



ICONOGRAPHIC REVIEW / *Thoracic imaging*

Imaging of lung transplant complications



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KEYWORDS

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Abstract Since the late 1980s, lung transplantation has emerged as a valid treatment option for some patients with advanced non-neoplastic lung disease. Long-term survival of lung transplant recipients, however, is lower than that of patients with other types of transplantation, because of numerous specific postoperative complications. Thanks to X-ray and CT, radiologists can guide clinicians, helped in this diagnostic approach by the time between the date of injury and date of transplantation. We will detail in this pictorial review the immediate and late surgical complications, the immunological complications, the infectious complications and other late complications.

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There were 244 lung transplants in France in 2010, mostly bilateral [1]. The survival of lung transplant recipients at 1 and 5 years is only 70% and 50% respectively [2,3] and is still less than that of patients who receive other types of transplant. This is explained by the many and often serious postoperative complications, which may have non-specific clinical and radiological features [2,4,5].

The aim of this pictorial review is to describe the radiological features of all of the intra-thoracic complications of lung transplantation. Chest CT, with an acquisition protocol adapted for the disease being investigated (Table 1) is the unequivocal instrument for the diagnosis of most of these disorders. We will therefore examine the “mechanical” surgical complications, distinguishing the immediate from the late, and then the immunological, infectious and other complications.

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Table 1 Chest CT protocols for lung transplant patients. The target doses are drawn from our experience and from reference [34].

Disease being investigated	Protocol	Contrast	Settings	Target dose (DLP)
Vascular anastomotic abnormality	Acquisition after contrast media injection at a mixed pulmonary arterial and aortic phase	High dose iodine medium (350 to 400 mg.mL ⁻¹)	100 kV	200–300 mGy.cm
Lung torsion		High flow injection (3 to 4 mL.s ⁻¹)	Autoregulation of mAs	
Pulmonary embolism	Unenhanced	Dose 70 to 100 mL	Iterative reconstruction	150–250 mGy.cm
Rejection		–	100 to 120 kV	
Infection		–	Autoregulation of mAs	
Bronchial anastomotic abnormality	Unenhanced, in inspiration then in expiration	–	Iterative reconstruction	250–350 mGy.cm for both volumes
Bronchiolitis obliterans			The expiration acquisition can be made at 120–135 kV with lower mAs (manual adjustment or increased noise)	

Immediate surgical complications

Haemorrhage

The majority of haemorrhages are pleural in origin [6], as intra-parenchymal or mediastinal haemorrhages are rarer (Fig. 1). The main risk factor is preoperative pleural adhesions, which are characterised by the disappearance of the extra-pleural fat line. These pleural adhesions are secondary to infections (tuberculosis, cystic fibrosis) or a previous procedure (pleurodesis or thoracotomy) [7].

Tube malposition

Selective intubation rapidly results in collapse of the contralateral lung. The barotrauma secondary to mechanical ventilation may cause a pneumomediastinum, a pneumothorax or an interstitial pulmonary emphysema. Malposition of the central catheter or Swan-Ganz catheter (Fig. 2) may cause a pneumothorax, hemothorax or intra- or extra-thoracic haematoma. Malposition of the pleural drain results in ineffective drainage [8].

Discordance between donor/recipient size

Implanting a large lung transplant into an overly small thoracic cavity may result in atelectasis and ventilatory problems.

Similarly, transplanting a small single-lung transplant into a thoracic cavity enlarged by emphysema may result in compression of the transplant due to hyperextension of the remaining emphysematous lung (Fig. 3), with displacement of the mediastinum towards the transplant [9].

Lung torsion is a rare early complication predisposed to by differences in calibre between the recipient thoracic cavity and an overly small transplanted lung [9]. Partial, often paucisymptomatic, torsion results in lobar atelectasis. Complete lung torsion produces serious clinical features with pain, dyspnoea or even shock, and is diagnosed on CT angiography by displacement of hilum and scissures, torsion of the pulmonary artery and adjacent bronchi with distal obliteration. Untreated, it rapidly progresses to parenchymal necrosis.

Acute primary transplant failure

Acute primary transplant failure, also called reperfusion oedema, is an acute respiratory distress syndrome characterised by a PaO₂/FIO₂ ratio of under 200 within the first 48 to 72 hours after transplantation. It is a diagnosis of exclusion, with an incidence of approximately 10% [10].

Radiologically, this is a non-cardiogenic oedema with a combination of posterior alveolar condensation and interstitial overload with thickening of the septal lines. Increased volume of the hila is highly suggestive.

CT, which is rarely performed, will show non-specific lesions: areas of ground glass opacities increase in vessel diameter and peribronchovascular oedematous infiltration.

Late surgical complications

Bronchial anastomotic complications

Healing of the respiratory tract anastomosis is the most delicate because of the removal of systemic blood vascularisation [11] and bronchial mucosal necrosis occurs in

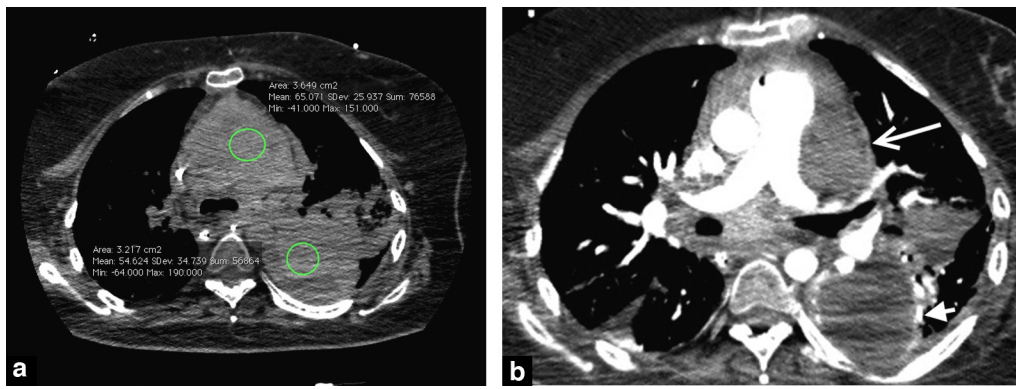


Figure 1. Fifty-eight-year-old female patient with a heart and lung transplant for pulmonary hypertension secondary to scleroderma, D2 postoperation. Chest CT without and with contrast with a mixed arterial phase acquisition. Anterior mediastinal haematoma (Fig. 1a – spontaneous density 60 UH) diffusing to the hilar region and compressing the left pulmonary artery (Fig. 1b – long arrow). Ipsilateral, posterior intra-parenchymal haematoma; vascular encorbment visible around the lesion after enhancement and the acute pleural attachment angle confirm intra-parenchymal location of the haematoma (Fig. 1b – short arrow).

