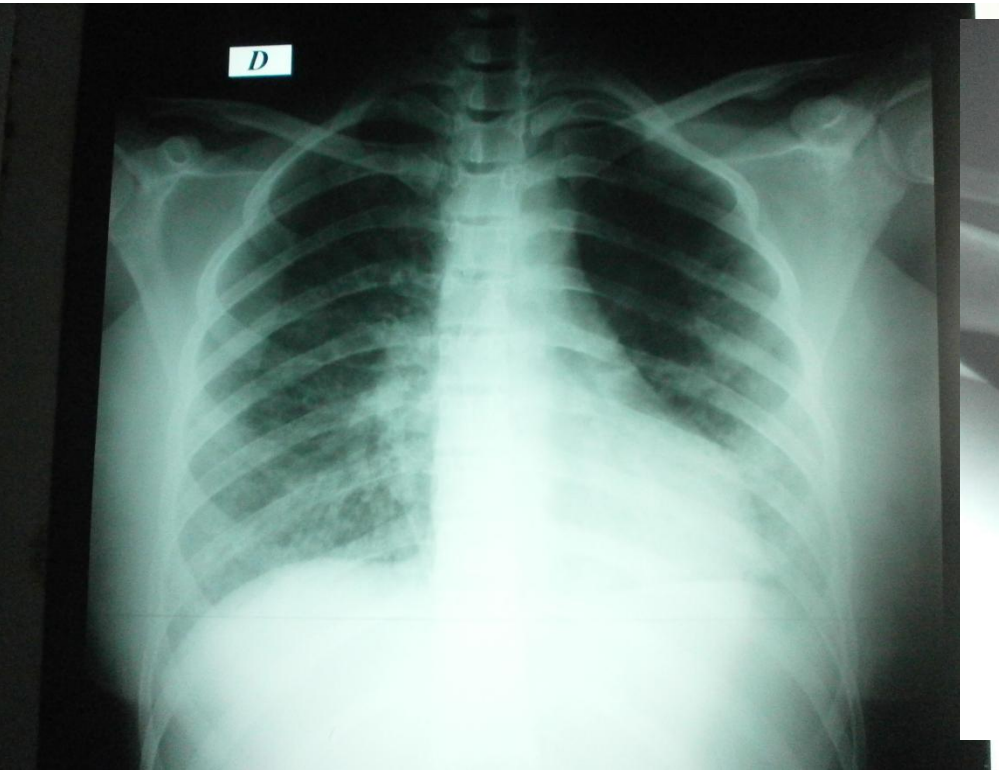
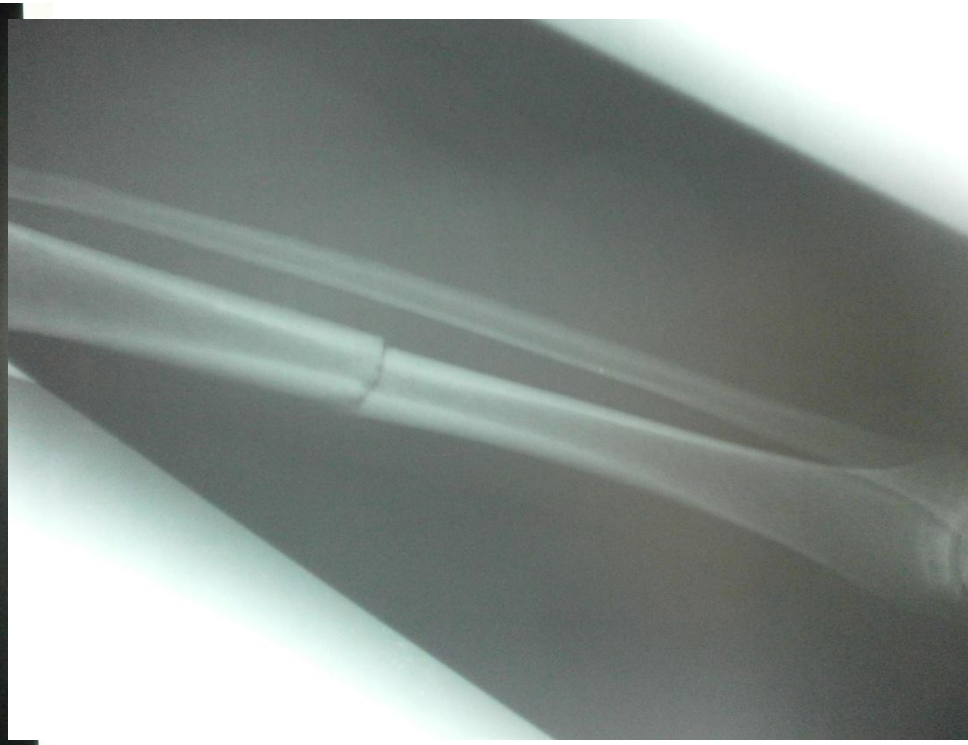
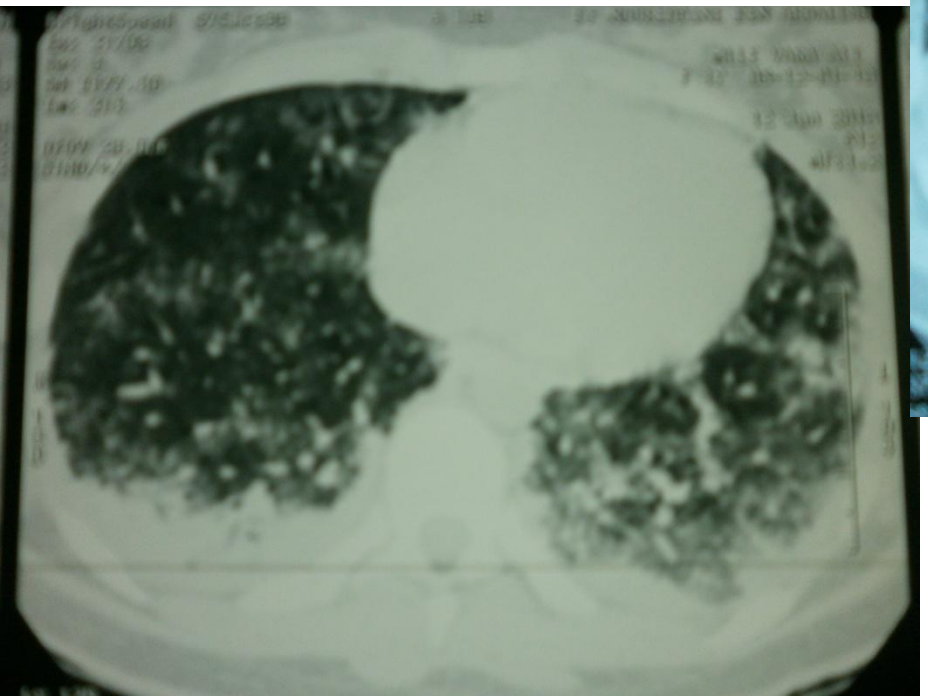
Mlle yosra A..18 ANS dyspnée, desorientation aux decours d’un fractues du Tibia.

