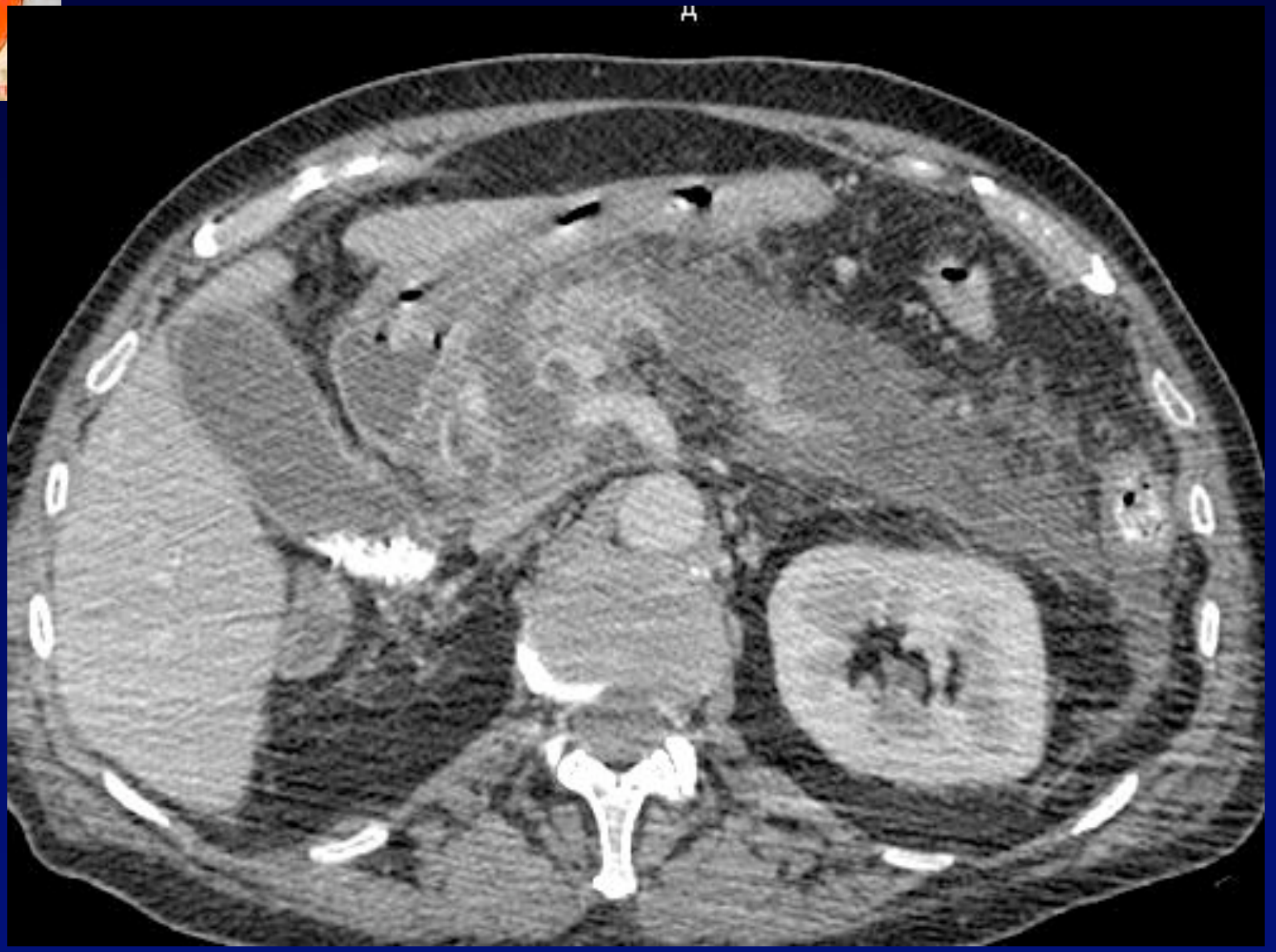
Alexis LACOUT

Homme de 73 ans