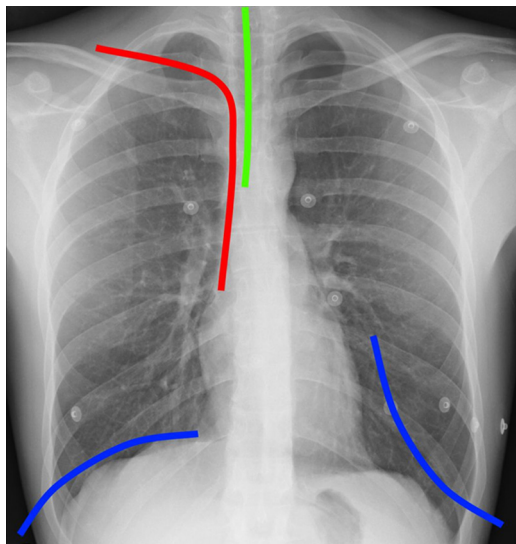


Figure 2. Normal position of the immediate postoperative tubes on a posteroanterior chest radiograph: intubation cannula (green), central venous line (red) and pleural drains (blue).

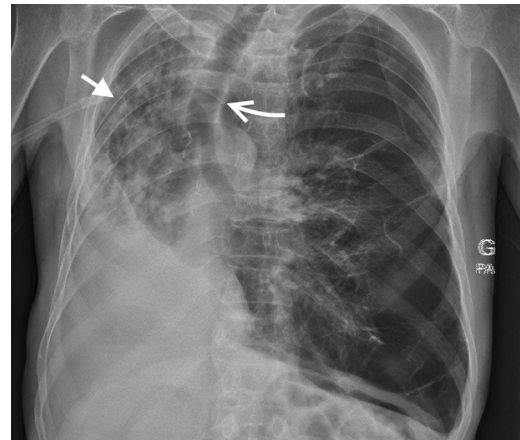


Figure 3. Fifty-four-year-old male patient, single right lung transplant 1 month previously for progressive centrilobular emphysematous dystrophy. Posteroanterior chest radiograph. Major distension of the patient's own left lung, pushing back the contralateral side of the mediastinum (long arrow), loss of transplant volume; disturbance of venous return and lymphatic drainage secondary to the compression (thickening of the interlobular septa – short arrow, poorly delineated alveolar ground glass opacities pleural effusion).

12% of cases; this either recovers without complications or progresses to fixed (stenosis – Fig. 4) or functional (bronchomalacia diagnosed with a CT in expiration – Fig. 5) anastomotic stenosis, which may require endoscopic dilatation ± stenting [7,12]. Granulomas may develop on the sutures or in an area of scarring and, depending on their size, cause bronchial stenosis (Fig. 6). Stenosis investigations are facilitated by coronal and oblique fine miniIP reconstructions, curvilinear reconstructions along the axis of the bronchus which may enable a semi-automatic measurement of the stenosis and three-dimensional volume reconstructions.

Dehiscence (a defect of the bronchial wall) is seen in 1 to 6% of cases and occurs after the first month (Fig. 7) [7,12]. This may be complicated by respiratory failure, bronchopleural fistulas (Fig. 8) or pneumothorax [13]. “Virtual bronchoscopy” reconstructions can visualise these lesions more easily. Depending on the size of the dehiscence [14]



Figure 4. Thirty-six-year-old male patient 15 days following bilateral lung transplant. Axial CT without enhancement showing a tight stenosis of the distal part of the left main bronchus at the level of the anastomosis (arrow).

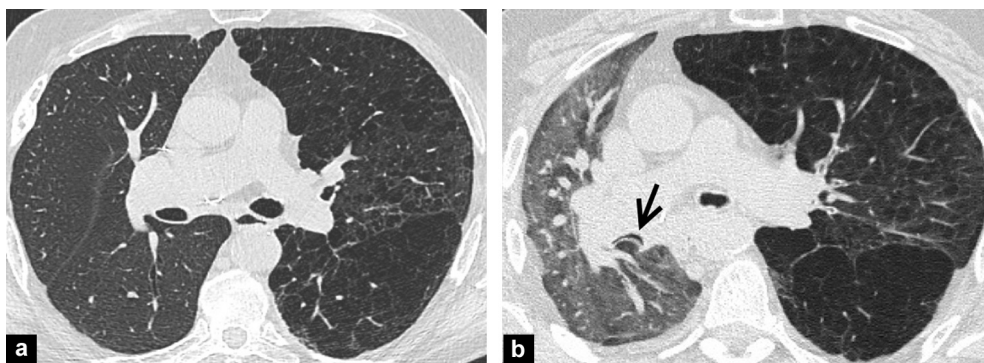


Figure 5. Sixty-seven-year-old male patient 1 year after a single right lung transplant. Chest CT without enhancement in inspiration (Fig. 5a) and then expiration (Fig. 5b): bronchomalacia shown on the expiration acquisition, with severe collapse of the right main bronchus (arrow).

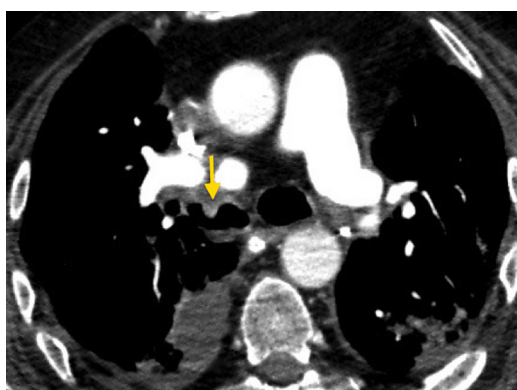


Figure 6. Sixty-year-old female patient. Enhanced chest CT 1 month after bilateral lung transplants: a granuloma has developed on the sutures at the right bronchial anastomosis (arrow) causing a 50% stenosis.

and its consequences, either no treatment, conservative treatment with pleural drainage or revision surgery are required.