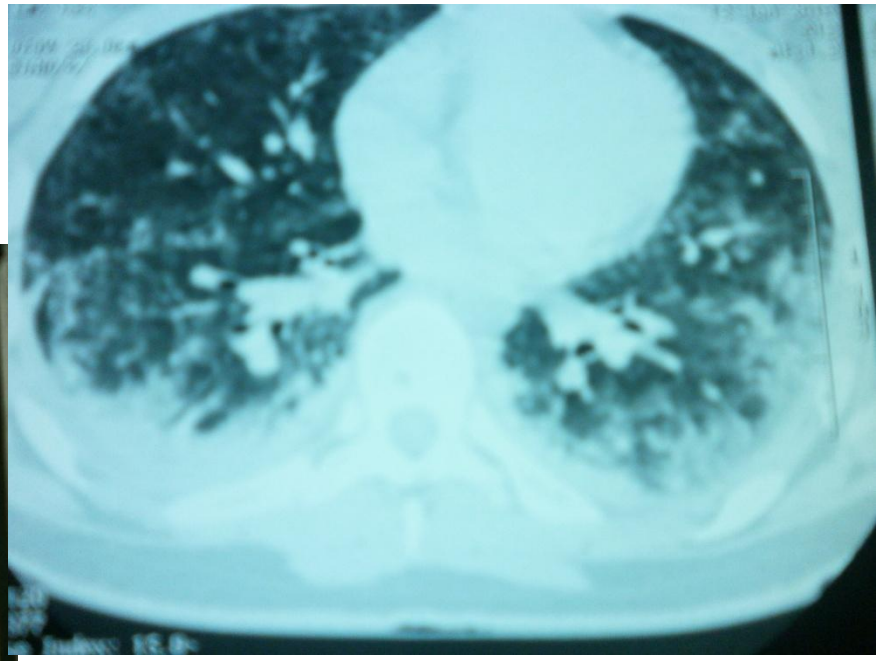
Po2 =55.Pco2 =29



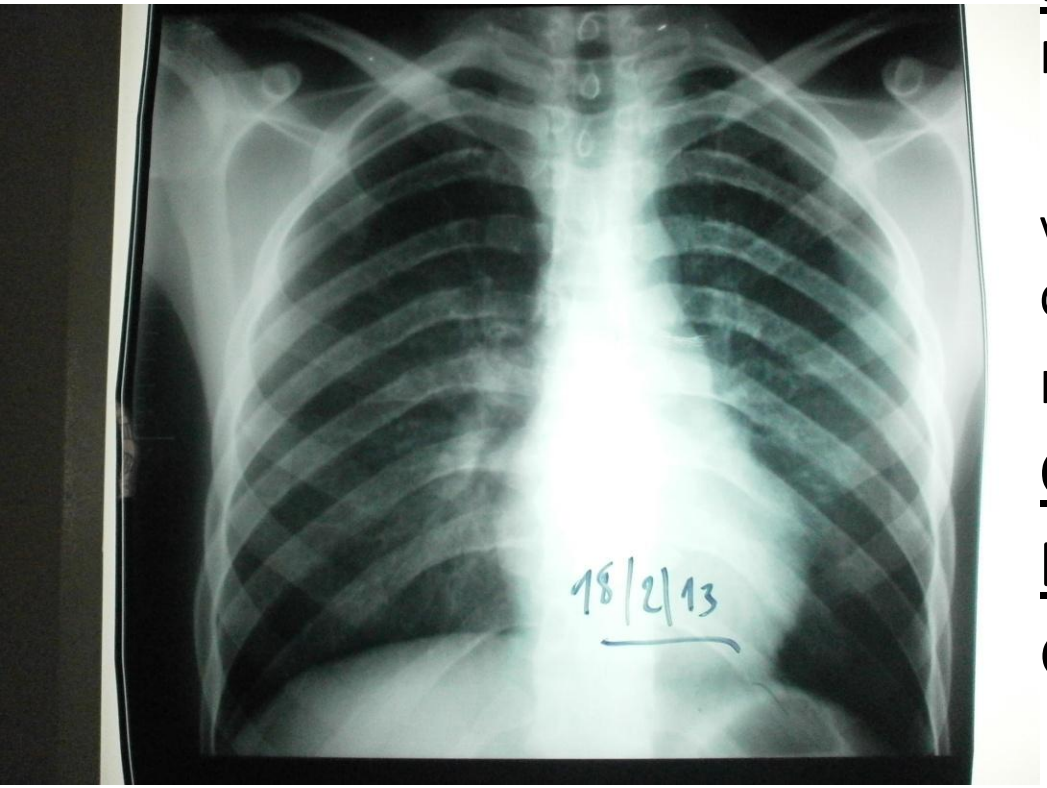


EMBOLIE GRAISSEUSE





Mr DHOUIB M…23 ANS M' CONSULTATIE LE 20/02/2013 pour HEMOPTISIE + DOULEUR THORACIQUE depuis 15 JOURS



EXAMENS BIOLOGIQUES

NFS: GB = 1170 10 3/mm3- HB= 5 gr/l - PLAQUETTES = 362000 103/mm3

VS : 1ér H: 30. 2ére H:60

CREATININE:40mg/L

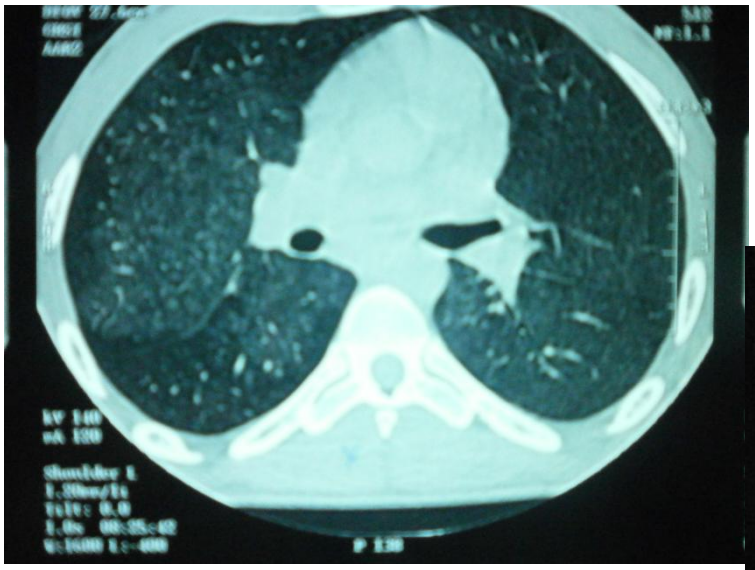
HIV:Nègatif

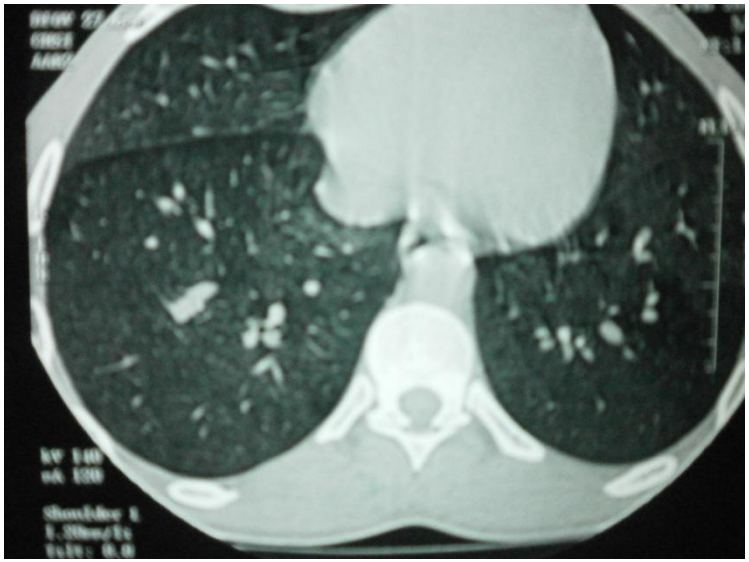
CRACHAT :2 BK:Nègatif

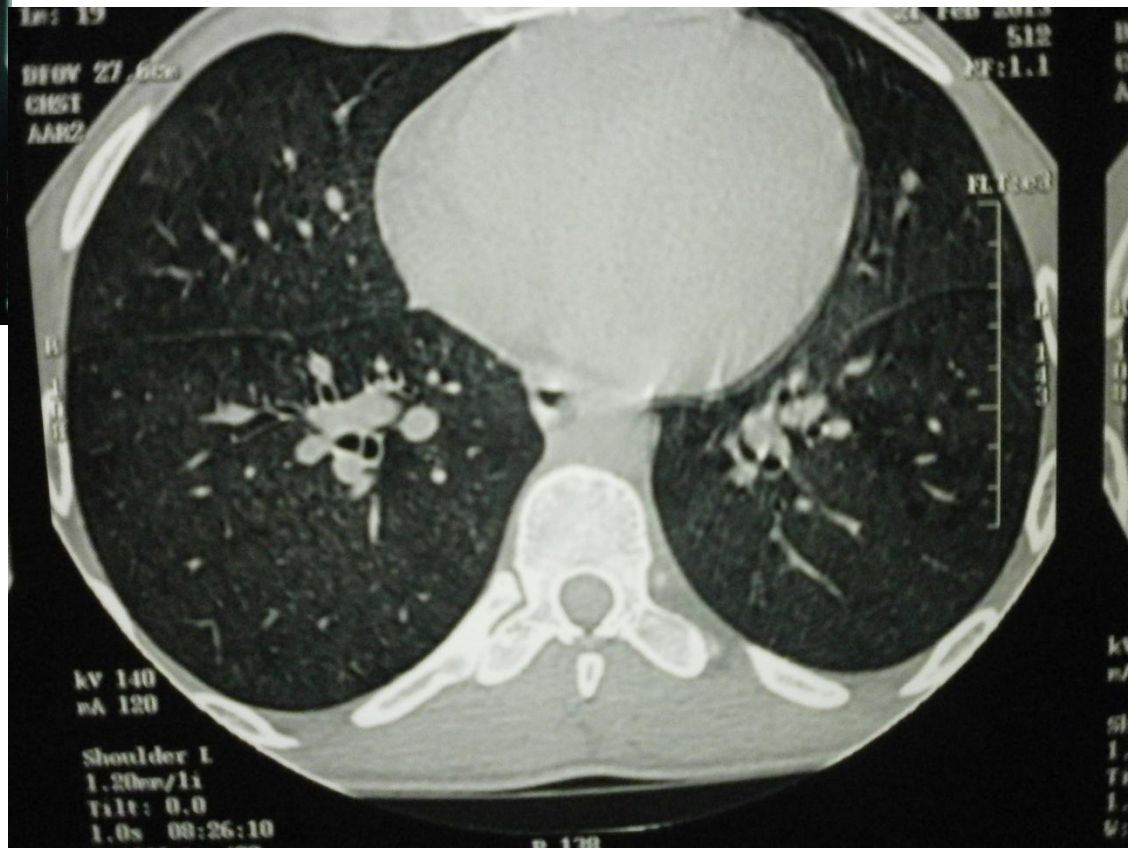
Biopsie rénale:

Glomérilonephrite extramembranaire

Hémorragie alvéolaire :Ver dépolie .Micronodule centrolobulaie a contour flous non en arbre de bourgeon



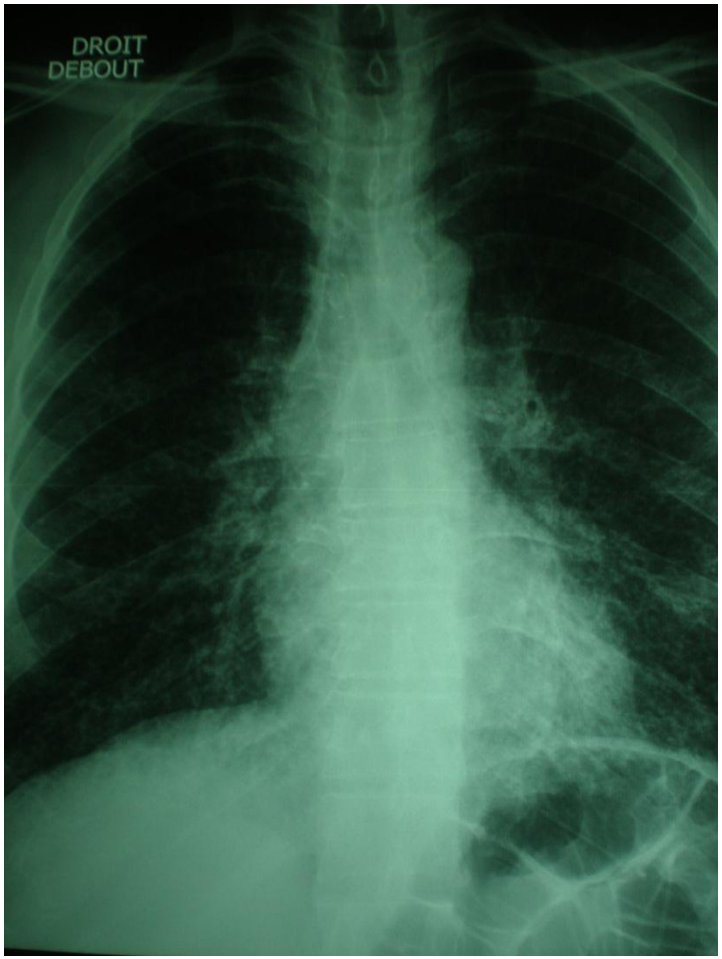


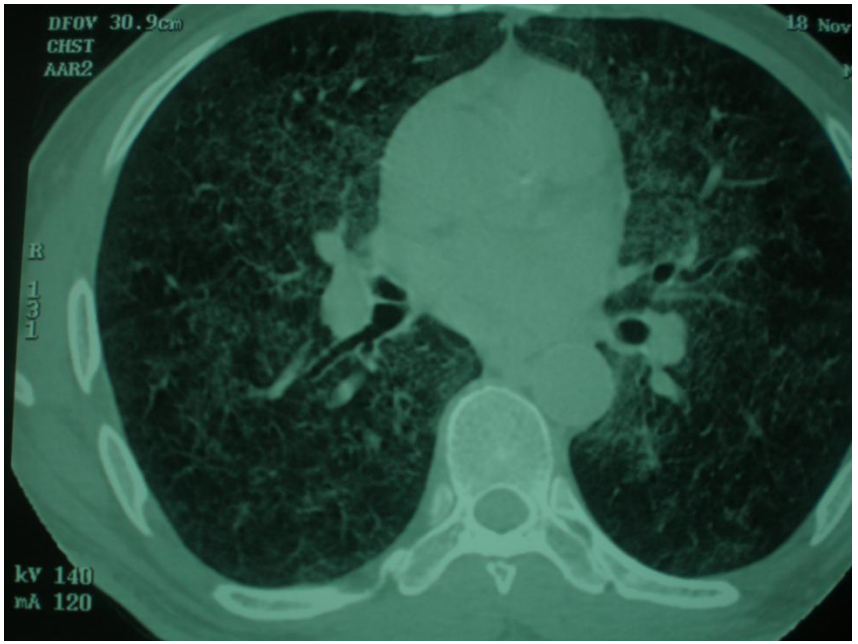


Pneumocystose

RX Thorax :s inertielles a minima.

Scanner thoracique: Ver dépolie et microkyste





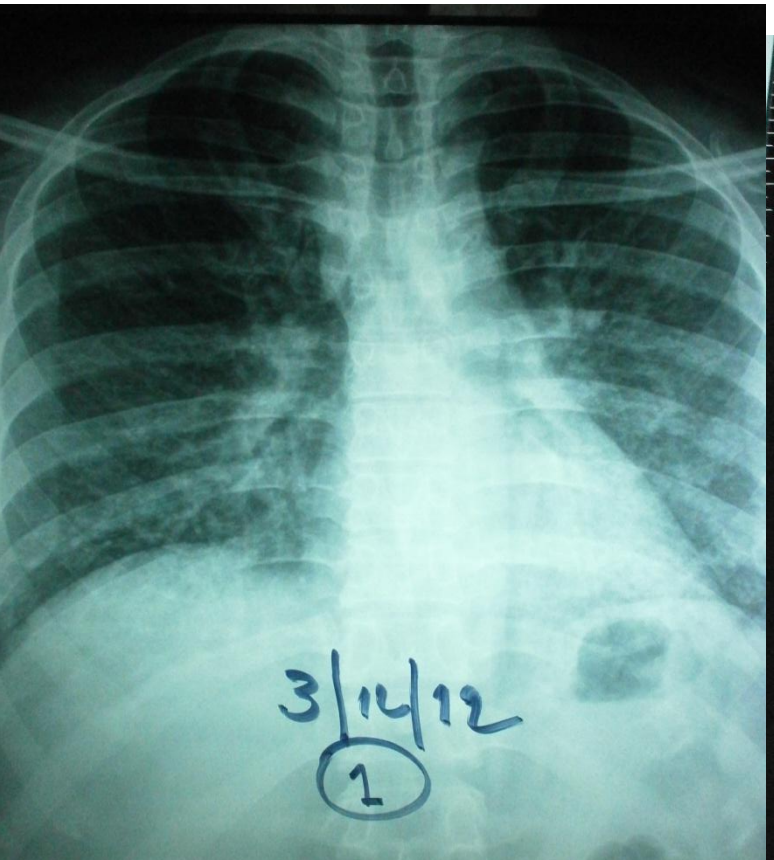
BOUBAKER M…37 ans m' a consulteé le 19/02/13. HIV +, SS ANTIVIREAUX et NIAZIDE DEPUIS 1ANS .. .. 3 SEMAINES TOUX DYPNEE ET FIEVRE..Majoration de la dyspnée sous Bactrim et AZYTHROMYCINE;

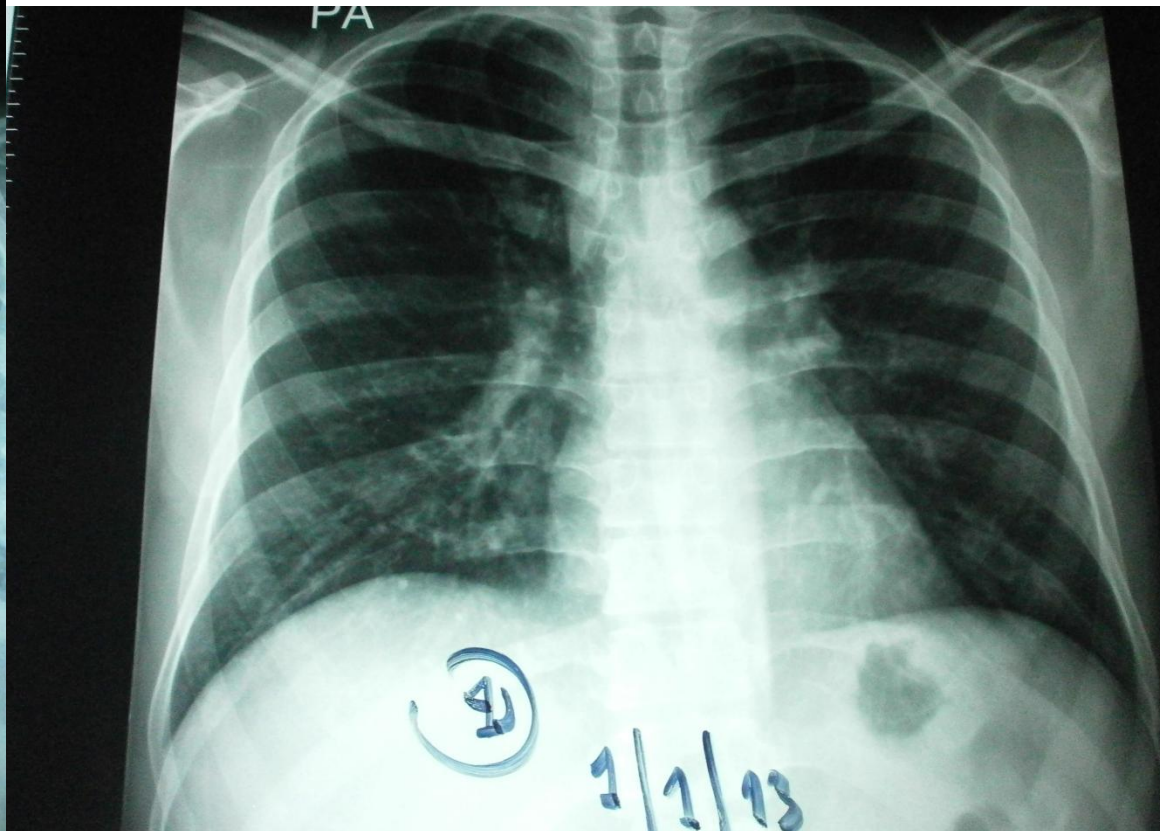
GDS : Po2: 58 Pco2 : 27 So2:92 Hco3- : 21 PH:7,50 -→Non modifiée sous 6 lires d' oxygène.