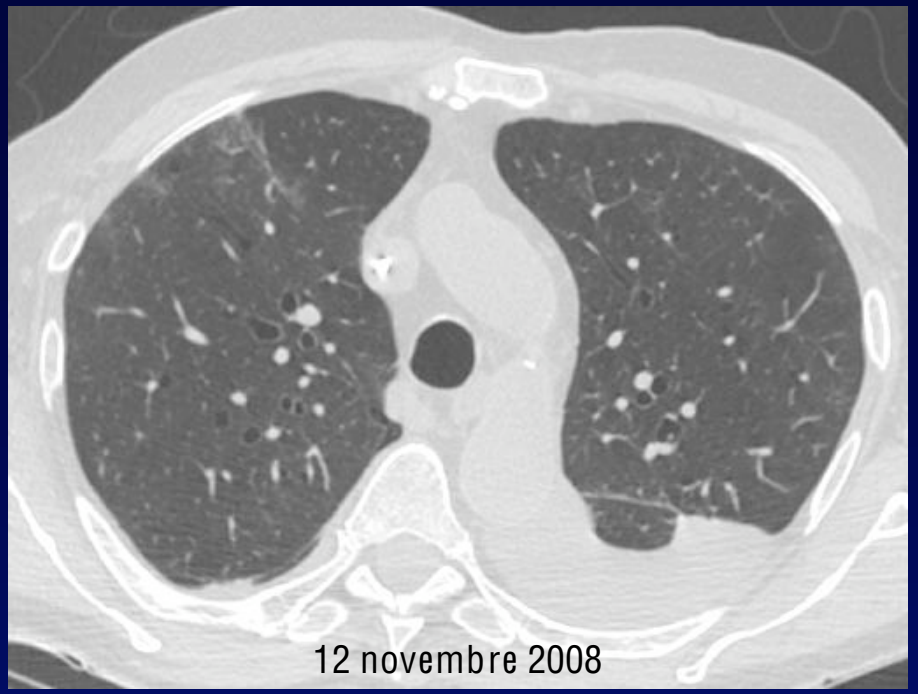
Pancréatite aigue grave nécrosante d'origine biliaire