Vascular anastomotic complications

Vascular sites of anastomosis (pulmonary arteries and veins) are seen as a 1 to 2 millimetres fold on the walls of the blood vessels, without a significant reduction in vascular diameter (Fig. 9).

Pulmonary artery stenosis may occur in the early or late post-transplantation period and is often associated with pre-stenotic arterial dilatation (Fig. 10). Treatment is by dilatation, stenting or surgical reconstruction.

Stenosis of a pulmonary vein is more common and becomes symptomatic more rapidly (Fig. 11). It induces a parenchymal oedema in the area drained by the vein(s) in question [7,8].

Other surgical complications

Neuronal

Diaphragmatic paralysis is a common complication of bilateral lung transplantation, usually secondary to a

stretching of the phrenic nerve. It usually recovers without treatment over a variable period ranging from 3 to 8 months.

Gastric atony secondary to bilateral vagus nerve damage may also occur [15].

Infectious

Wall abscesses, sternal or parietal dehiscence and mediastinitis are particularly worrying postoperative complications in these immunosuppressed patients (Fig. 12) [7,15].

A positive diagnosis can be made by chest CT, with a wall collection, diastasis of the sternotomy margins indicating sternal dehiscence and possibly accompanied by osteolysis of the margins if sternitis is present, mediastinal infiltration and collections and a pericardial effusion.

Immunological complications

Hyper-acute rejection

Hyper-acute rejection recurs from de-clamping onwards and results in massive sudden congestion of the entire transplanted organ which permanently ceases to function. This complication is invariably fatal [16].

Radiography, which is rarely performed, is reported to show homogeneous diffuse infiltration throughout the transplanted lung [8].

Acute rejection

Acute rejection occurs from day five postoperatively, as the patient is protected until this point by infusions of antilymphocyte serum. Eighty percent of rejections occur during the first 3 months, particularly around the 8th/9th day post-transplantation. The incidence of acute rejection during the first postoperative year is between 40 and 50% [17–19], following which the prevalence falls. The clinical features of rejection are variable and non-specific: dyspnoea, cough, fever, reduced FEV1 and even acute hypoxemia. The formal diagnosis requires histology from a trans-bronchial biopsy [7].

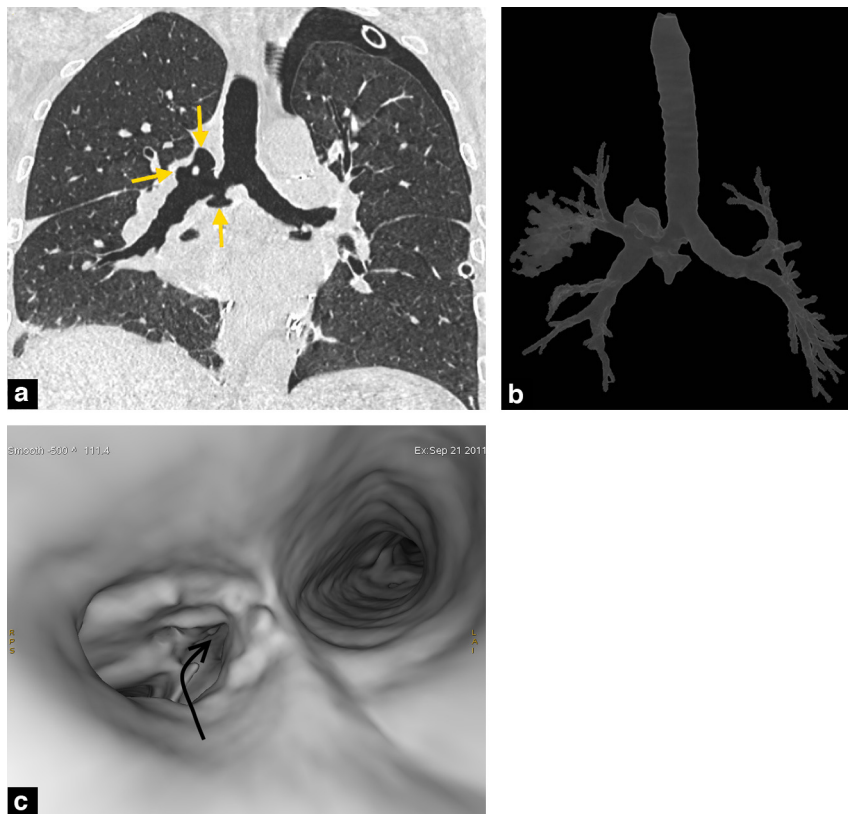


Figure 7. Seventeen-year-old male patient 10 days after bilateral lung transplants who developed respiratory distress. Chest CT without enhancement, coronal (Fig. 7a) and volume (Fig. 7b) reconstructions and virtual bronchoscopy (Fig. 7c): loosening of the bronchial suture with 3 dehiscences (arrows in 7a) at the level of the right main bronchus anastomosis (clearly visible on virtual bronchoscopy – arrow in 7c). The patient died a few hours after the investigation.

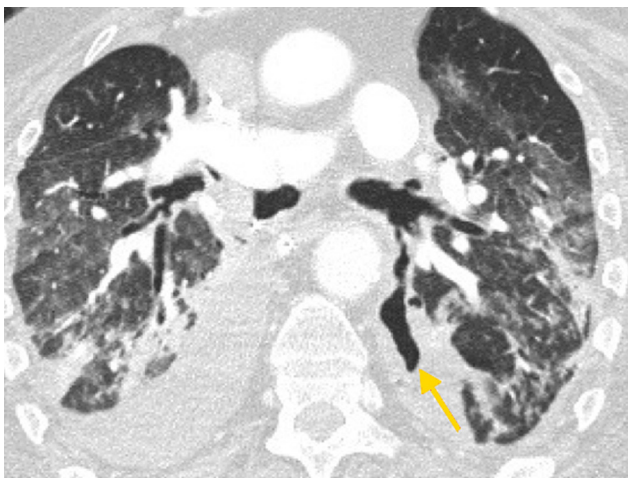


Figure 8. Sixty-one-year-old male patient. Enhanced chest CT 1 month after bilateral lung transplants complicated by acute rejection: left broncho-pleural fistula (arrow).

The chest radiograph may be normal. The diagnosis can be suggested in some cases by a combination of septal lines and pleural effusions without a concomitant rise in the cardiothoracic index or vascular redistribution [9,20].

