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Mme JAMILA A..53 ANS m'a consultée le 16/04/2013 pour dyspnée progressive depuis 2 mois chez une cardiaque

ANTECEDENTS ET TARES :

DIABETIQUE (5ans)

VALVULOPATHIE OPEREE EN 2008

(Cordarone-Warafine.Aladactone-Lasilix)

EXAMEN PHYSIQUE :

TA: 14/8 - RC : 88 /min .**Sat: 88 %.** – crepitantes très fine- Goitere et d'adenoptahies cervicales-

GDS: **Po2: 55- Pco2 : 34** -Hco3- : 23 PH:7,45

EXAMENS BIOLOGIQUES :

Glycemie :140

NFS: GB = 14700 10 3/mm3- HB= 11,70 gr/l - PLAQUETTES = 103/mm3

CREATININE:7,78

IONO: NA:139. K: 4,3 CL:102

CPK:114.LDH:478

ProBNP:61

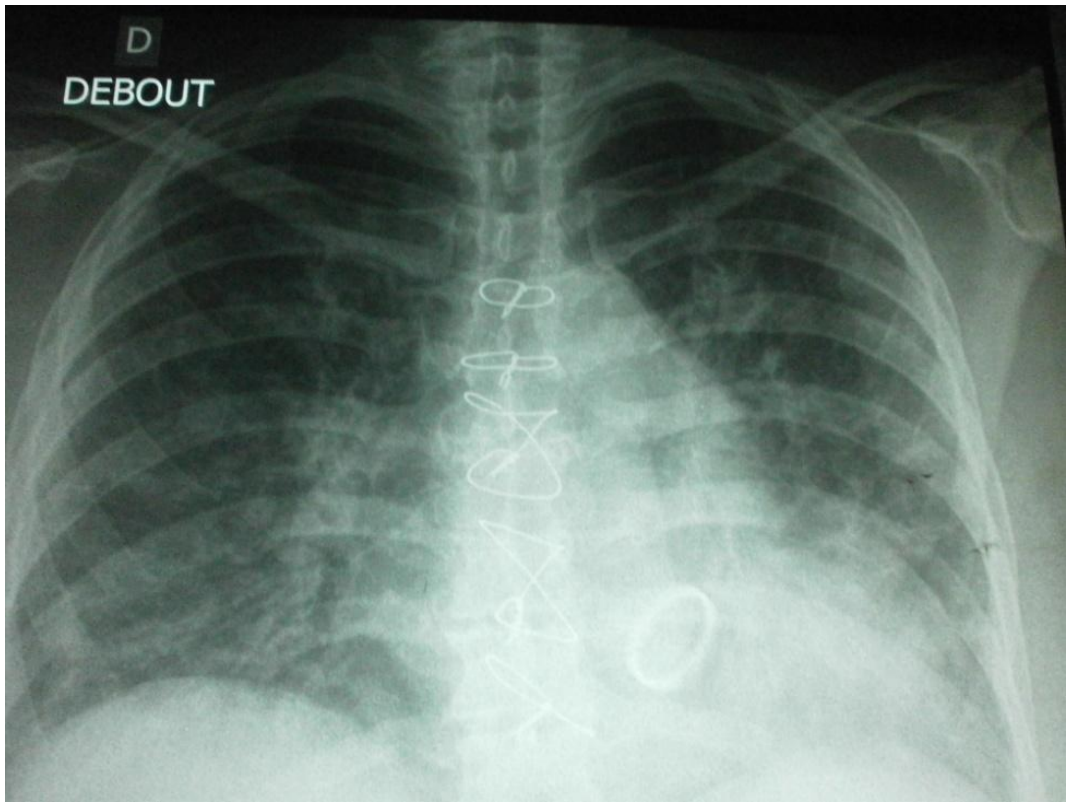
HIV:NEGATIF

E.C.G :NORMALE

ECHO CARDIO :BONNE FONCTION VG .valve en place

Echographie CERVICALE:

Goitre thyroïdien avec hypertrophie globale.Présence de quelques petites adénopathies jugulo-carotidiennes bilatérales