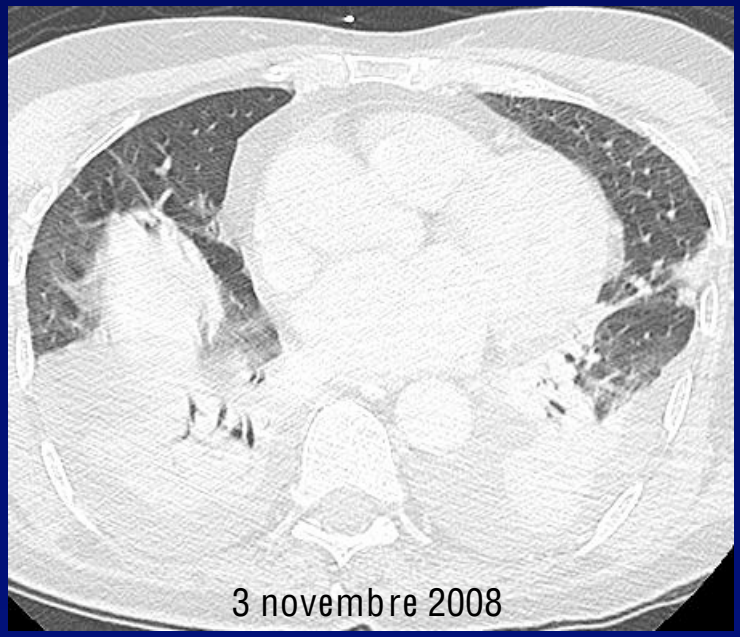
3 novembre 2008

Premier scanner datant
du 3 novembre 2008

3 novembre 2008



12 novembre 2008



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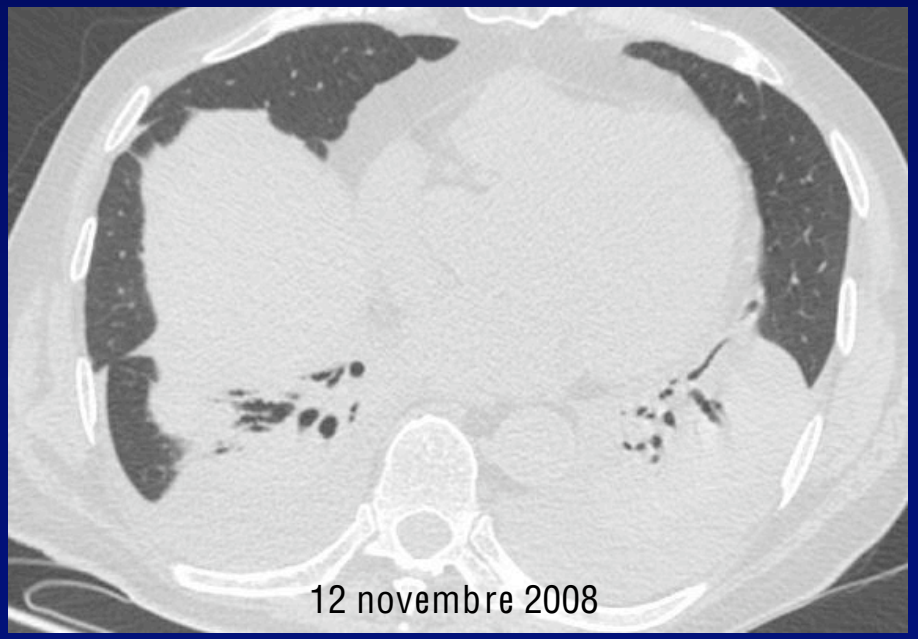
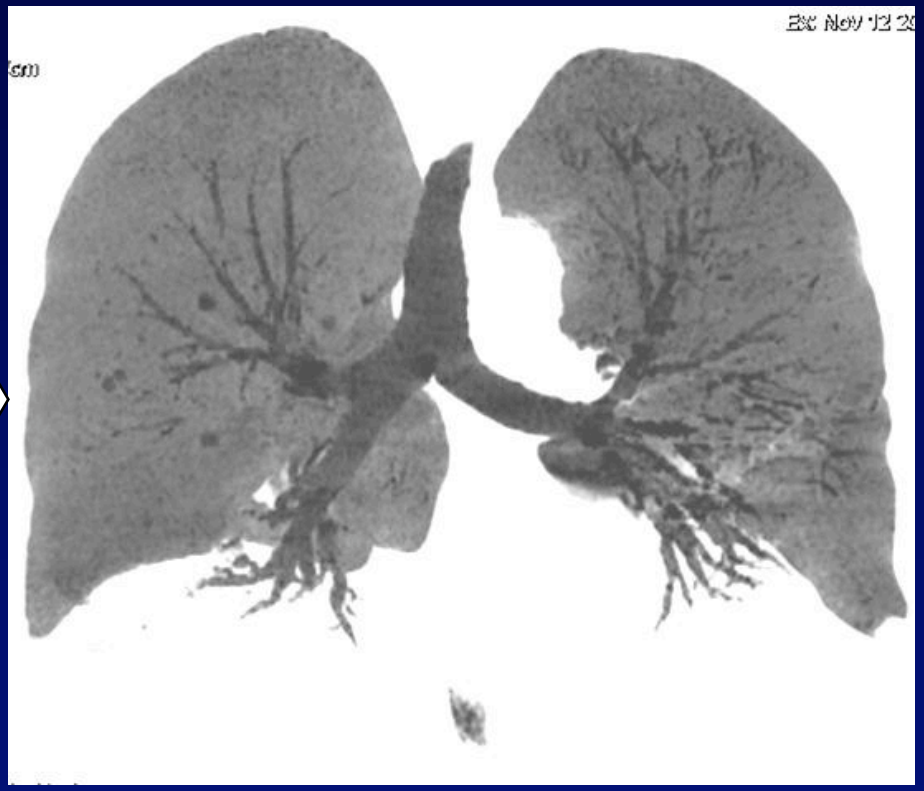