CT shows interstitial oedema with diffuse ground glass opacities (often with a basal distribution) followed by



Figure 9. Fifty-seven-year-old female patient. Enhanced chest CT 1 month after bilateral lung transplants: bilateral folds at the level of the pulmonary artery anastomoses (arrows), predominantly on the left, with no significant stenosis (< 50%).

peri-hilar alveolar consolidation, septal thickening, reduced lung transplant volume and occasionally basal pleural effusions. Involvement is bilateral and symmetrical (Fig. 13). The diagnosis can be excluded from the absence of ground glass opacities [8].

Rapid improvement after increasing immunosuppression is a true diagnostic test.



Figure 10. Fifty-three-year-old male patient. Enhanced chest CT 1 month after bilateral lung transplants: 50% left pulmonary artery stenosis (arrow) with pre-stenotic arterial dilatation.

Chronic rejection

Chronic rejection can occur from the 4th month post-transplant, although it is rare before 6 months. This affects

up to 60% of recipients after 5 years [21] to become the leading long-term cause of death [22]. It is promoted by previous acute rejection [18].

The clinical features are of bronchiolitis obliterans (Bronchiolitis Obliterans Syndrome – BOS). Clinically, the patient develops increasing dyspnoea, a productive cough and a progressive fall in FEV1.

Different radiological signs may be found: lung hyperinflation, reduction in peripheral vascularisation, subsegmental atelectasis and bronchiectasis. Radiography, however, is often normal.

The main computer tomography findings are bronchiolar obstruction, with distension and relative reduced density in the parenchymal areas located distal to the obstruction. The distension is associated with late vascular redistribution towards normally ventilated areas, and the parenchyma therefore takes on a heterogeneous “mosaic” appearance. These characteristic appearances [23] are unmasked in expiration because of their trapping in the pathological areas (Fig. 14). The bronchi are dilated and malacic, collapsing on expiration, occasionally with bronchiectasis and thickening of the bronchial walls. Septal thickening and subpleural reticulations are signs of fibrosis and occur later [4].

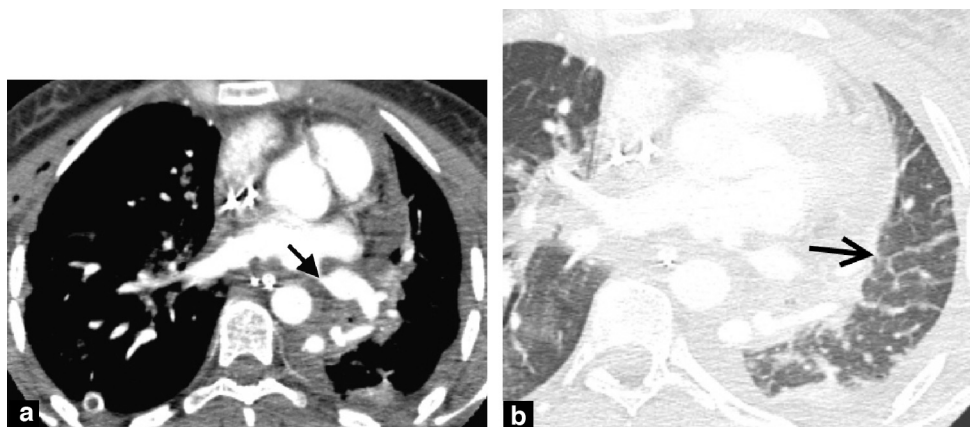


Figure 11. Forty-nine-year-old female patient. Enhanced chest CT 15 days after bilateral lung transplants: left inferior pulmonary vein stenosis (Fig. 11a – short arrow) with pre-stenotic venous dilatation and interstitial oedema in the distal pulmonary area, appearing as regular thickening of the interlobular septa (Fig. 11b – long arrow).

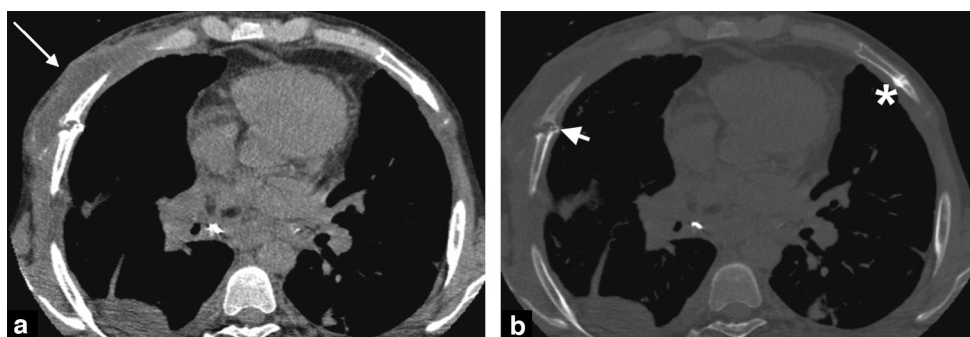


Figure 12. Sixty-year-old male patient 6 months after bilateral lung transplants. Unenhanced chest CT in the mediastinal (Fig. 12a) and bone (Fig. 12b) windows: chronic collection in the right anterolateral chest wall (fine parietal calcifications – long arrow) causing a pseudarthrosis of the 5th right rib (short arrow) 6 months after rib division during a bilateral anterolateral thoracotomy. Compare with the good healing of the controlateral rib (asterisk).