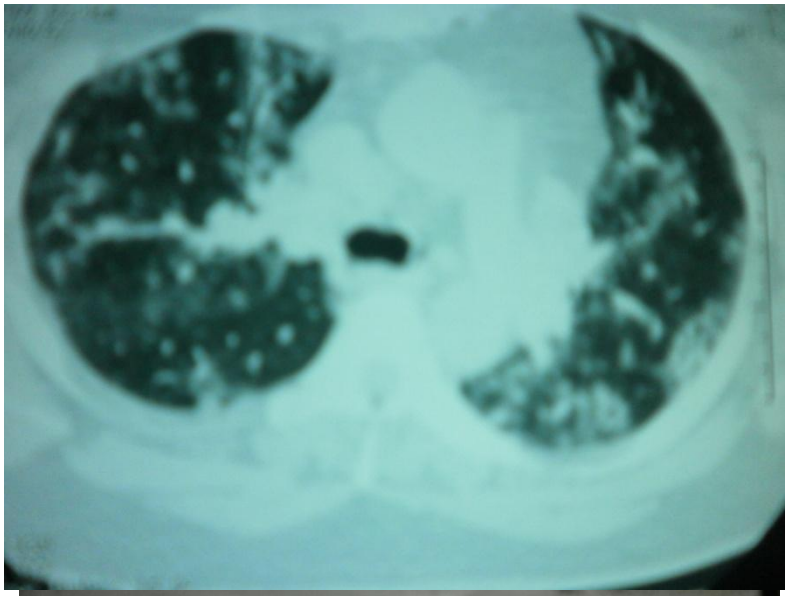


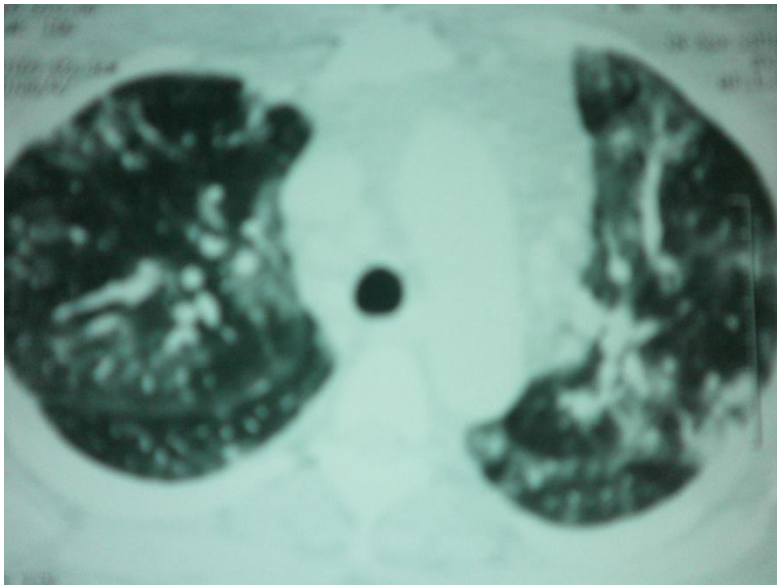
RX THORAX FACE:

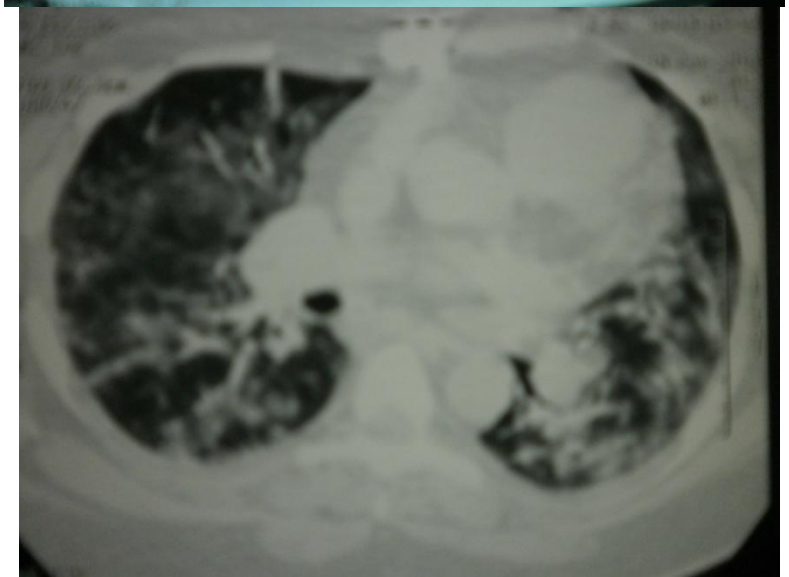
CARDIOMEGALIE MODEREE - SYNDROME INTERTIELLE -STERNOTOMIE -VALVE MITRALE

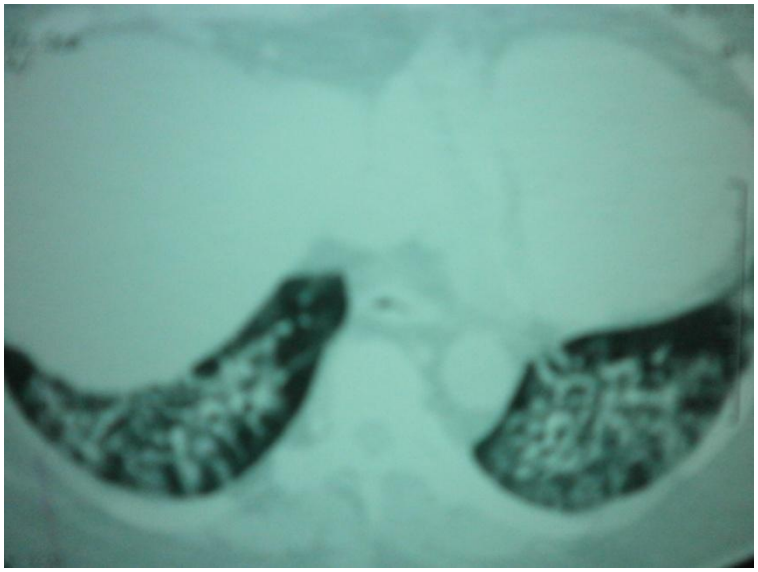
Syndrome interstitiel bilatérale :Nodulaire +ver dépolie +Réticulation

MICRONODULAIRE Centro lobulaire a contour floue non en bourgeon d' arbre. **Ver dépolie** en carte géographique.
Reticulation sous pleurale = ligne septale épaissie irrégulièrement (Chroniciteé)