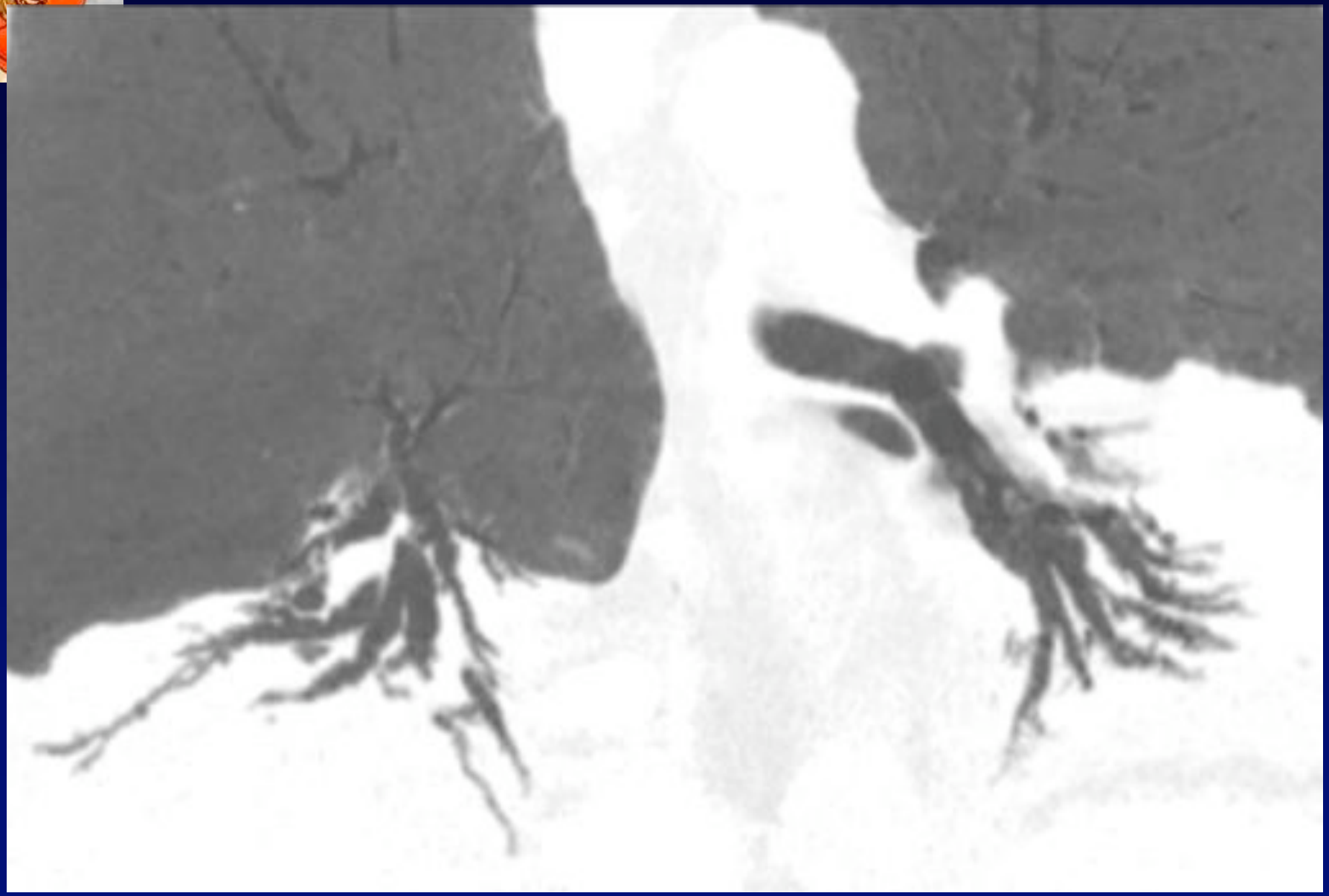
12 novembre 2008

Coronal minimip

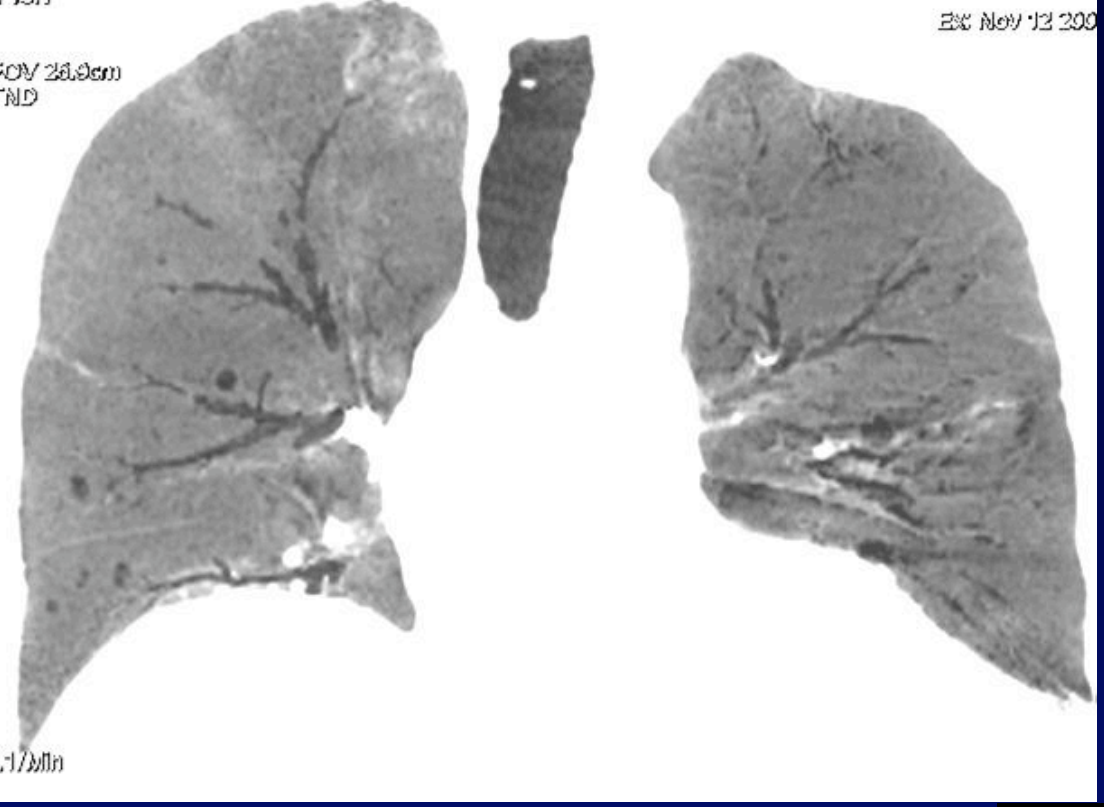