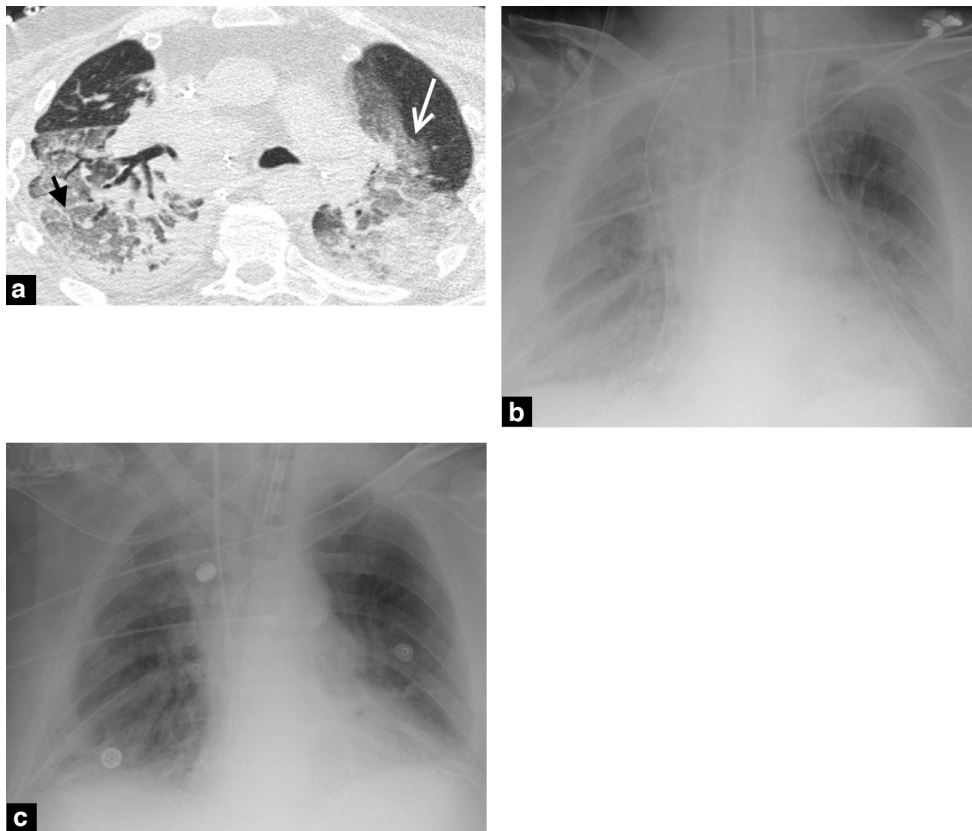


Figure 13. Sixty-one-year-old male patient 1 month after bilateral lung transplants. Unenhanced chest CT (Fig. 13a): peri-bronchovascular ground glass infiltrate (long arrow) thickening of the interlobular septa (short arrow), bilateral pleural effusions due to suffusion. Corresponding posteroanterior chest radiography (Fig. 13b): diffuse bilateral interstitial ground glass appearances with predominantly peri-hilar alveolar opacifications and bilateral basal effusions. Repeat radiograph 3 days after increasing immunosuppressants (Fig. 13c): rapid improvement confirming the diagnosis of acute rejection.

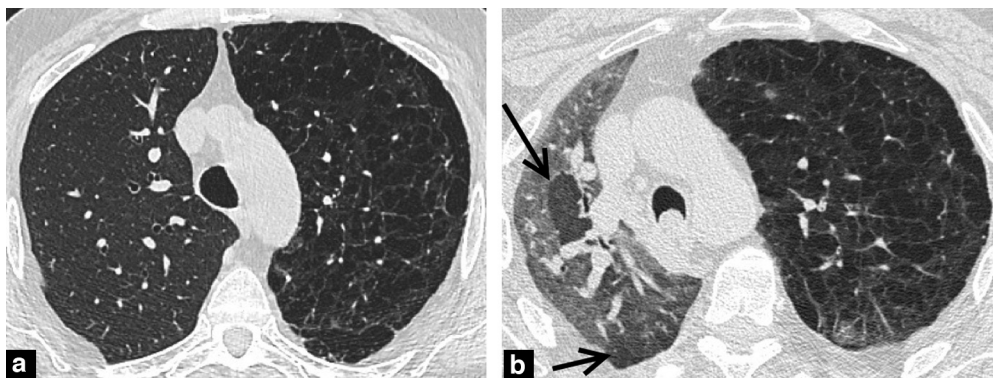


Figure 14. Sixty-nine-year-old male patient with single right lung transplant for progressive emphysematous dystrophy. Follow up chest CT 18 months post-transplantation in inspiration (Fig. 14a) then expiration (Fig. 14b). The transplanted lung parenchyma is homogeneous on inspiration. In expiration (seen by the swelling of the posterior aspect of the trachea), hypodense areas in the consolidated lung parenchyma (arrows) representing distal air trapping, are indicative of BOS.

Infectious complications

Infectious complications are the leading cause of early morbidity and mortality and the second cause of late deaths.

Bacterial infections

Pneumonia, due to Gram-negative bacteria such as *Pseudomonas aeruginosa* and *Klebsiella*, are the commonest

postoperative infectious causes. During the first postoperative month, 35 to 70% of recipients will develop a bacterial pneumonia [24].

Typical or atypical Mycobacteria infections are less common, although tuberculosis is increasing.

CT shows non-specific abnormalities such as parenchymal consolidation, branched centrilobular micronodules, thickening of the interlobular septa and pleural effusions.

Fungal infections

The most worrying opportunistic infection is with *Aspergillus fumigatus*, which occurs usually between the 10th and 60th day post-transplant. There are at least four different types: endobronchial or pulmonary *Aspergillus* colonisation without associated disease, pseudomembranous or necrotising *Aspergillus* tracheobronchitis, invasive pulmonary aspergillosis and disseminated invasive aspergillosis [25,26].

Pseudomembranous or necrotising tracheobronchial aspergillosis is a form of *Aspergillus* infection which is specific to lung transplants (Fig. 15), and are due to infection of the mucosa with tracheobronchial ulcerations and pseudomembranes confirmed on bronchoscopy. As the bronchial anastomosis is particularly vulnerable, the infection frequently causes bronchial stenosis or fistula, which is a highly suggestive CT finding. It could secondarily evolve towards invasive pulmonary aspergillosis [9].

On CT, invasive aspergillosis results in a more or less excavated parenchymal opacification surrounded by a ground glass halo; which represents the alveolar hemorrhage at the periphery of the infarction.

Other fungal infections (*Candida*) are far rarer.

Viral infections

CMV is the most common of the opportunistic infections in lung transplant patients, and affects almost 50% of

recipients [8]. It presents between the 1st and 6th month after transplantation, particularly around D21 [9].

The most common CT findings are ground glass opacities with disseminated micronodules with the tree in bud pattern, bronchiectasis and diffuse consolidation in the transplanted lung (Fig. 16) [8].

The other community acquired respiratory virus infections include adenovirus, respiratory syncytial viruses [27], influenza and para-influenza viruses and are thought to predispose to bronchiolitis obliterans [26].