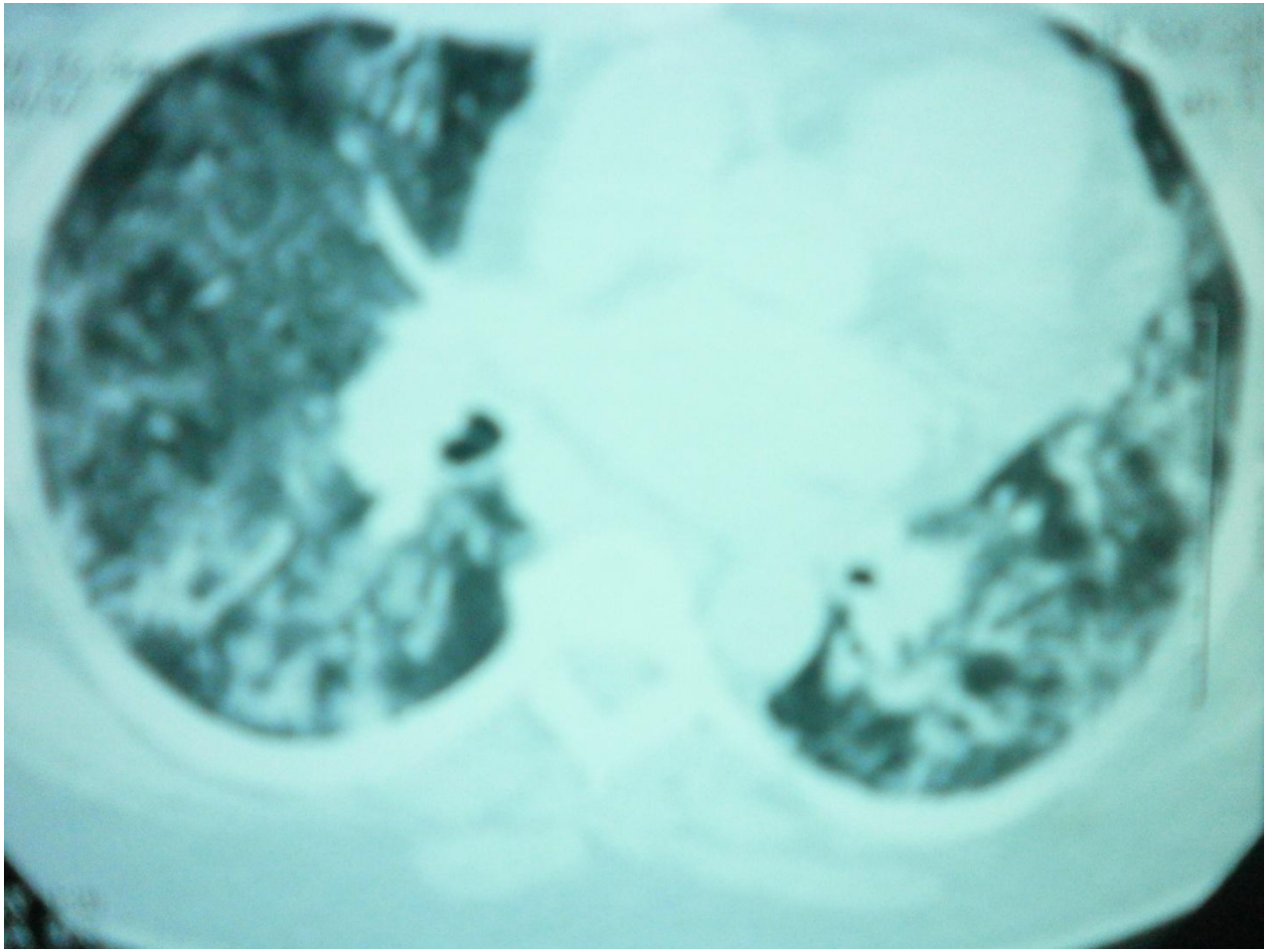








Il s' agit d' une femme de 52ans -valvuolapthie opérée en 2008
PNEUMOPTHIE INTERTIELLE AUX STADE D'HYPOXIE –
"PNEUMOPATHIE A LA CORDARONE"



Cli Res Car 2010 Nov;99(11):693-700. doi: 10.1007/s00392-010-0181-3. Epub 2010 Jul 10.

Amiodarone-induced pulmonary toxicity: an under-recognized and severe adverse effect?

Schwaiblmair M, Berghaus T, Haeckel T, Wagner T, von Scheidt W.

Source

martin.schwaiblmair@klinikum-augsburg.de

- Several forms of pulmonary disease occur among patients treated with amiodarone, **i.e. chronic interstitial pneumonitis, organizing pneumonia, ARDS, a solitary pulmonary mass of fibrosis. The prevalence is estimated to be about 5%.** Two major hypotheses of amiodarone-induced pulmonary injury include direct cytotoxicity and a hypersensitivity reaction. Given the frequency and potential severity of amiodarone-induced pulmonary toxicity, early detection is desirable. Unfortunately, there are no adequate predictors of pulmonary toxicity due to amiodarone. Patients who should benefit from amiodarone should be carefully selected and the lowest effective dosage of amiodarone should be taken. Amiodarone-induced pulmonary toxicity is a diagnosis of exclusion. Pulmonary evaluation with chest X-ray and pulmonary function testing, including diffusion capacity for carbon monoxide is recommended when amiodarone is started. A documented decline in the diffusing capacity of greater than 20% is useful in suggesting the need for closer monitoring or for further diagnostic testing. Although the optimal frequency of follow-up has not been determined, most cases of amiodarone-induced lung injury develop during **the first 2 years of treatment** and disease onset usually is slow. Pulmonary function tests and imaging may be performed every 3-6 months, depending on the presumed individual risk. Treatment of amiodarone pulmonary toxicity consists primarily of stopping amiodarone. Corticosteroid therapy can be life-saving for severe cases and for patients with less severe disease in whom withdrawal of amiodarone is not desirable. Due to its accumulation in fatty tissues and long elimination half-life, pulmonary toxicity may initially progress despite drug discontinuation and may recur after steroid withdrawal. The prognosis of amiodarone lung disease is generally favourable.
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