3 novembre 2008

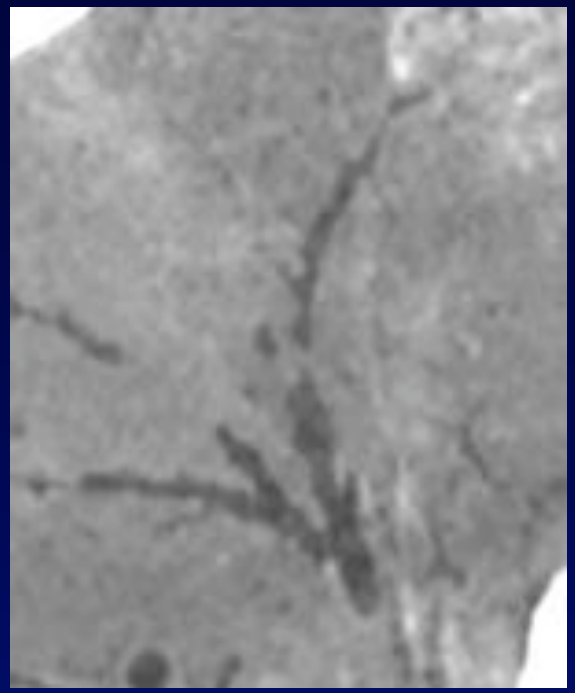


12 novembre 2008

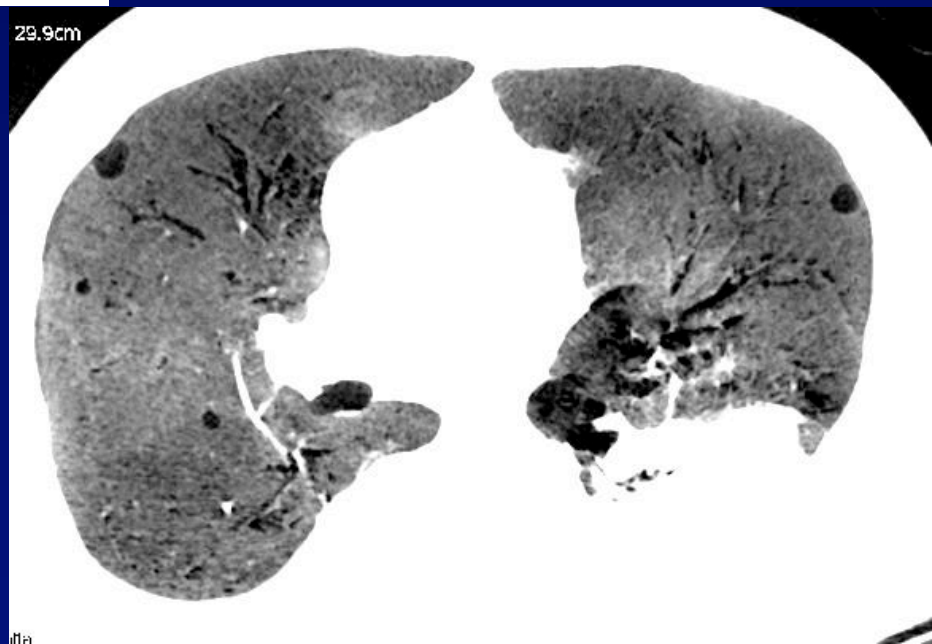


