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Nonthrombotic pulmonary embolism

To the Editors:

We read with great interest the article in the *European Respiratory Journal* by JORENS *et al.* [1] on the different types of nonthrombotic pulmonary embolism. Among them, hydatid disease is of major importance as it is endemic in sheep-raising regions worldwide. We wish, therefore, to outline the management of metastatic pulmonary hydatidosis secondary to a primary hydatid cyst located in the liver according to our experience of six cases.

First, the diagnosis of hydatid pulmonary vascular obstruction should be looked for systematically in patients with a primary hepatic hydatid cyst located near the inferior vena cava and/or supra-hepatic veins, by perfusion lung scan and/or spiral thoracic computed tomography.

Secondly, once the pulmonary vascular obstruction has been diagnosed, the surgical treatment of the hepatic cyst at the origin of the embolism must be carried out as the first step. However, mobilisation of the liver may cause hydatid embolism and haemorrhagic complications when the hydatid cysts are in contact with the walls of the inferior vena cava. We suggest the following preventive measures: a wide laparotomy to control the inferior vena cava and have an extracorporeal bypass ready. These guidelines may prevent the fatal intraoperative pulmonary embolism as previously reported, and which occurred in two patients from our series [2].

Thirdly, surgery for a hydatid pulmonary vascular obstruction is quite similar to that for usual pulmonary embolism: embolectomy by arteriotomy for proximal pulmonary intra vascular hydatid cysts (five times in four patients in our series) using cardiopulmonary bypass in two cases.

Finally, chronic pulmonary arterial hypertension may worsen even after hydatid embolectomy as the vascular obstruction is also distal and associated with a granulomatous reaction and vascular fibrosis [3]. Pulmonary transplantation may be a therapeutic option because immunosuppressive treatment does not adversely affect the course of hydatid disease [4]. Recently, the use of pulmonary hypertension medical therapy, such as endothelin-1-receptor antagonists, phosphodiesterase-5-inhibitors and prostacyclin analogues, has been reported to be clinically effective [5].

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Effects of nanoparticles on lung damage in humans

To the Editors:

We read with great interest a recent article in the *European Respiratory Journal* by SONG *et al*. [1] and we would like to add some questions/comments to their paper. First, we are interested in the (estimated) total weight of the polyacrylate nanoparticles detected in the lungs and/or pleural fluids obtained from the patients. In our *in vivo* experiments, mice were exposed seven times per week to nanoparticles (carbon black [2] and latex [3]) at the weight of 50 $\mu g \cdot time^{-1} \cdot animal^{-1}$ (~83 mg in the human body), and did not die within 6 months thereafter (unpublished observation). Taken together, results