PNEUMOCYSTOSE /SDRA

-> SOLUPRED 20 mg. 3 cp(3 j)puis 2cp(7j)puis 1cp(15j)puis 1/2cp(15 j)

CONSULTATION N° 2 (07/01/2013) Recul : 19 jours--→NETTE AMELIORATION CLINIQUE ET RADIOLOGIQUE

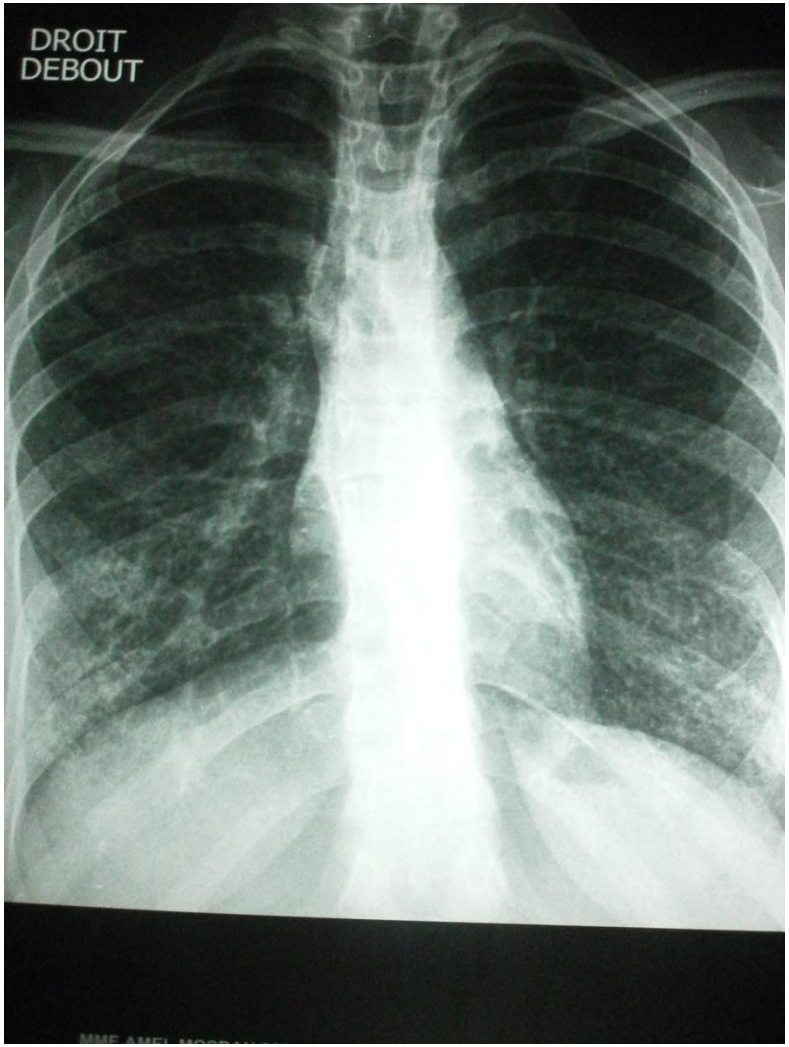


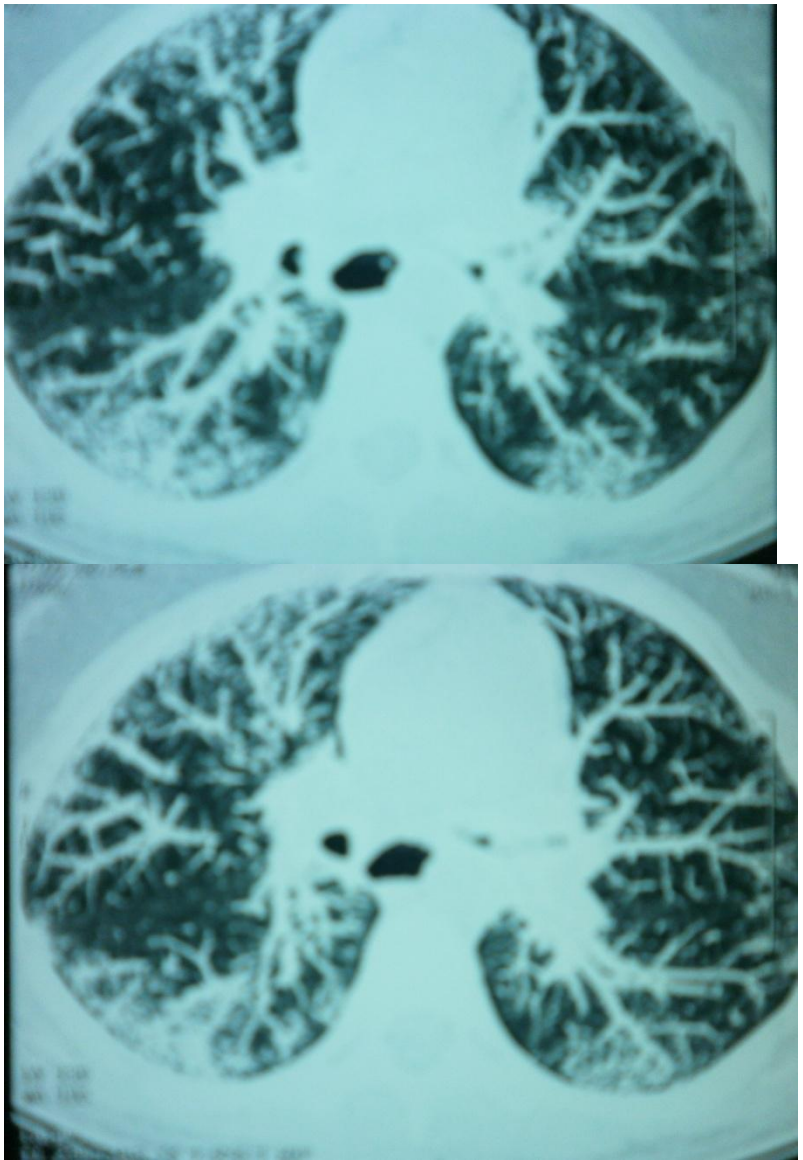


Mlle AMAL M..32 ans m'a consultée le 16/1/13 pour dyspnée ,toux et asthénie chronique.

RX THORAX FACE: S intertielle prédominant aux 2 bases et en périphérie.

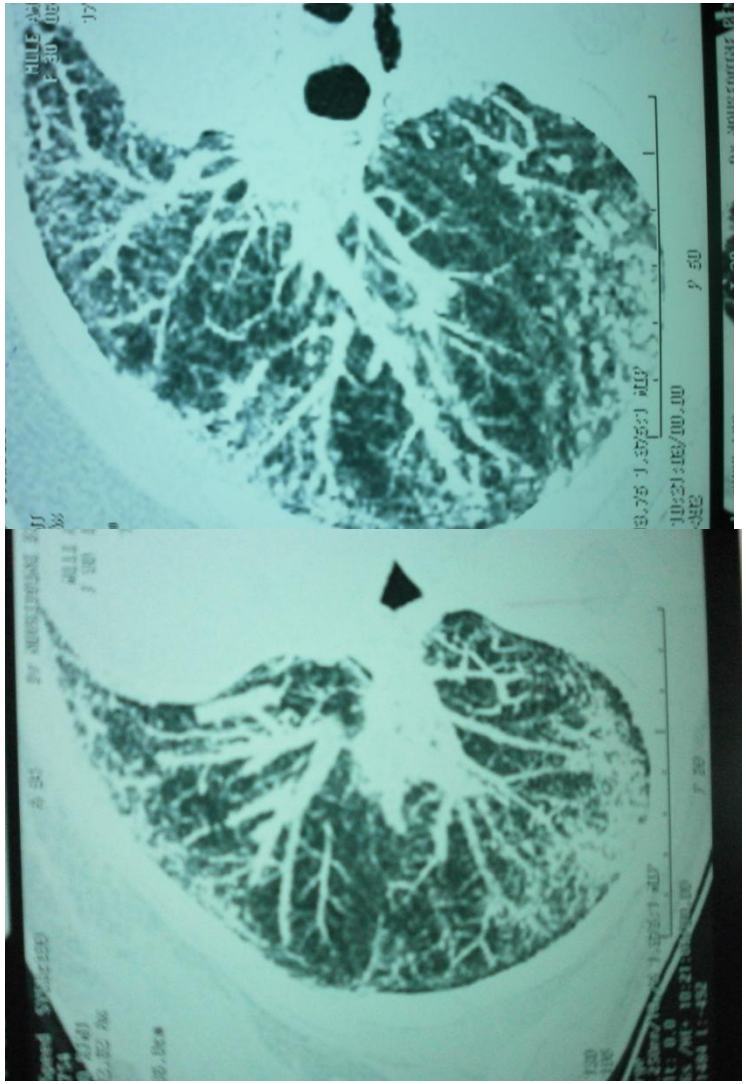
Scanner thoracique: P I D Type micro-nodulaire

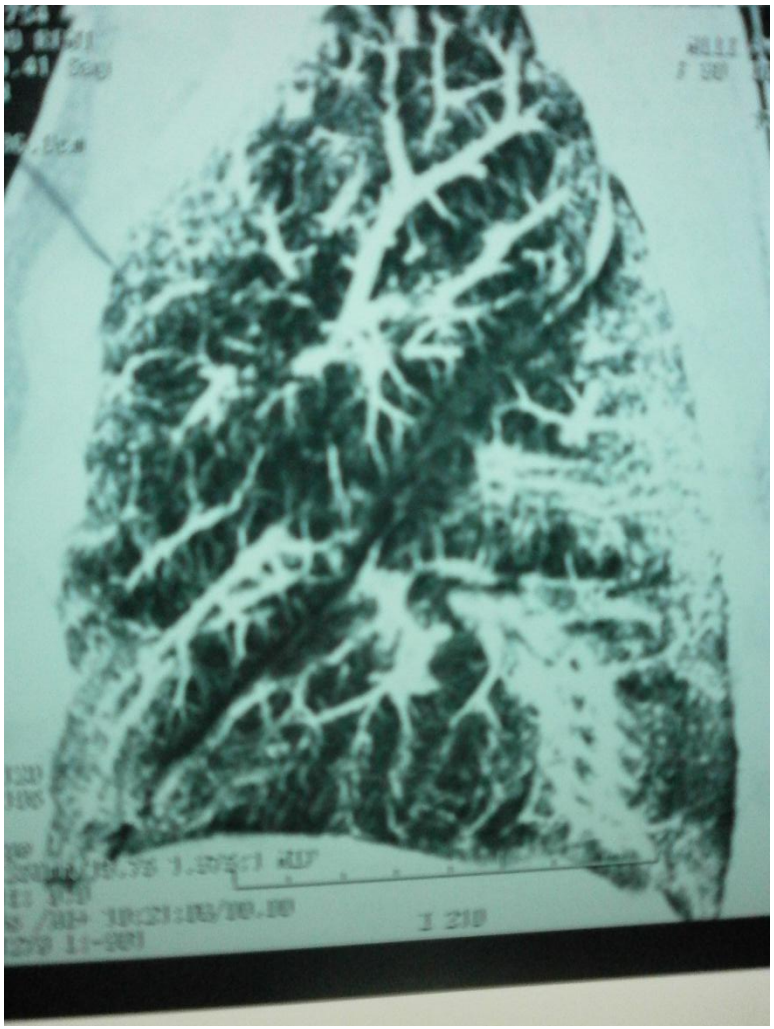




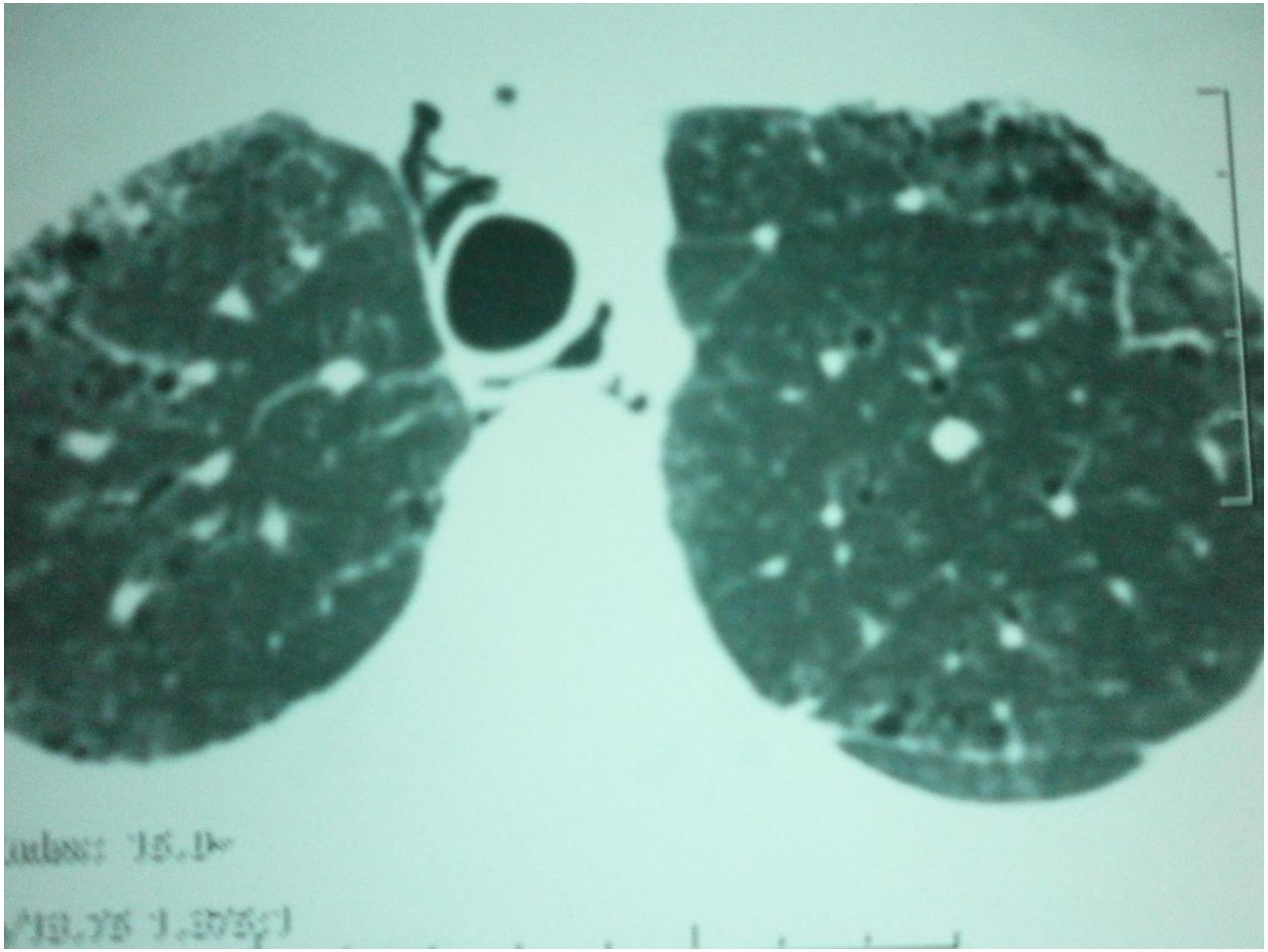
Micronodules distribution peribronchiolaire avec aspect en bourgeon d'arbre.

= Atteinte bronchiolaire (Chronique ?? TBC ??)

