos pacientes con distorsión anatómica del tórax

To the Editor,

Computed tomography pulmonary angiography (CTPA) has become the gold standard imaging technique in the evaluation of patients with suspected pulmonary thromboembolism (PTE).[1] PTE – acute or chronic – is confirmed on CTPA by filling defects in the pulmonary arteries. Several factors, whether technical or disease- or patient-related, can result in a suboptimal, low-quality CTPA, which can be misinterpreted by the radiologist.[2]

We report 2 cases of mistaken diagnosis of acute PTE in the emergency department of our hospital. Both patients complained of chest pain and dyspnea: both had elevated D-dimer levels and significant structural changes in the left hemithorax. In one of the patients, changes were due to post-radiation sequelae from lung cancer in clinical remission (Fig. 1A), and in the other they were caused by long-term pleural thickening from calcified tuberculosis (Fig. 1B). Anatomical distortion of the chest structures in general (loss of left hemithorax volume, mediastinal shift), and of the pulmonary arteries in particular (traction and kinking) caused artefacts related with turbulent flow of both blood and intravenous contrast medium in the pulmonary arteries of the left hemithorax, visualized on CTPA as apparent pulmonary artery defects (Figs. 1C and D). Filling defects were ruled out by late phase acquisitions on chest CT (obtained when the intravenous contrast medium is more diluted), thus confirming that the observations were due to artefacts. Neither patients had other pulmonary circulation filling defects or signs of right cardiac cavity overload or deep vein thrombosis.

Factors which may contribute to misreading a CTPA examination may be patient-related (such as artefacts caused by breathing or heart movements), technical (such as X-ray beam hardening or

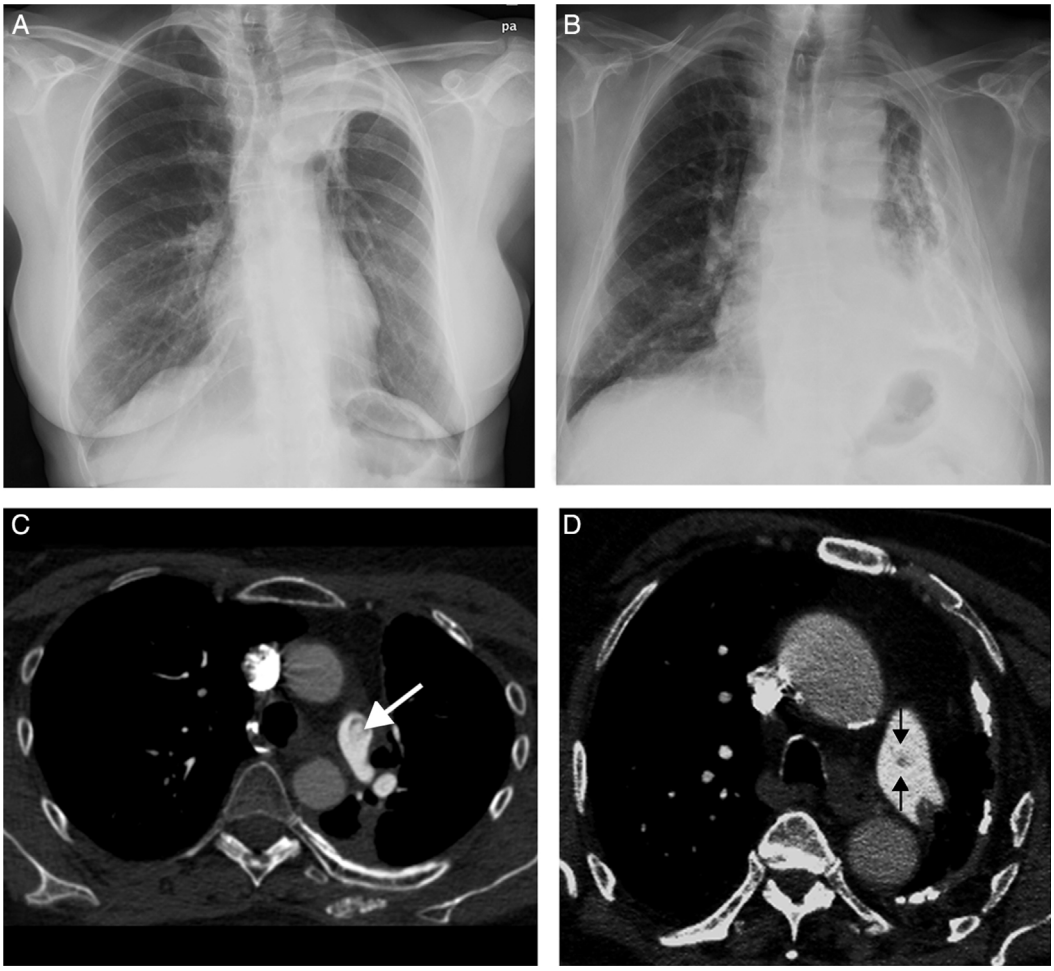

Fig. 1. (A) Posteroanterior chest X-ray of a 52-year-old patient (henceforth patient X) with a history of lung cancer treated with chemotherapy and radiation therapy 7 years previously, showing complete atelectasis of the left upper lobe. (B) Posteroanterior X-ray of an 85-year-old patient (henceforth patient Y), showing extensive left pleural thickening and calcification and significant loss of volume in the ipsilateral hemithorax. (C) Axial CT of patient X apparently showing a filling defect in the left main pulmonary artery (arrow), mistakenly interpreted as PTE. (D) Axial CT of patient Y apparently showing another filling defect in the left main pulmonary artery (arrows) that was also mistakenly interpreted as PTE.