12 novembre 2008





12 novembre 2008

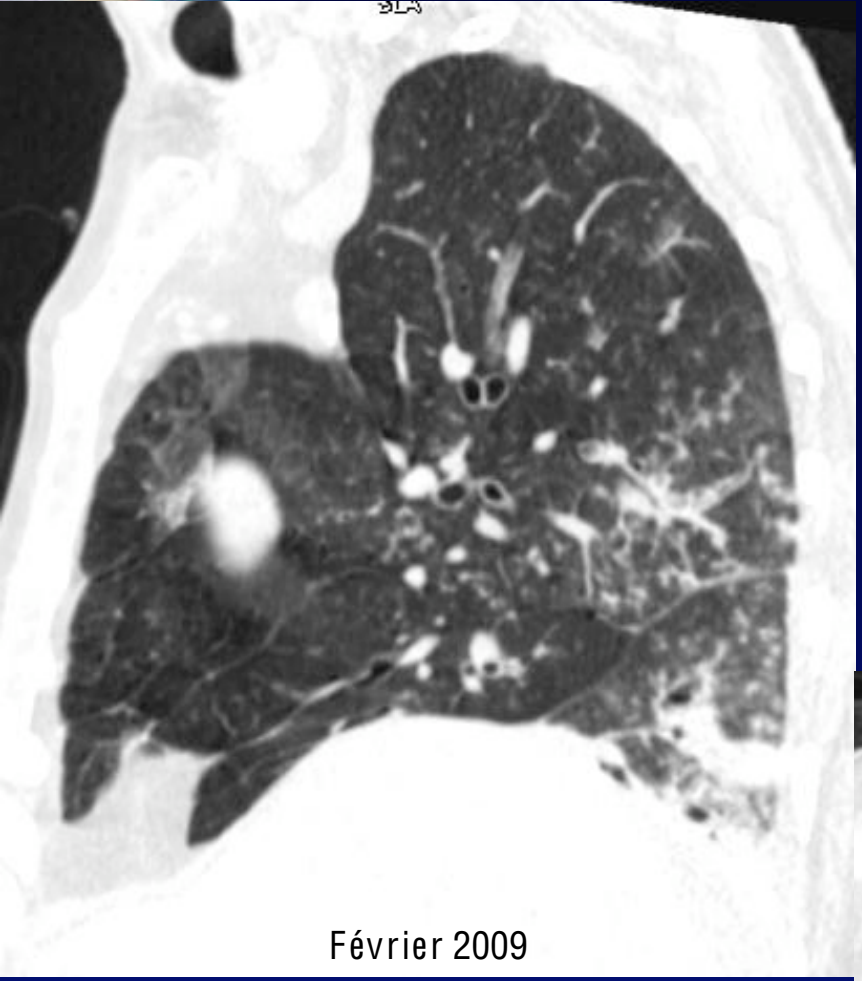


Diagnostic ?