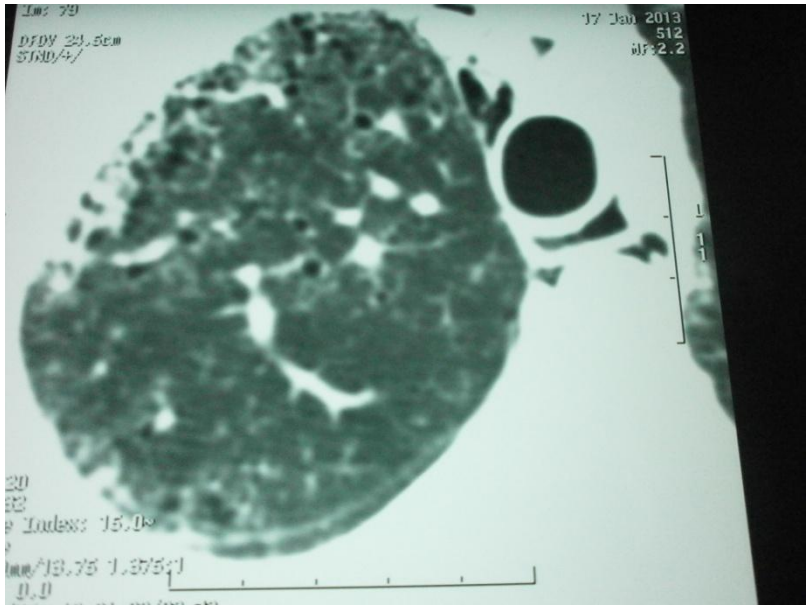


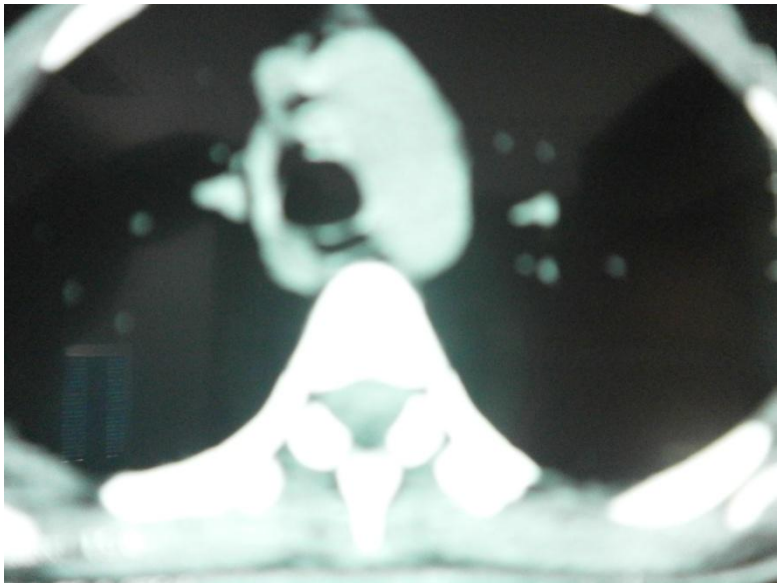


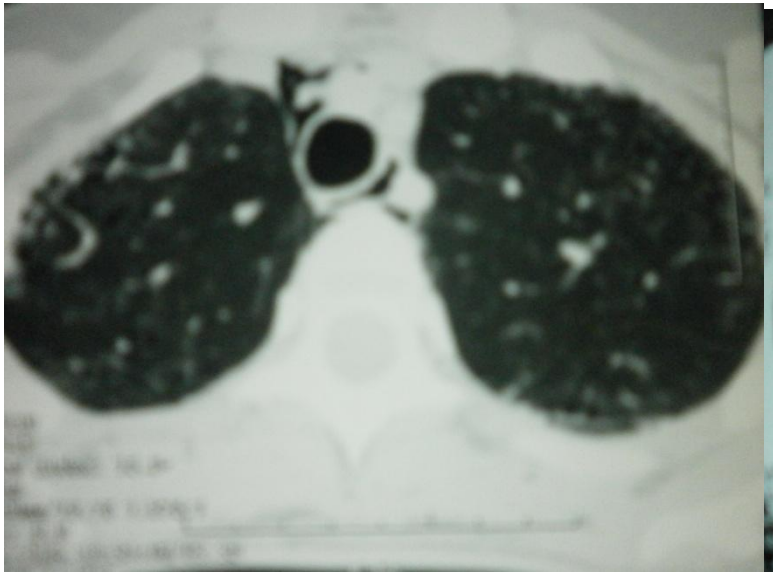
Quelques lésions kystiques et réticulations sous pleurale apicale.
+ Pneumomédiastin ??

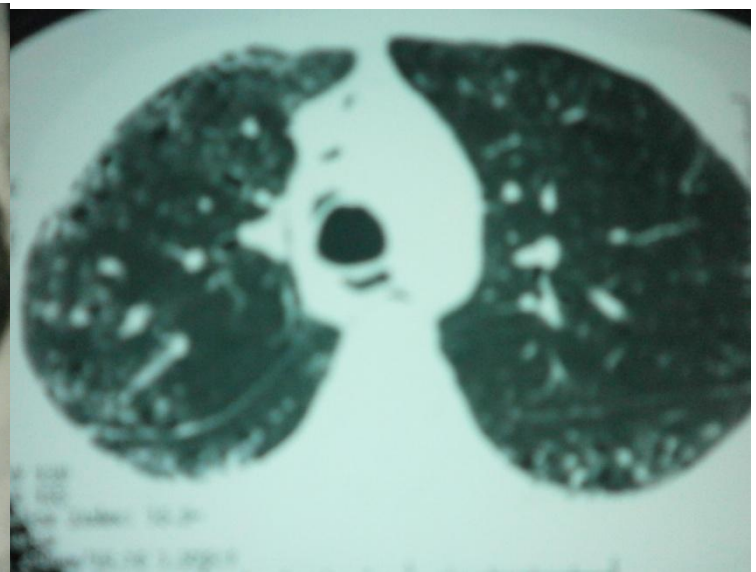


Quelques lésions kystiques et réticulations sous pleurale apicale.
+ Pneumomédiastin ??

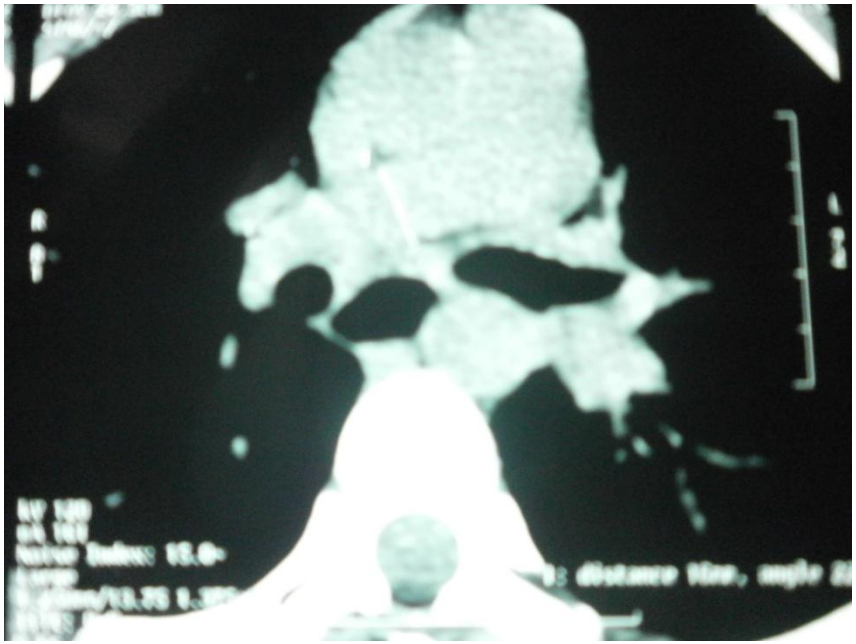


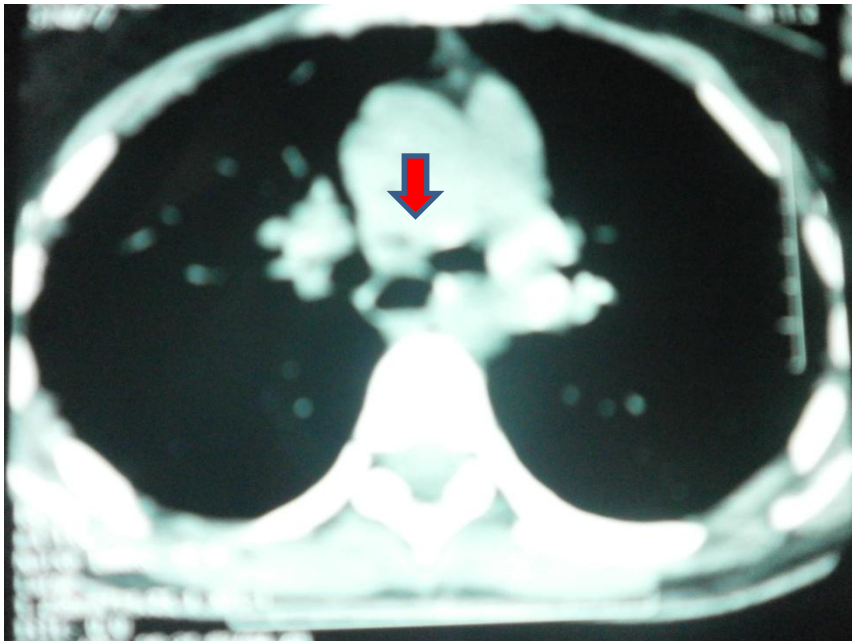




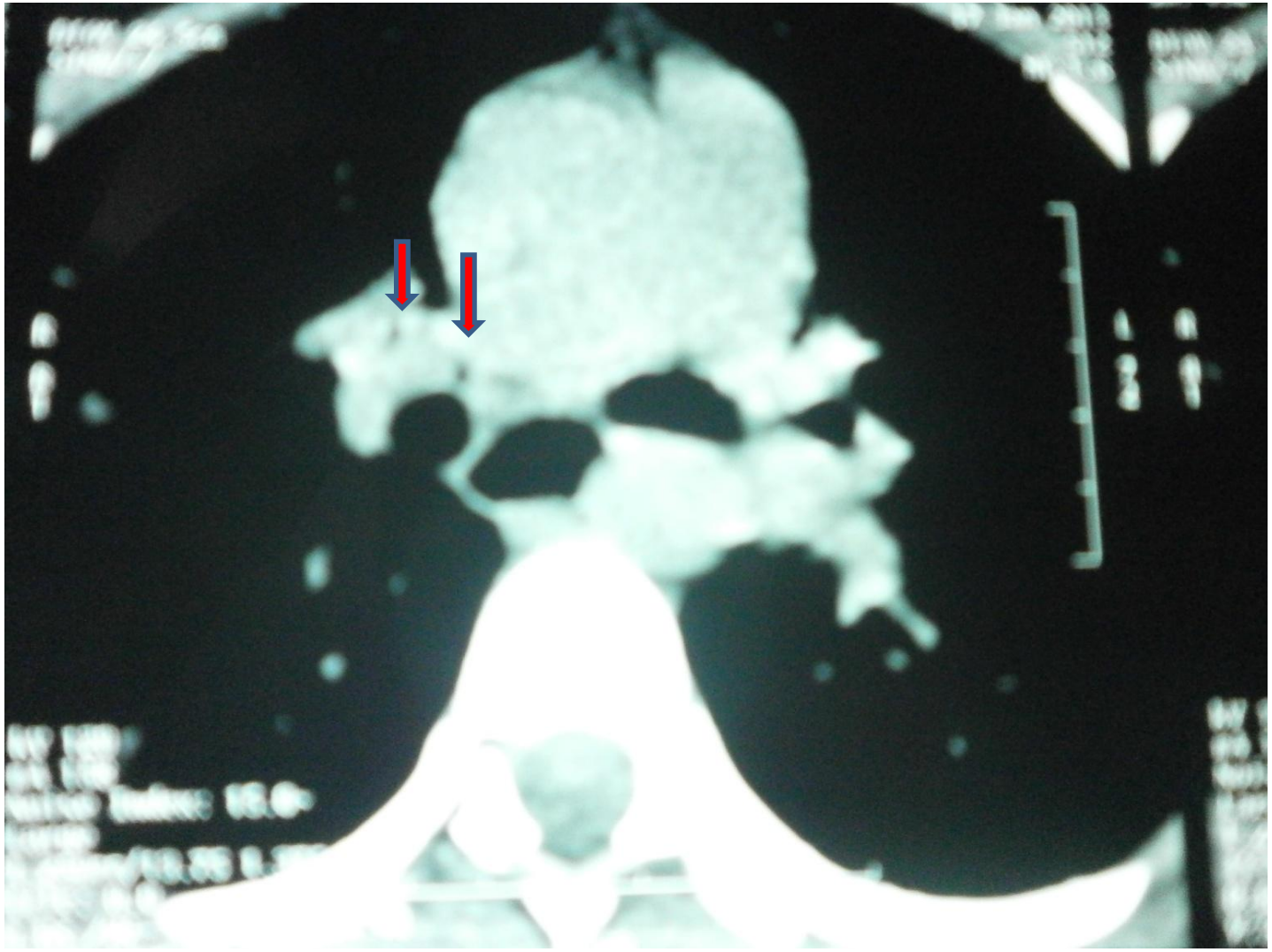


Adénopathies médiastinales ..Image aréique ?





Fistule ganglionotrachéobronchique



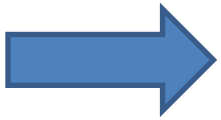
EXAMEN PHYSIQUE :
CYANOSEE .CASCHECIQUE..Sat:91 %. T° 37. Pd: 42 kg.Ta:165 cm-

EXAMENS BIOLOGIQUES :
NFS: GB = 11300 10 3/mm3- HB=15,60 gr/l - PLAQUETTES = 508 103/mm3
VS : 1ér H: 14 2ére H:40
HIV:négatif

CRACHAT :
Absence de BAAR

Conclusion

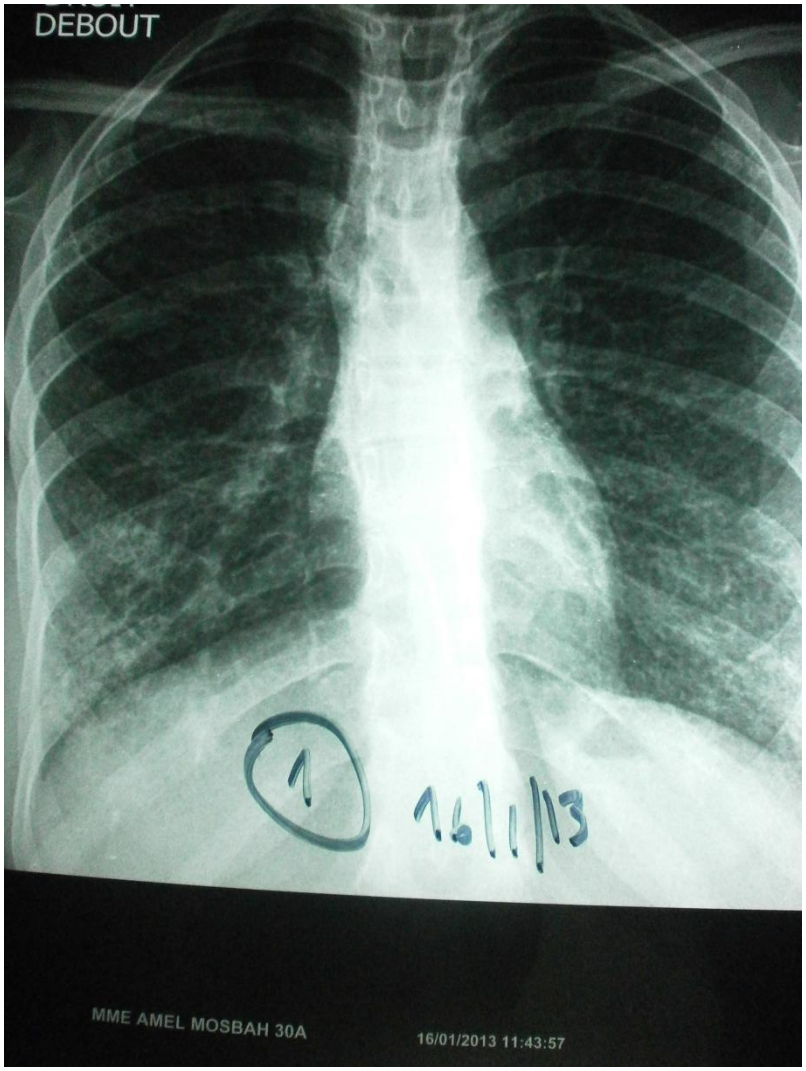
Il s'agit d'une jeunne fille de 32 ans qui présente un pneumopathie intertielle avec atteinte bronchiolaire et adénopathies médiastinale fistuliseé dans l'arbre tracheobronchique