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partial volume), or disease-related.[3] Disease-related artefacts have been reported to include impacted mucus in the bronchi, perivascular edema, or pulmonary artery sarcoma. However, as far as we are aware, this is the first description of structural changes (such as presented by our 2 patient) causing anatomical distortion of the pulmonary arteries, that in turn produced intravascular turbulence of blood and intravenous contrast flow.[4,5]

We believe that these 2 cases underline the importance of understanding the physiopathology and intravascular hemodynamic consequences of structural changes in the chest that cause an anatomical distortion of the pulmonary arteries, in order to avoid misinterpretation during CTPA procedures.

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Immune-mediated Leukopenia due to *Chlamydophila pneumoniae* Pneumonia☆

Leucopenia inmunomediada secundaria a neumonía por Chlamydophila pneumoniae

To the Editor,

Chlamydophila pneumoniae is known to affect the immune response of hosts, but this bacteria has not been described as a cause of autoimmune leukopenia. To our knowledge, this is the first report of a patient with *C. pneumoniae* pneumonia who developed antineutrophil IgG antibodies.

A 37-year-old woman presented with a 1-week history of cough, bloody sputum and fever. Physical examination showed temperature of 38 °C and crackles in right lung field. Chest X-ray showed bilateral alveolar infiltrates, corresponding with computed tomography findings of right lower lobe consolidation, and ground glass opacities in left upper lobe and both lower lobes (Fig. 1). ECG was normal. Arterial blood gases: pH: 7.52, $PaCO_2$ 34 mmHg, PaO_2 67 mmHg. Clinical laboratory tests: blood glucose 95 mg/dl, urea 18 mg/dl, creatinine 0.77 mg/dl, sodium 139 mmol/l, potassium 2.8 mmol/l, chloride 98 mmol/l, GOT 44 U/l, GPT 44 U/l, LDH 510 U/l, C-reactive protein 287 mg/l, procalcitonin 0.18 ng/ml, leukocytes 3080 μl, neutrophils (76.7%), hemoglobin 10.1 g/dl, platelets 222,000 μl. Quick time 68%. Microbiology: sputum, blood culture, and pneumococcal and *Legionella* urinary antigen tests negative. *C. pneumoniae* serology was positive in acute-phase serum, with elevated antibody titer with seroconversion (17/4/2013 IgG negative [0.157], IgM negative [0.307]; 2/5/2013 IgG indeterminate [0.992], IgM positive [1.428]) on micro-immunofluorescence. During hospitalization, leukopenia worsened to 1770/μl with neutrophil count of 850/μl in the presence of IgG-positive antineutrophil antibodies. Antineutrophil antibodies were detected with fluorescence-labeled polyclonal rabbit anti-human IgM and IgG antibodies (fluorescein isothiocyanate [FITC]) (DAKO). Cell suspension was analyzed using flow cytometry (FACS®, Becton Dickinson). The patient received levofloxacin for 3 weeks, resulting in radiological resolution of the pneumonia and normalization of blood panels.

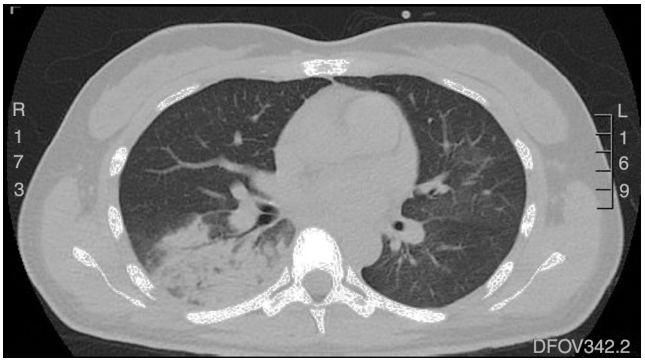

Fig. 1. Chest computed tomography showing right lower lobe consolidation.

C. pneumoniae is a Gram-negative bacteria, previously known as Taiwan acute respiratory agent (TWAR). It causes 10% of community-acquired pneumonias (CAP) in Europe (12% of CAPs that do not require hospitalization and 3% of those that do).[1,2] It may be responsible for immunological phenomena related to coronary arteriosclerosis,[3] but it is unusual for it to cause autoimmune leukopenia associated with antineutrophil IgG antibodies, as occurred in our case.

In autoimmune neutropenia, antibodies develop that act directly against cell membrane antigens, causing peripheral destruction of neutrophils. These antineutrophil antibodies promote phagocytosis of neutrophils opsonized by splenic macrophages.[4] Autoimmune neutropenia may be primary or secondary and is more common in adults. It is often related to autoimmune diseases (primary biliary cirrhosis, Sjögren syndrome, lupus erythematosus, and rheumatoid arthritis), as well as to exposure to medication (fludarabine, rituximab), solid tumors and blood cancers, neurological diseases, such as multiple sclerosis, or infections. Viral infections, such as human immunodeficiency virus, *es Parvovirus*, or Epstein–Barr virus, bacteria, such as *Helicobacter pylori*, *Escherichia coli*, *Neisseria meningitidis*, *Brucella* ssp., *Salmonella* spp. and *Mycobacterium tuberculosis*, and other pathogens, including *Toxoplasma gondii*, *Leishmania* spp. and malaria, have also been implicated. However, a review of the literature did not yield any report of autoimmune neutropenia developing as a result of *C. pneumoniae* infection.[5]

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