Parasitic infections

Opportunistic pneumocystis infection due to *Pneumocystis jiroveci* occurs in 90% of recipients unless prophylactic measures are taken. It may occur if serious side effects require prophylaxis to be stopped or if therapeutic adherence is poor. The infection is polymorphic and may range from being paucisymptomatic to pneumonia causing hypoxemia [26].

Chest radiograph is normal in a third of cases, or shows progressive bilateral peri-hilar parenchymal opacifications; the bases are affected secondarily [9].

CT shows diffuse ground glass opacifications (often sparing the extreme periphery of the lung cortex) combined, to a greater or lesser extent, with focal areas of consolidation, thickening of the interlobular septa (crazy paving appearance) and/or with micronodules. In 10 to 30% of cases, cystic lesions are seen predominantly in the upper lobes. These are

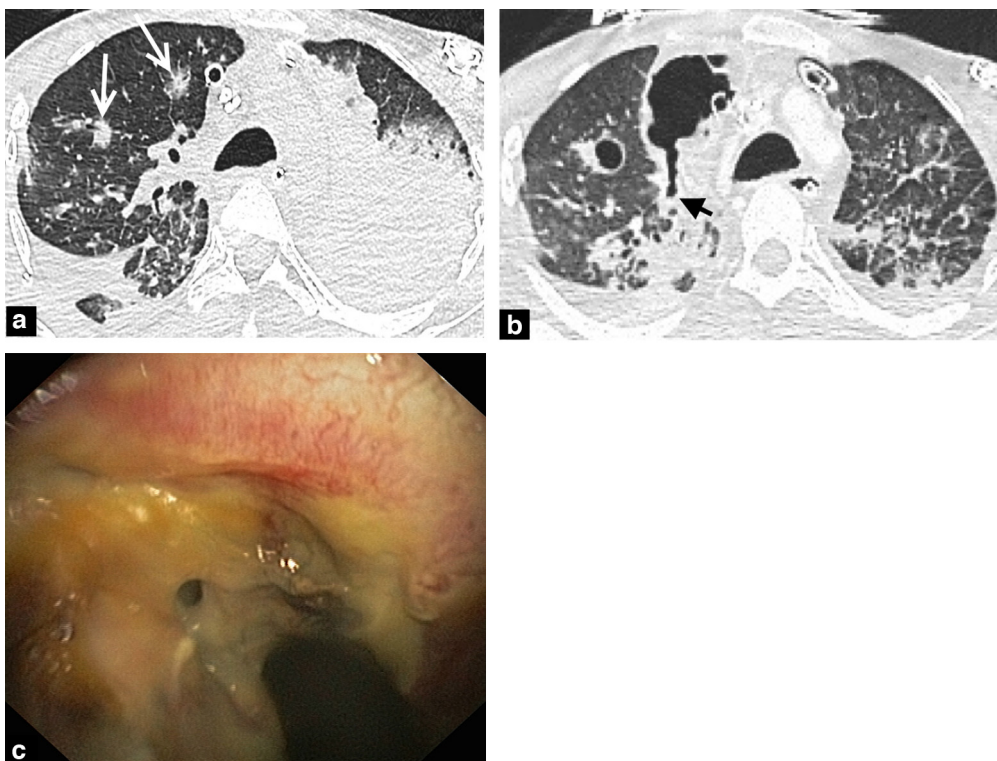


Figure 15. Twenty-two-year-old male patient 1 month after bilateral lung transplants for cystic fibrosis whose general health deteriorated rapidly with dyspnoea. Unenhanced chest CT (Fig. 15a): alveolar opacifications centred on the bronchus and surrounded by a ground glass halo in the right upper lobe (long arrows) due to invasive pulmonary aspergillosis. Repeat CT without enhancement 1 month after onset of symptoms (Fig. 15b): a bronchial fistula (short arrow) has formed and the cavities have increased in size. Bronchoscopy with samples taken (Fig. 15c) confirmed pseudomembranous tracheobronchial aspergillosis.

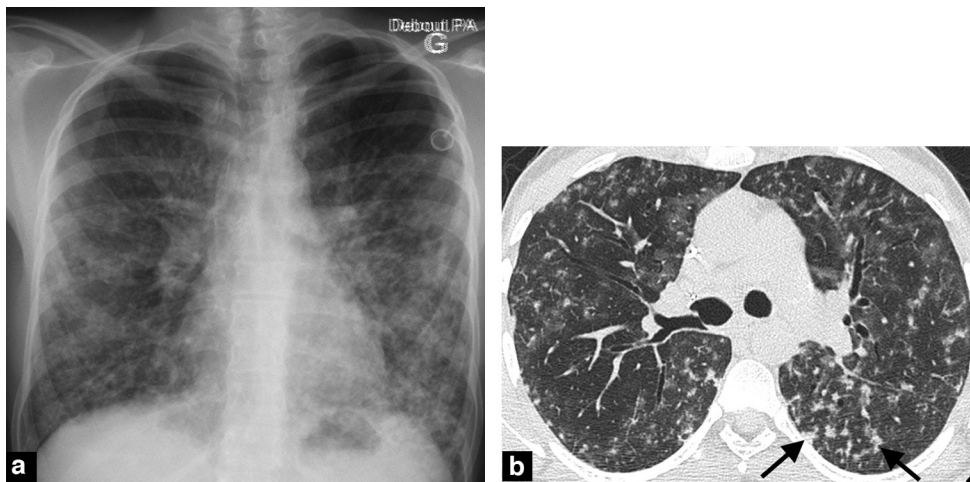


Figure 16. Twenty-eight-year-old male patient 4 months after bilateral lung transplants for cystic fibrosis. Negative CMV serology before the transplant and CMV positive donor. Posteroanterior chest radiograph (Fig. 16a): ground glass opacifications, diffuse micronodules and bilateral basal alveolar opacifications with blurred outlines. Chest CT without enhancement (Fig. 16b): multifocal areas of ground glass changes with blurred centrilobular and peri-lymphatic micronodules (arrow). BAL confirmed CMV pneumonia.