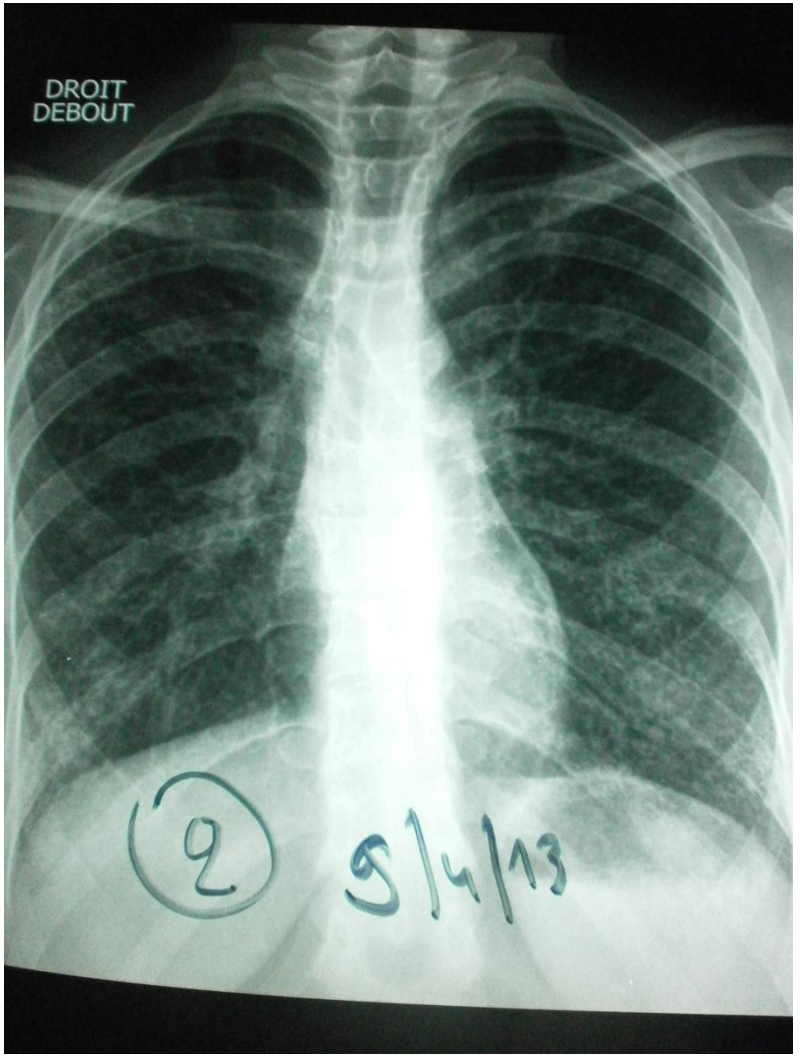


Traitement anti-TBC

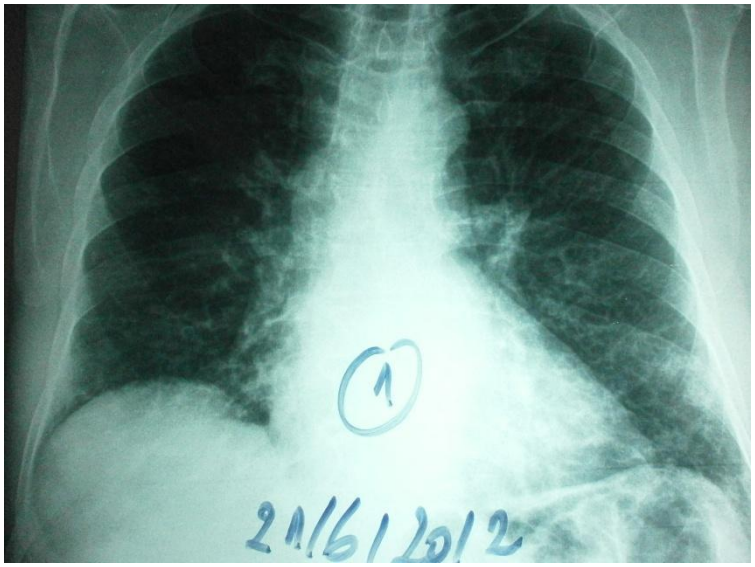
CONSULTATION N° 2 (09/04/2013) Recul : 2 mois 24 jours..Traitement anti-TBC arrête depuis 24 jours .

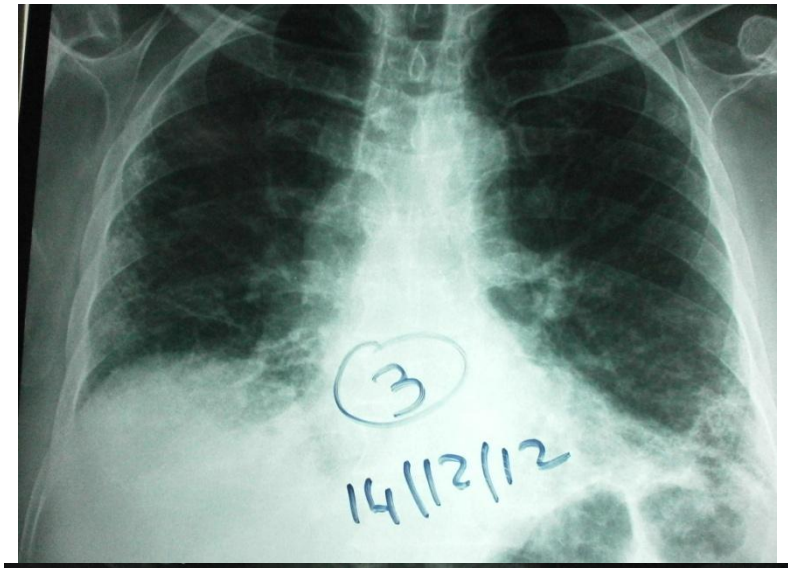
Amélioration clinique(Poids 45 KG + 3) et radiologique

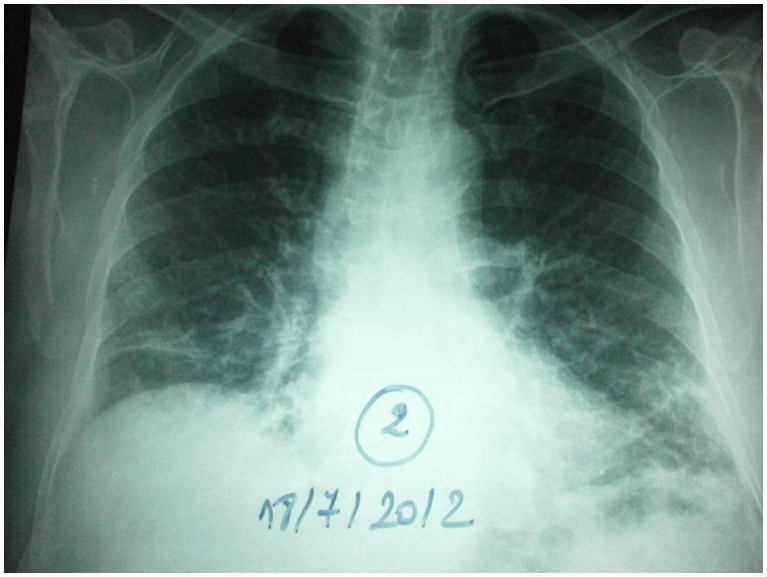


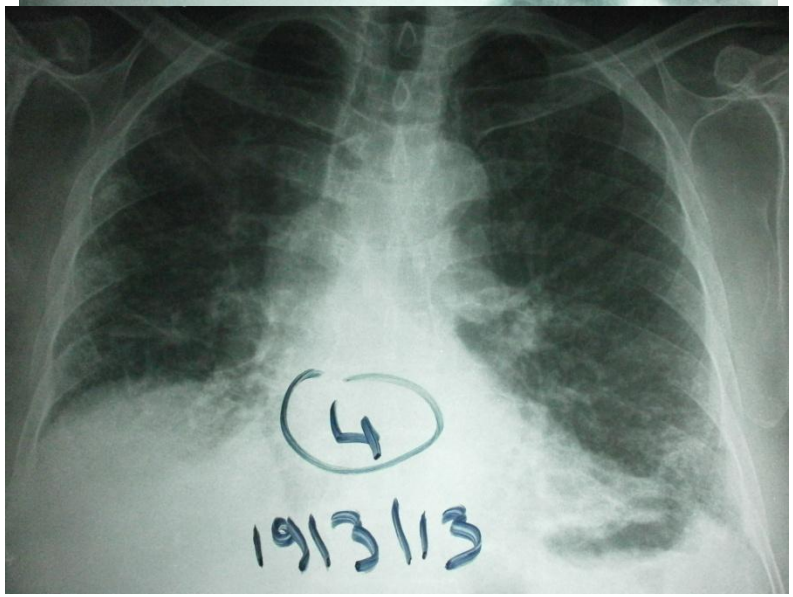


Mr Mohaméd H…75 ans Toux et dyspnée progressive

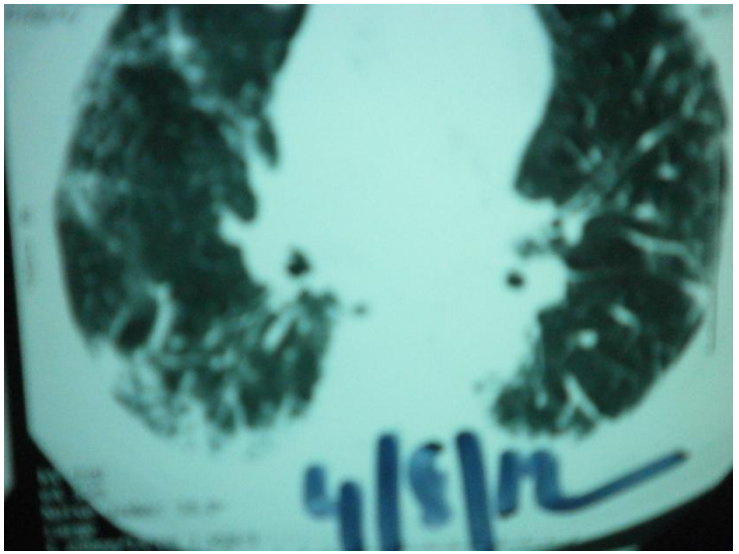


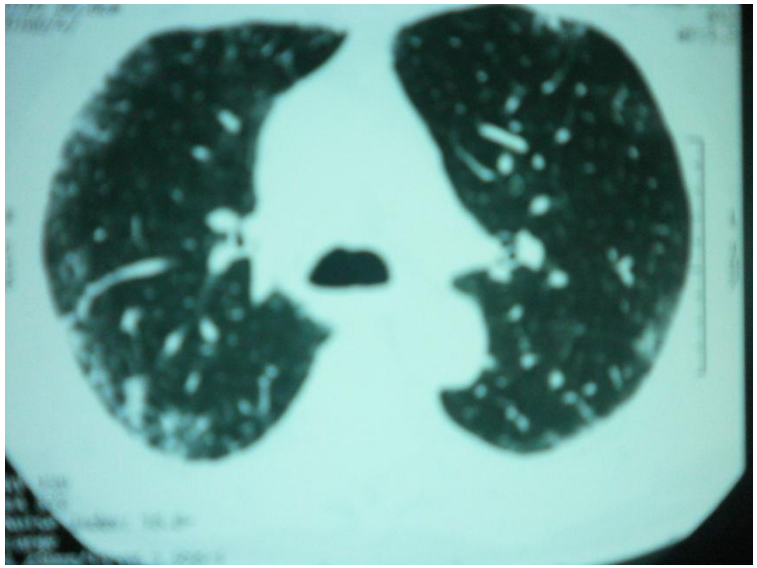


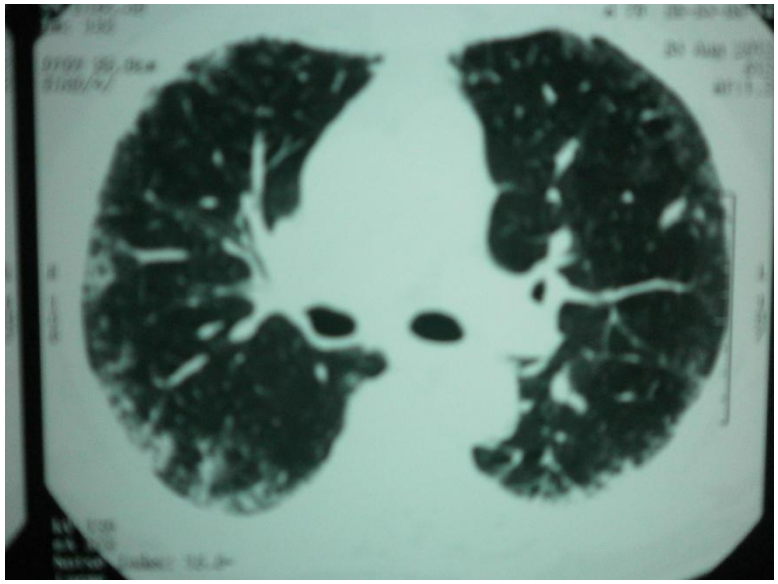


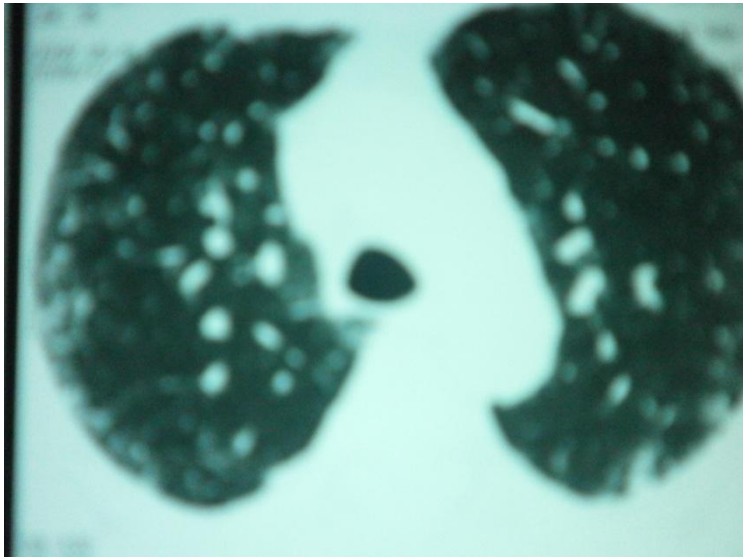


Ver dépolie. Micronodulaire centrolobulaire a contour floue non a bourgeon d’arbre. Réticulation sous pleurale









ANTECEDENTS ET TARES :
CARDIOPATHIE. ISHEMIQUE ASPEGIC 2501/J+SECTRAL 200-+CORVASAL
INDICATION PONTAGE REFUSEE

HABITUS :
Agent de steg.
Activité Agricole

HDLM:
Depuis 1999 une a deux bronchite par ans.