Diagnostic

Condensations parenchymateuses

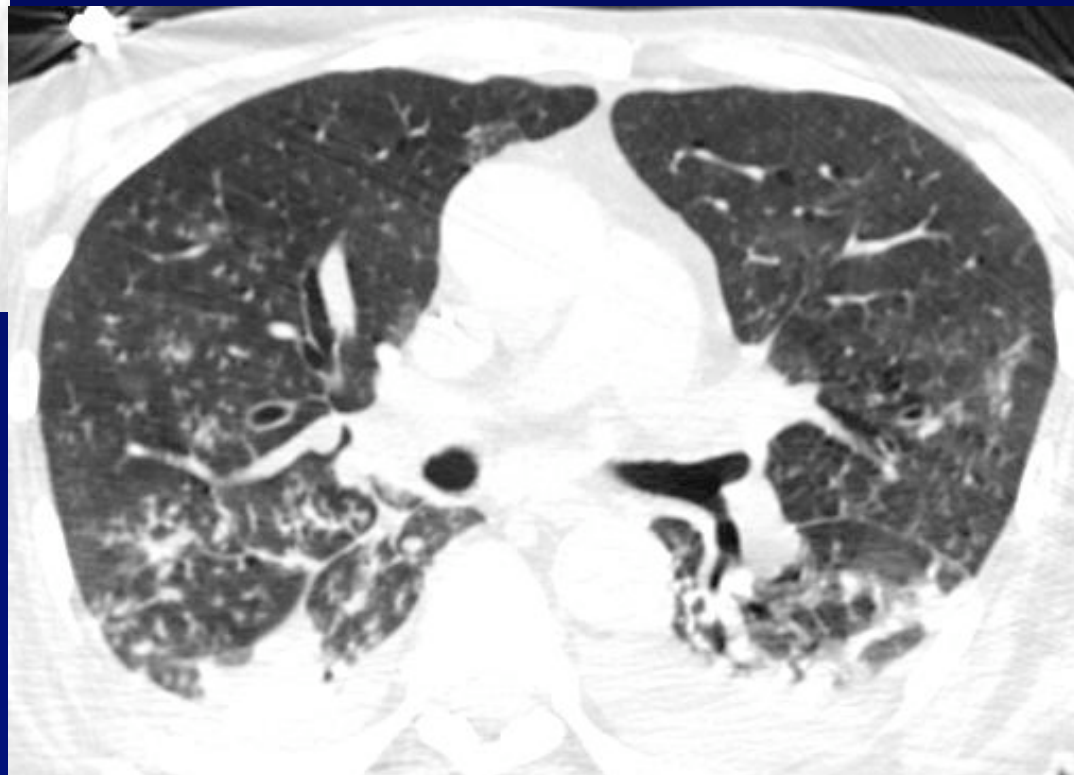
Apparition de bronchectasies **en 9 jours : secondaires à l'atteinte** pulmonaire ? Mécanisme ?



Février 2009

Persistance des DDB ;
Bronchiolite

Suivi 3 mois après



Pancréatite aigue et Poumon

Imagerie : Infiltrats secondaires à une augmentation de la perméabilité capillaire ; corrélés à la mortalité

Clinique : simple hypoxémie jusqu'au SDRA

Cytokines, enzymes pancréatiques circulantes → poumon d'autant plus facilement qu'il existe une augmentation de la perméabilité capillaire

Catherine M. Pastor, Michael A. Matthay and Jean-Louis Frossard. Pancreatitis-Associated Acute Lung Injury. New Insights. CHEST 2003; 124:2341–2351

and lymph. Thus elevated concentrations of cytokines and pancreatic enzymes might be associated with the development of lung injury, but additional information is needed. Additionally, patients with

recently in another study.[15] Thus, these studies provide reasonable evidence that the pulmonary infiltrates and pulmonary edema in most patients with acute pancreatitis can be attributed to an increase in lung vascular permeability.

Alveolar-capillary membrane injury may result from the pulmonary absorption of proteolytic enzymes and vasoactive substances released by the injured pancreas via the veins and lymphatic vessels that drain the pancreas and the peripancreatic tissues. Two

Catherine M. Pastor, Michael A. Matthay and Jean-Louis Frossard. Pancreatitis-Associated Acute Lung Injury. New Insights. CHEST 2003; 124:2341–2351