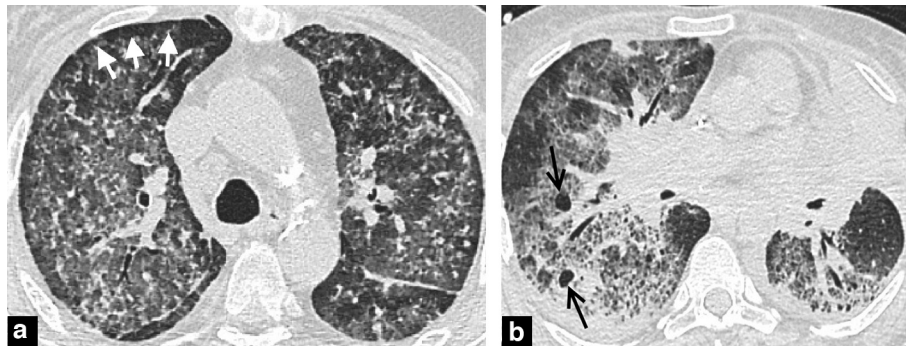


Figure 17. Fifty-four-year-old male patient with bilateral lung transplants with *Pneumocystis* infection confirmed on BAL. Chest CT without enhancement (Fig. 17a): diffuse bilateral ground glass areas, intralobular reticulations and septal thickening. Note a spared subpleural band (short arrows), which is commonly seen in *Pneumocystis* infection. CT of another transplant patient (Fig. 17b): diffuse ground glass infiltrations with thickening of the inter- and intralobular septa ('crazy paving'), areas of consolidation and development of several cystic structures (long arrows): typical *Pneumocystis* infection.

very suggestive of the diagnosis and may be complicated by pneumothorax or pneumomediastinum (Fig. 17) [28].

Other complications

Pulmonary embolism and infarction

Pulmonary embolism is reported to occur in 27% of transplant patients, mostly during the first 4 months following the transplant [8,29]. Pulmonary infarction is then almost invariable in the early postoperative period because of a lack of systemic circulation in the transplanted lung.

Neoplasia

The lymphoproliferative syndrome (post-transplant lymphoproliferative disorder: PTLD) groups together a spectrum of

diseases ranging from acute infectious mononucleosis to high grade immunoblastic lymphoma. The risk of developing this complication is 33% in pre-transplant EBV negative recipients and less than 2% in EBV positive recipients [30].

On imaging, pulmonary lymphoma appears as one or more or less nodular parenchymal opacifications (Fig. 18).

Single-lung transplant patients transplanted for emphysema due to smoking are at high risk of developing lung cancer in their other lung, with a 4 year incidence of 7% [31].

Cryptogenic organised pneumonia

Cryptogenic organised pneumonia occurs in 10 to 28% of lung transplant patients.

CT shows multiple subpleural, migrating alveolar consolidations; often producing an air bronchogram and

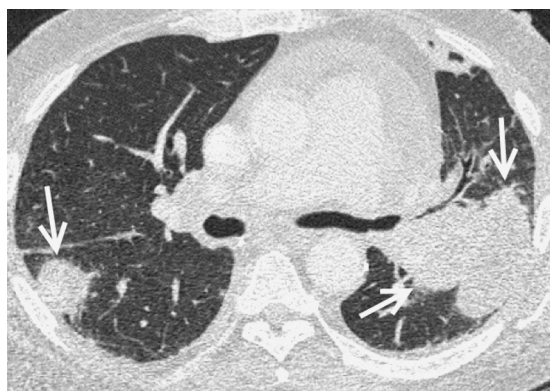


Figure 18. Fifty-one-year-old male patient 3 years after bilateral lung transplants for chronic bronchitis due to smoking. Multiple bilateral parenchymal opacifications (arrows): lymphoma confirmed on trans-bronchial biopsies.

occasionally with linear or reticular opacifications, bronchiectasis, ground glass appearances, air trapping and even fibrosis [31].

Neurotoxicity of the immunosuppressants

Neurotoxicity occurs in 10 to 30% of cases with the calcineurin inhibitors and their analogues. Multiple peripheral and central sensory and/or motor neurological effects occur.

MRI shows hyperintensity on T2 weighted FLAIR imaging in the subcortical white matter, mostly in the parieto-occipital region and relatively symmetrical. These cerebral lesions are commonly grouped together under the term “immunosuppressant-related leukoencephalopathy”.

Upper lobe fibrosis

Progressive upper lobe fibrosis develops one to 4 years after lung transplant [8,32].

Imaging shows a thickening of the interlobular septa, honeycomb appearances, intralobular reticulations, traction bronchiectasis, distortion of the lung architecture and loss of lung volume.

Recurrence of the primary disease

Recurrence of the primary disease occurs from 2 weeks until up to 2 years after lung transplantation. The most common disease to recur is sarcoidosis, in approximately 35% of patients [33].

Conclusion

Radiologists can guide clinicians through the broad spectrum of complications which occur after lung transplant from the radiological and computer tomography findings and time to onset of the complication following surgery (Fig. 19).

Follow up imaging is essential and is guided by the patient’s clinical course and respiratory function tests. The following monitoring protocol can be recommended:

- chest radiograph in the operating room at the end of the procedure in order to identify early complications which are mostly haemorrhage and reperfusion oedema;
- take a follow-up film every 24 hours for the first 14 days for tube positioning, fluid overload, infection and acute rejection;
- chest CT angiogram in a mixed arterial phase (approximately 35 to 40 seconds after contrast injection) 1 month after the transplant in order to check the vascular and bronchial anastomoses and obtain a morphological baseline which is useful for follow-up;
- unenhanced chest CT in inspiration and expiration from 6 months postoperatively and annually thereafter, to investigate for chronic rejection (bronchiolitis obliterans), check the bronchial anastomosis and investigate for other complications.