Toux fébrile. Sifflement sporadique .

Plaintes ORL.Rachilagie et arthralgie.
Douleur thoracique

EFR : Trouble ventilatoire restrictive

CVF : 1,85 43 %
VEMS: 1,85 55 %
TIFNAUX : 100%

IMMUNOLOGIE:

Sérologie poumon éleveur d'oiseau : Poule Canard présence de 2Arcs
sérologie micrpoluspora faenei présence d'Arc

- *Poumon de Fermier et d'eleveur d'oiseaux*

:

:

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

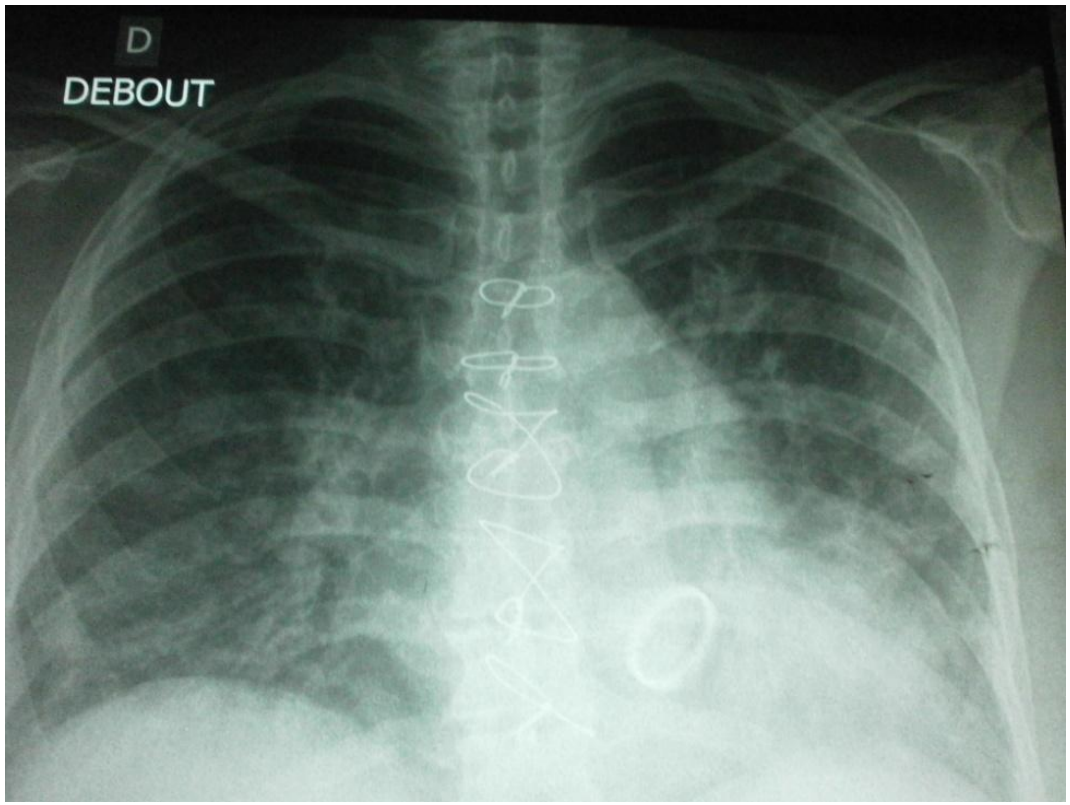
HIV:NEGATIF

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

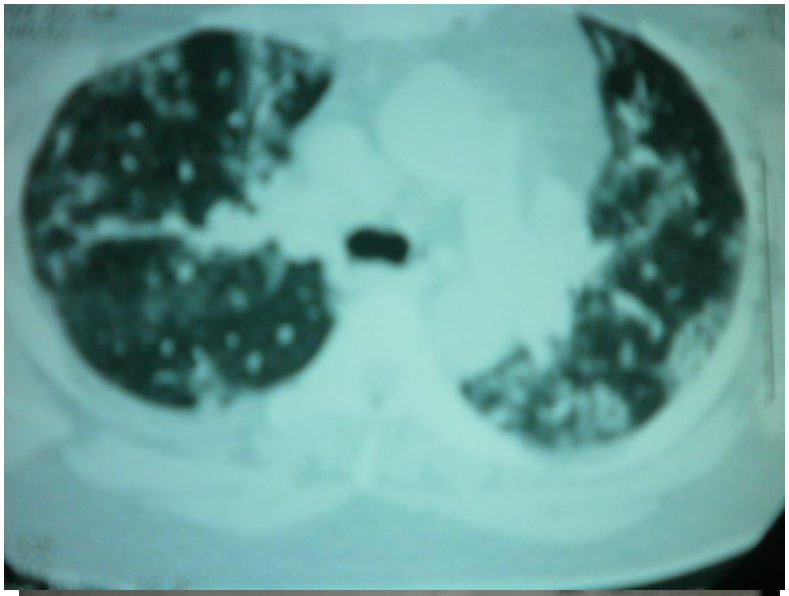


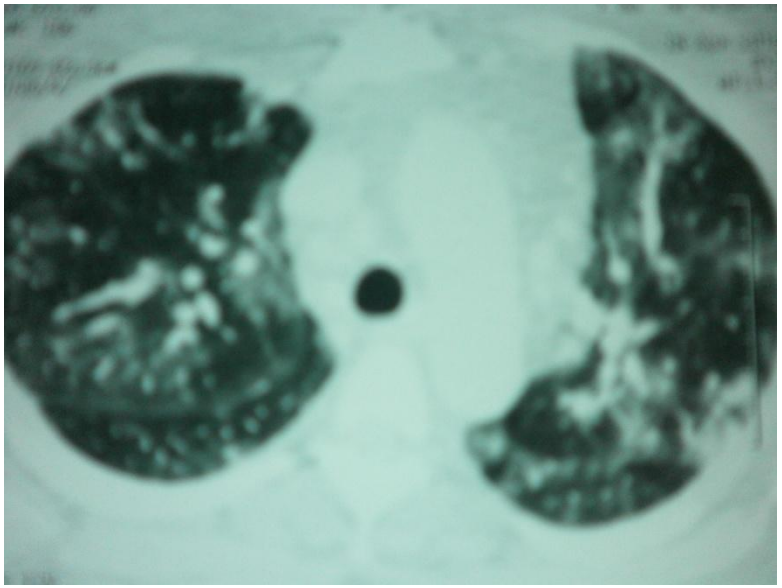
RX THORAX FACE:

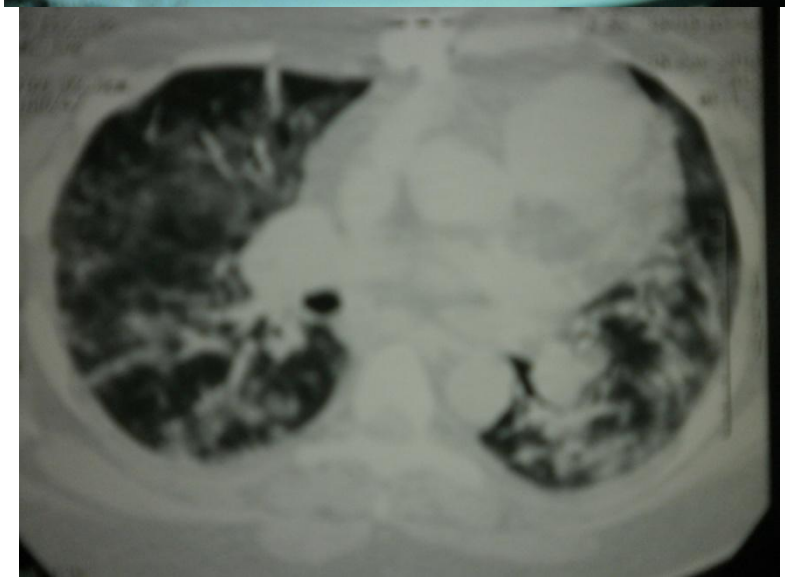
CARDIOMEGALIE MODEREE - SYNDROME INTERTIELLE -STERNOTOMIE -VALVE MITRALE

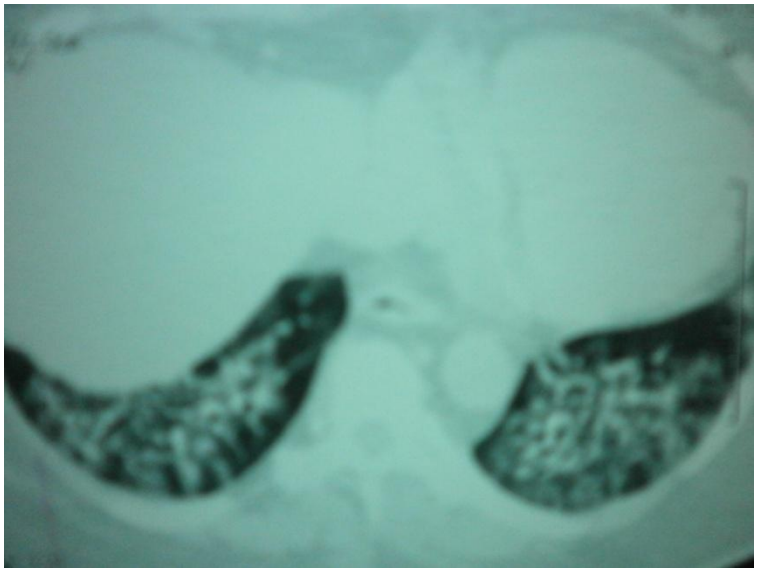
Syndrome interstitiel bilatérale :Nodulaire +ver dépolie +Réticulation

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

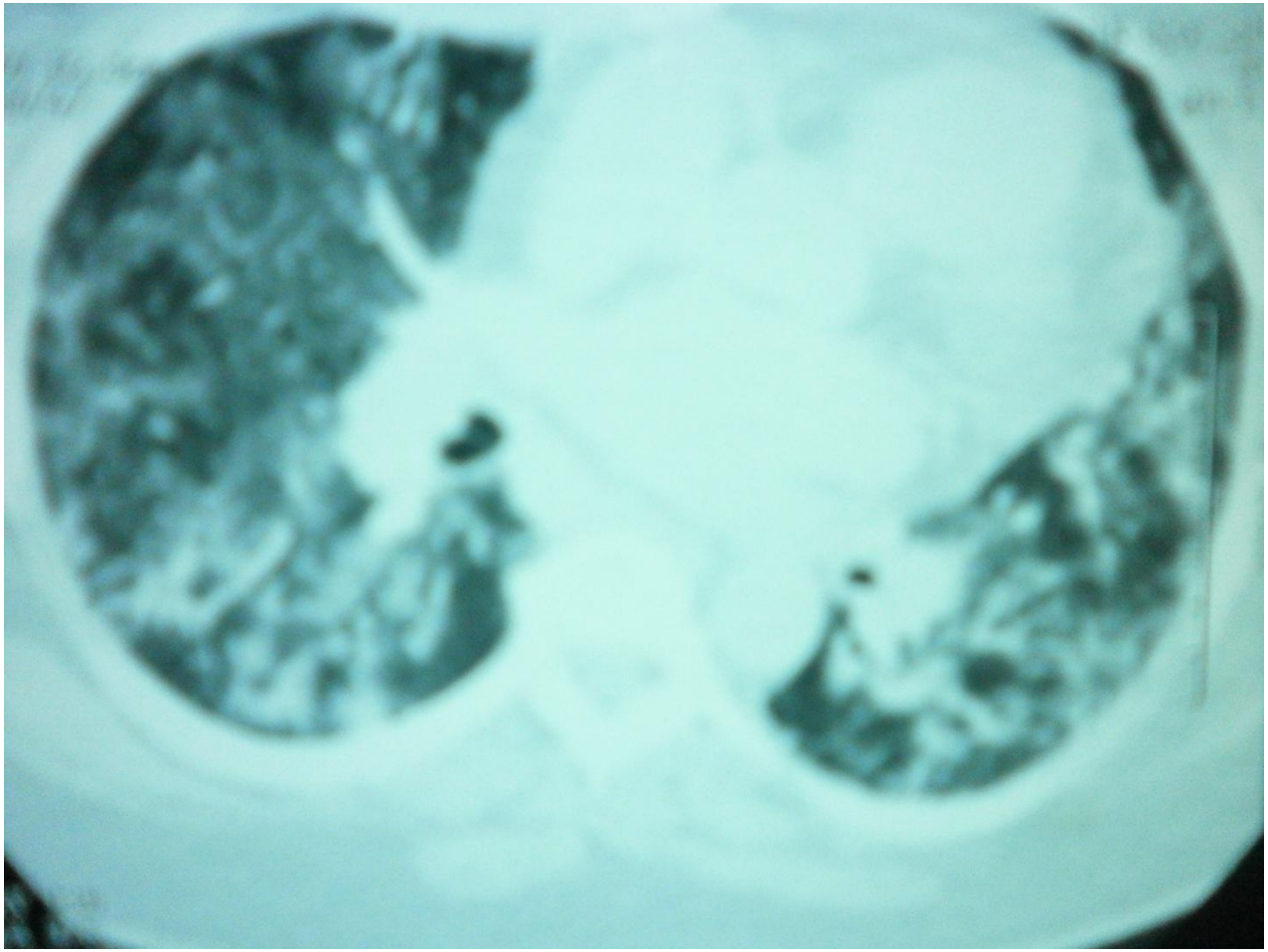








Il s' agit d' une femme de 52ans -valvuolapthie opérée en 2008
PNEUMOPTHIE INTERTIELLE AUX STADE D'HYPOXIE –
"PNEUMOPATHIE A LA CORDARONE"



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?

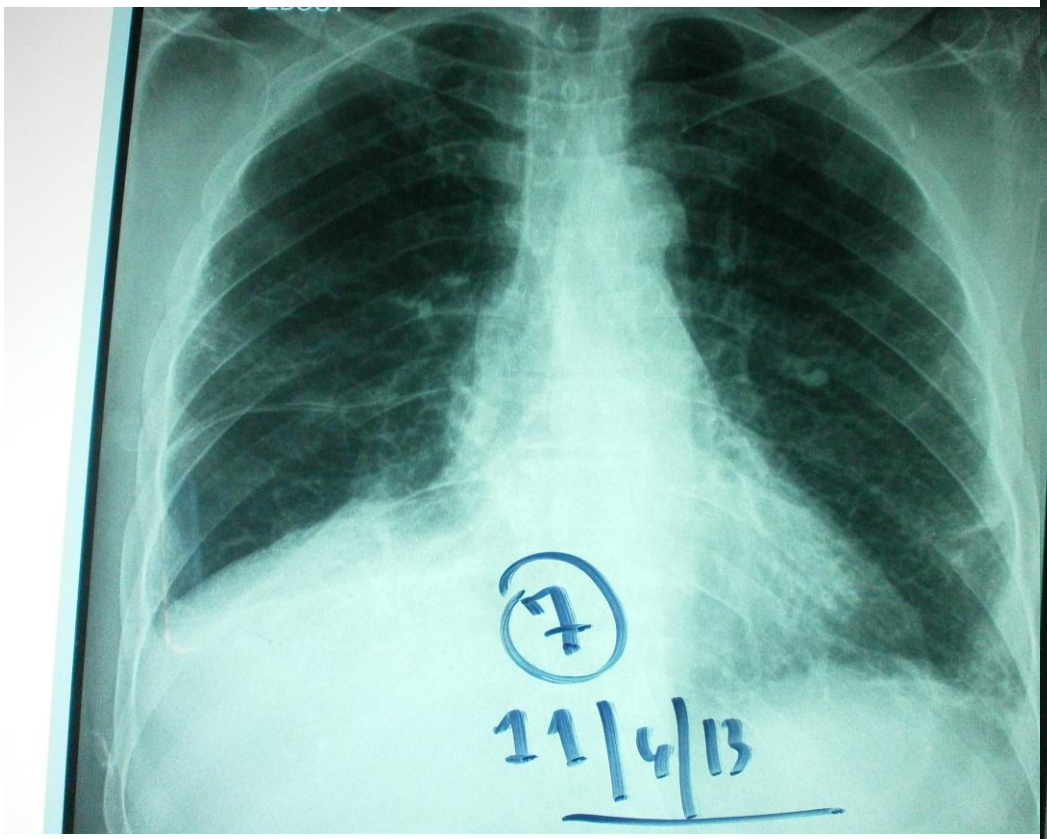
Schwaiblmair M, Berghaus T, Haeckel T, Wagner T, von Scheidt W.

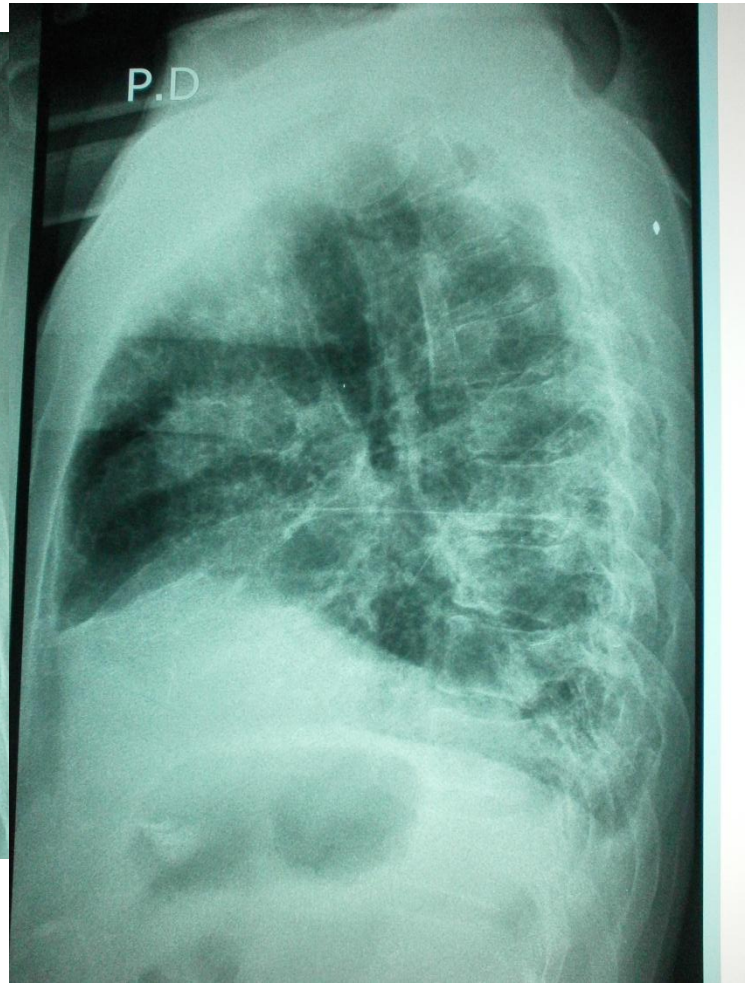
Source

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.
-

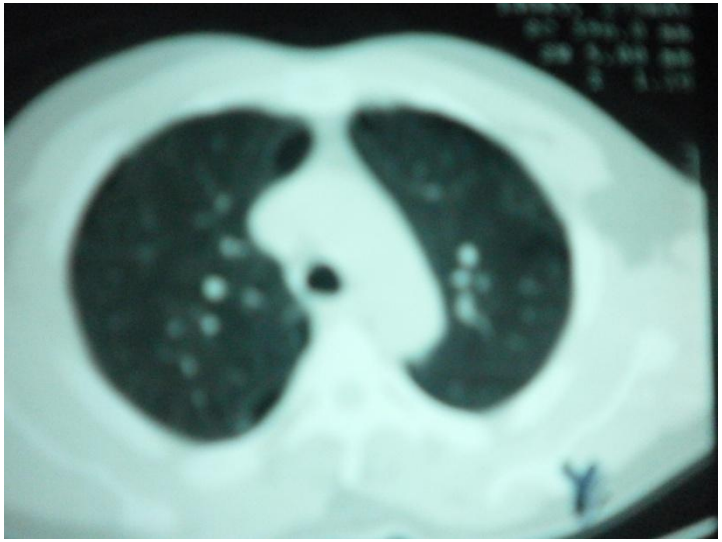
Mr MUSTAPHA A…56 Ans m' a consultée le 11/04/13 pour dyspnée progressive depuis 3 MOIS chez une maladie connu pour Polyarthrite rhumatoïde

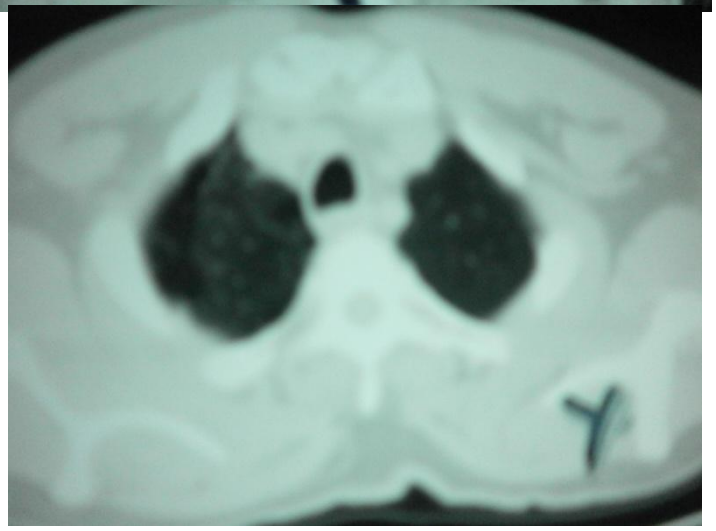


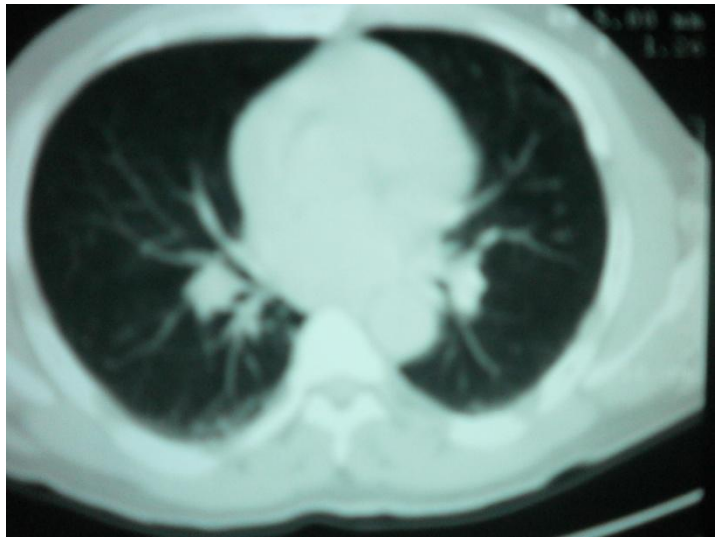


Scanner thoracique 29/12/09 QUASI -NORMALE

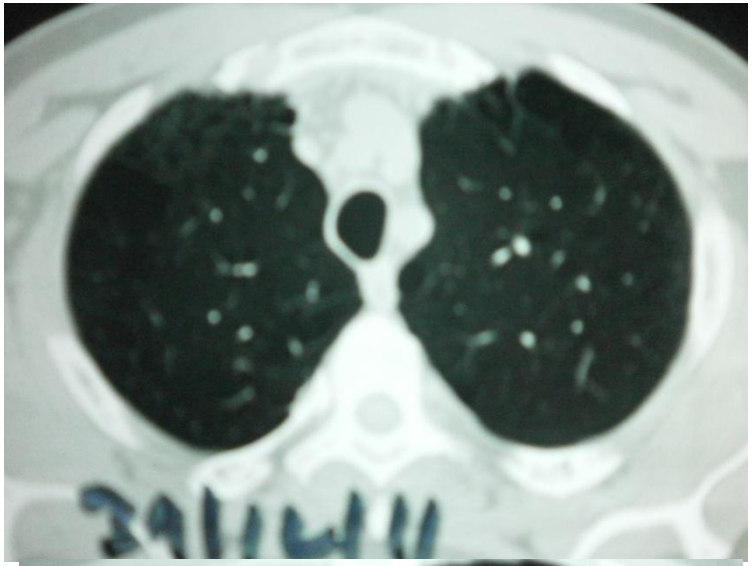
QQ BLEBS -S INTETIELLE A MINIMA PERIFERIQUE (Réticulation) aux nivaux des 2 bases

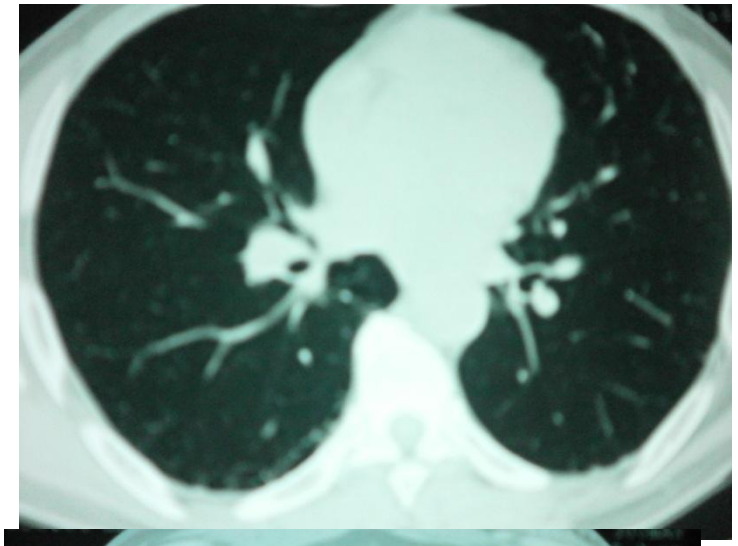


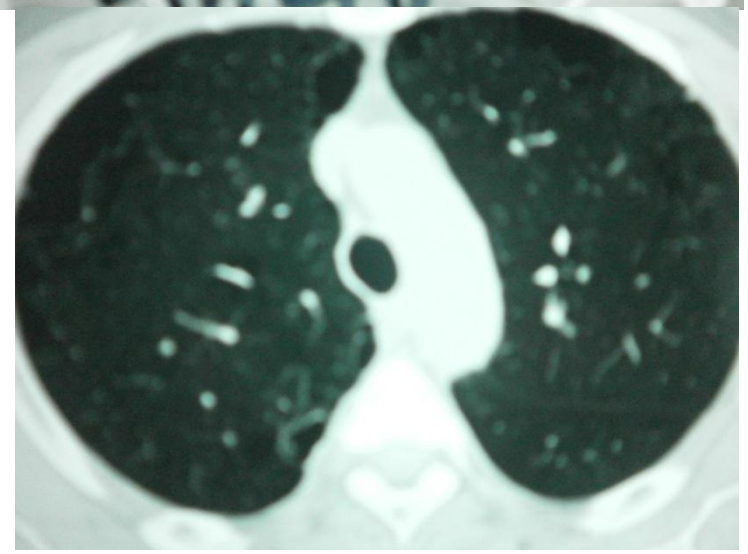


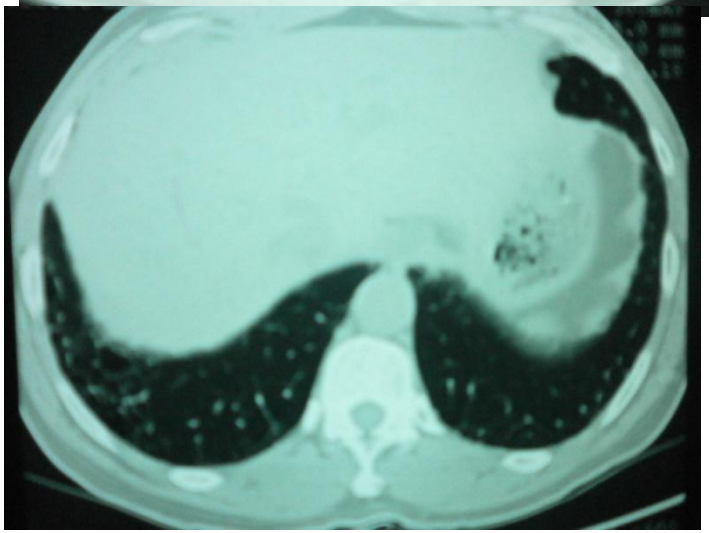


Scanner thoracique31/12/11 Normale (Emphyseme/Tabac)

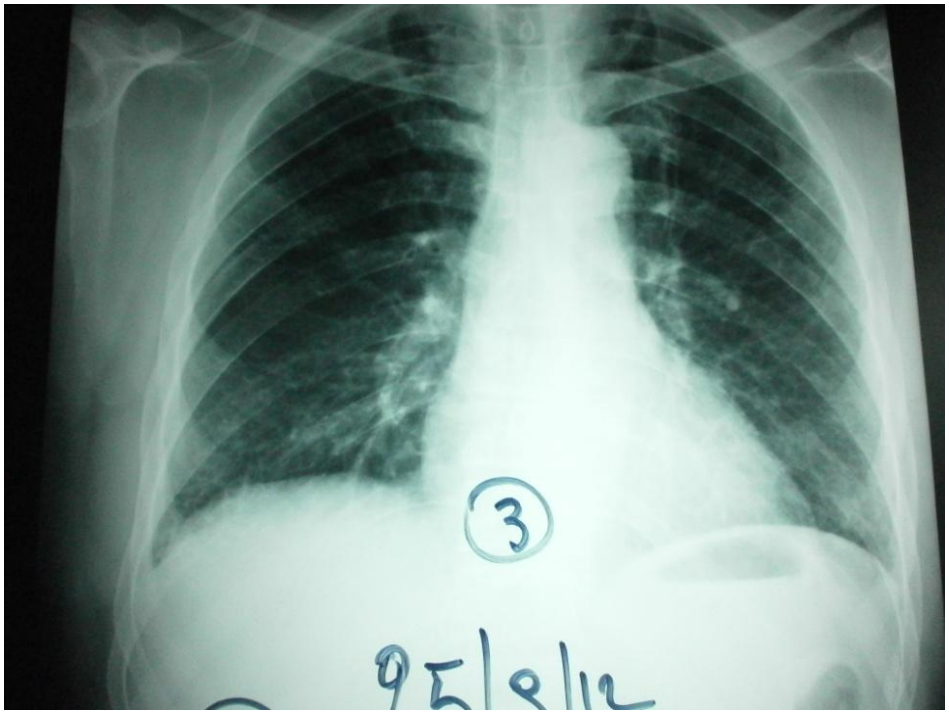








..Aété sous corticoide puis Vamid+plaquenil



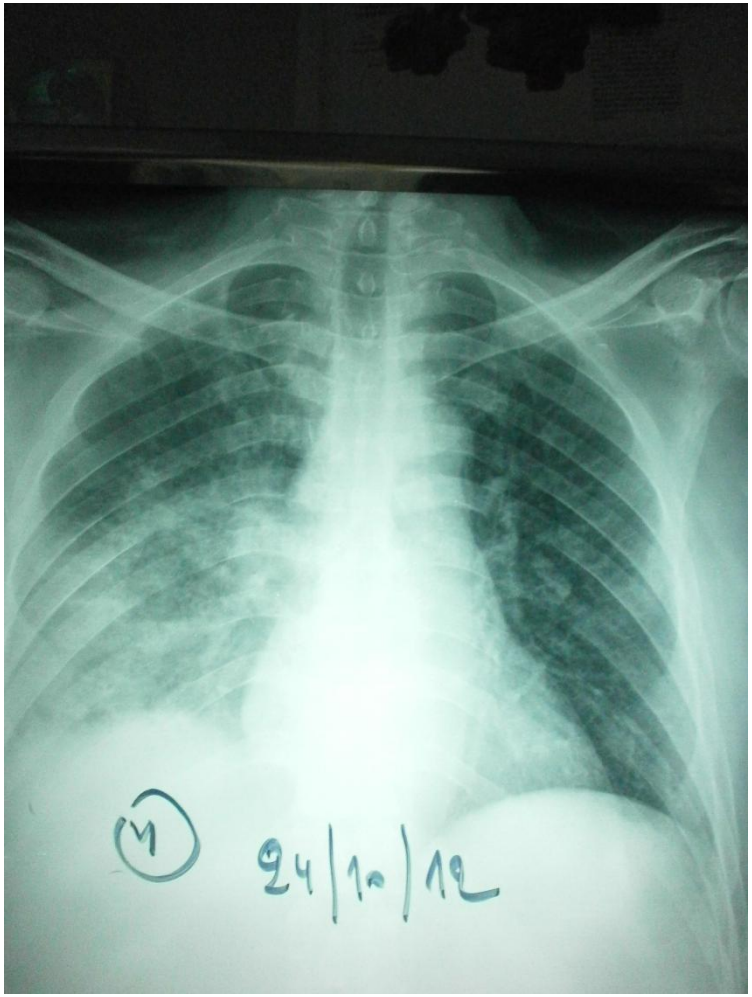
Association avec
Rémicade(INFLIXIMAB)
depuis le 24/09/1

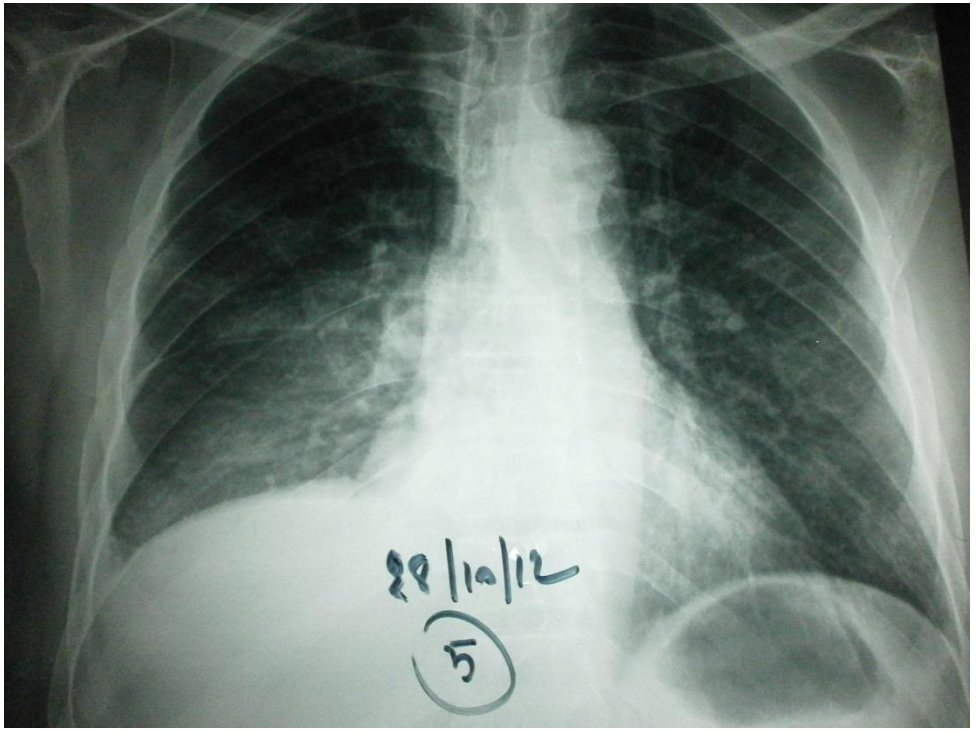
RX Thorax Normale.

Relais
le5/11/12..31/12/12.1
8/02/13..26/03/13)…

Le 24/10/12 PLEUROPNEUMPOTAHIE pneumopathie droite …PYOSTACINE TAVANIC

..

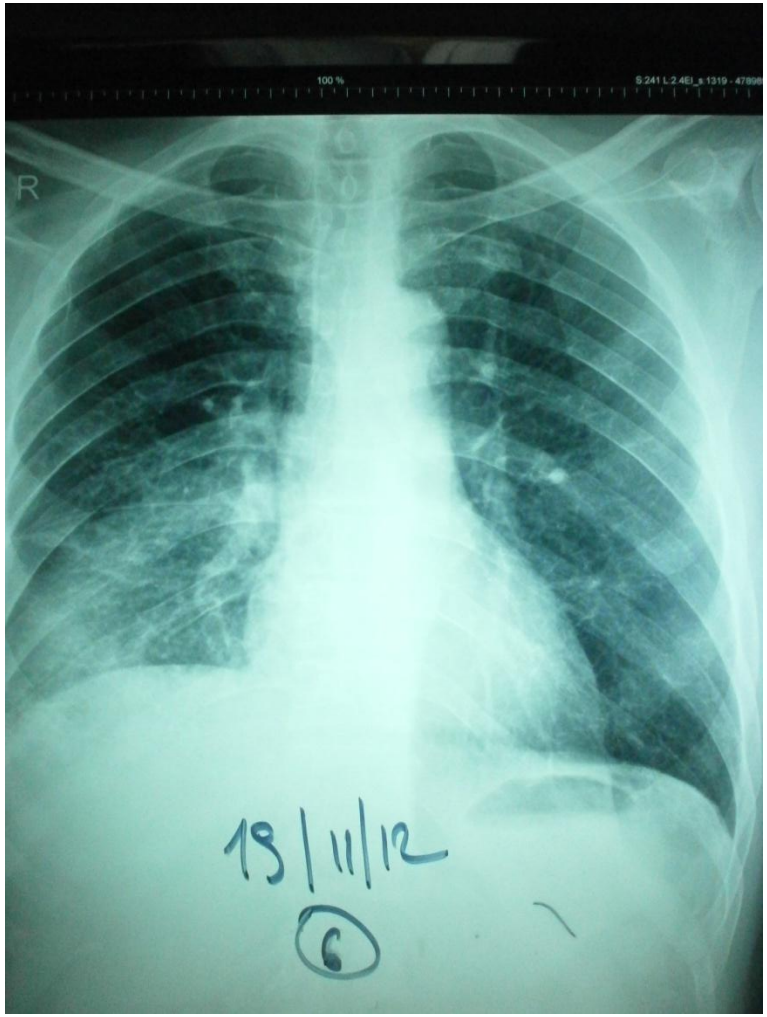


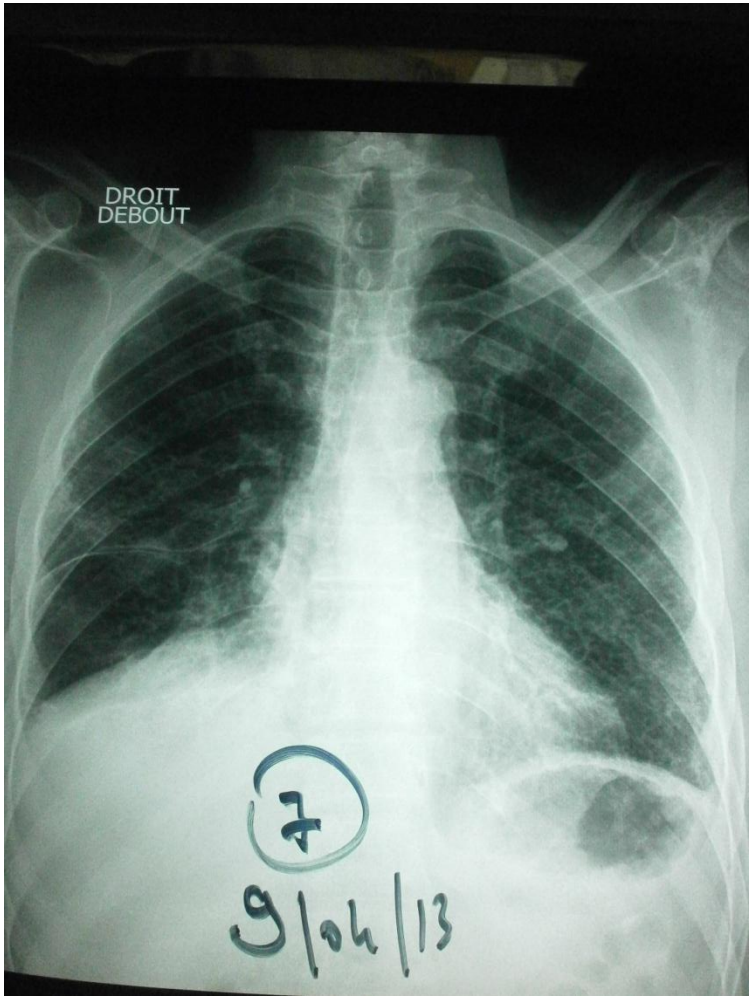


RX THORAX du 19/11/12 AUCUNE AMLIORATION…

Evolution(9/4/13) vers la rétraction a droite et extension controlatérale latérale

(s intertielle bilatérale-emoussements des 2 culs des sac)





ANTECEDENTS ET TARES :
Tabac 30 ANS
1980 LIPOME DU QUADRIPES
AVC 2008

EXAMEN PHYSIQUE :

TA: 15/8 RC : 80 /min .Sat: 98 %. T°37 .

MV PLUS RUDE A DROITE QQ CREPITANTES TRES FINES AUX 2 BASES

EXAMENS BIOLOGIQUES :

CRP:150,5.VS 75/120.

NFS: GB = 8500 10 3/mm3- HB=11,90 gr/l - PLAQUETTES =339 103/mm3

CREATININE:6,23.IONO: NA: 139 K: 3,8 CL:100

ProBNP:68

AcAnti-CCP:5,80.TEST AU LAREX:64,6.reaction de waaler-rose:52,7

CRACHAT :BK Négatif

IDR Négative

GAZ DE SANG :Po2: 70 Pco2 : 35 So2: 94 Hco3- : 24 PH:7,45

PONCTION PLEURALE :

ASPECT:trouble:RIVALTA:positif.PROTIDE:63.LDH:2538

Neutrophiles:84%.HEMATIE:6000.LYMPHO(%):16%

Absence de cellule néoplasique.BK Négative

TRIGLYCERIDE:0,40

CHOLESTROL TOTAL:1,22

Ac Anti-CCP:7,90.Test au latex:73,1.Reaction de Waaler rose:58,5

Homme de 55 ans maladie rhumatismale sévère -
Pleuropneumopathie Survenant un mois après début de l' INFLIXIMAB
1)PNEUMOPATHIE A L'INFLIXIMAB
2)POUMON RUMATHOIDE(échapant a INFLIXIMAB)

Am j Med Sc 2012 Jul;344(1):75-8. doi: 10.1097/MAJ.0b013e31824c07e8.

Infliximab-induced nonspecific interstitial pneumonia.

Sen S, Peltz C, Jordan K, Boes TJ.

Source

Internal Medicine at Riverside Methodist Hospital, Columbus, OH 43214, USA. soumitrasen.1983@gmail.com

Abstract

Infliximab has well-established complications including injection site and allergic reactions, cytopenias, induction of autoimmune and demyelinating diseases and malignancy, especially lymphoma**. Pulmonary complications are well documented and include serious respiratory infections from tuberculosis, fungal and opportunistic pathogens. This has prompted a Food and Drug Administration black-box warning recommending close surveillance for these diseases. Nonspecific interstitial pneumonitis (NSIP) secondary to tumor necrosis factor-alpha inhibitor (TNF-alpha) therapy is less well described. Rarely, TNF-alpha inhibitor therapy has been reported to cause NSIP when used in conjunction with other immunosuppressive agents**. Literature search revealed 12 independent patients with presumed infliximab-induced NSIP in 8 separate publications; all patients were on concomitant steroid sparing immunosuppressive agents, complicating cause and effect. The authors report a case in which infliximab is surmised to cause NSIP in the absence of other steroid sparing immunosuppressants in a young female with ulcerative colitis. Of importance, the patient was taking no additional steroid sparing immunomodulating agents. The diagnosis was based on clinical presentation and radiologic and histopathological data. Cessation of infliximab and high-dose steroid therapy resulted in complete resolution of the patient's presenting signs and symptoms.