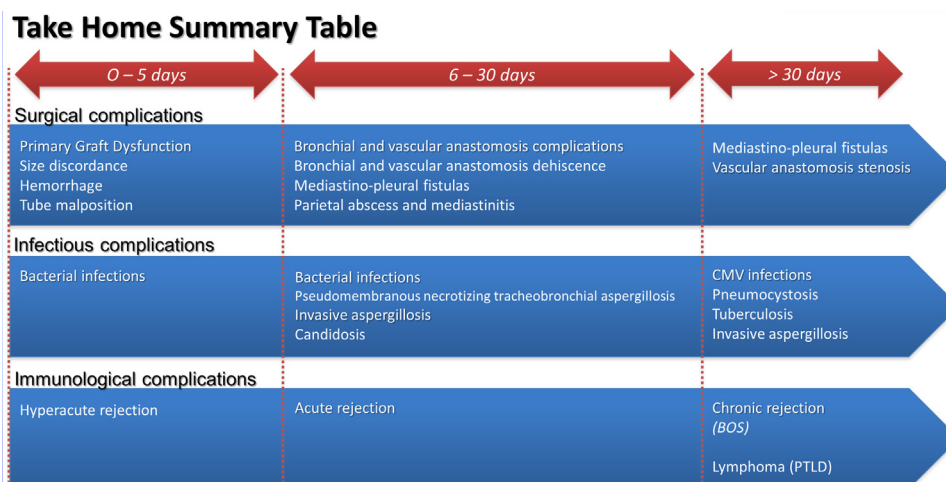


Figure 19. Main complications of lung transplant by postoperative time.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References

- [1] Reynaud-Gaubert M, Houssière A. Résultats de la transplantation en 2010. *Rev Mal Respir* 2010;2:587–90.
- [2] Diez Martinez P, Pakkal M, Prenovault J, Chevrier MC, Chalaoui J, Gorgos A, et al. Postoperative imaging after lung transplantation. *Clin Imaging* 2013;37:617–23.
- [3] Quétant S, RoCHAT T, Pison C. Résultats de la transplantation pulmonaire. *Rev Mal Respir* 2010;27:921–38.
- [4] Ng Y, Paul N, Patsios D, Walsham A, Chung T, Keshavjee S, et al. Imaging of lung transplantation: review. *AJR Am J Roentgenol* 2009;192:51–13.
- [5] Soyer P, Devine N, Frachon I, Vinatier I, Stern M, Normand SL, et al. Computed tomography of complications of lung transplantation. *Eur Radiol* 1997;7:847–53.
- [6] Garg K, Zamora MR, Tuder R, Armstrong JD, Lynch DA. Lung transplantation: indications, donor and recipient selection, and imaging of complications. *Radiographics* 1996;16:355–67.
- [7] Dumonceaux M, Knopp C, Rondelet B, Estenne M. Complications de la transplantation pulmonaire: complications péri-opératoires, rejet aigu et chronique. *Rev Mal Respir* 2009;26:639–53.
- [8] Krishnam MS, Suh RD, Tomasian A, Goldin JG, Lai C, Brown K, et al. Postoperative complications of lung transplantation: radiologic findings along a time continuum. *Radiographics* 2007;27:957–74.
- [9] Isnard J, Trogrlic S, Haloun A, Sagan C, Germaud P, Bommart S, et al. Heart and heart-lung transplants thorax complications: major radiologic forms. *J Radiol* 2007;88:339–48.
- [10] Kundu S, Herman SJ, Winton TL. Reperfusion edema after lung transplantation: radiographic manifestations. *Radiology* 1998;206:75–80.
- [11] Devine N, Soyer P, Rebibo G, Colchen A, Leroy M, Stern M, et al. Bronchial anastomotic complications after pulmonary transplantation. X-ray computed tomographic evaluation. *J Radiol* 1996;77:477–81.
- [12] Hachem R, Meyers B, Yusen R, Patterson A, Trulock E. Airway complications after lung transplantation: incidence and risk factor in a contemporary series. *J Heart Lung Transplant* 2008;27:107.
- [13] Semenkovich JW, Glazer HS, Anderson DC, Arcidi JM, Cooper JD, Patterson GA. Bronchial dehiscence in lung transplantation: CT evaluation. *Radiology* 1995;194:205–8.
- [14] Schlueter FJ, Semenkovich JW, Glazer HS, Arcidi JM, Trulock EP, Patterson GA. Bronchial dehiscence after lung transplantation: correlation of CT findings with clinical outcome. *Radiology* 1996;199:849–54.
- [15] Bonnette P. Logistic, technic and postoperative complications of lung and heart-lung transplantations. *Rev Pneumol Clin* 2011;67:15–20.
- [16] Frost AE, Jammal CT, Cagle PT. Hyperacute rejection following lung transplantation. *Chest* 1996;110:559–62.
- [17] Christie J, Edwards L, Aurora P, Dobbels F, Kirk R, Rahmel A. Registry of the International Society for Heart and Lung Transplantation: 26th official adult lung and heart-lung transplantation report. *J Heart Lung Transplant* 2009;28:1031–49.
- [18] Knopp C, Estenne M. Acute and chronic rejection after lung transplantation. *Semin Respir Crit Care Med* 2006;27:521–33.
- [19] McNell K, Glanville A, Wahlers T, Knopp C, Speich R, Mamelok R, et al. Comparison of mycophenolate mofetil and azathioprine for prevention of bronchiolitis obliterans syndrome in de novo lung transplant recipients. *Transplantation* 2006;81:998–1003.
- [20] Bergin CJ, Castellino RA, Blank N, Berry GJ, Sibley RK, Starnes VA. Acute lung rejection after heart-lung transplantation: correlation of findings on chest radiographs with lung biopsy results. *AJR Am J Roentgenol* 1990;155:23–7.
- [21] Chabot F, Vial B, Siat J, Robert V, Borrelly J, Polu JM. La transplantation pulmonaire: indications, techniques et résultats. *Rev Pneumol Clin* 2000;56:301–12.
- [22] Bankier AA, Van Muylem A, Knoop C, Estenne M, Gevenoix PA. Bronchiolitis obliterans syndrome in heart-lung transplant recipients: diagnosis with expiratory CT. *Radiology* 2001;218:533–9.
- [23] Worthy S, Park C, Kim J, Müller N. Bronchiolitis obliterans after lung transplantation: high-resolution CT findings in 15 patients. *AJR Am J Roentgenol* 1997;169:673–7.
- [24] Avery RK. Infections after lung transplantation. *Semin Respir Crit Care Med* 2006;27:544–51.
- [25] Silveira F, Husain S. Fungal infections in lung transplant recipient. *Curr Opin Pulm Med* 2008;14:211–8.
- [26] Knoop C, Rondelet B, Dumonceaux M, Estenne M. Complications non chirurgicales de la transplantation pulmonaire. *Rev Pneumol Clin* 2011;67:28–49.
- [27] Ko JP, Shepard JAO, Sproule MW, Trotman-Dickenson B, Drucker EA, Ginns LC, et al. CT manifestations of respiratory syncytial virus infection in lung transplant recipients. *J Comput Assist Tomogr* 2000;24:235–41.
- [28] Kuhlman JE. Pneumocystic infections: the radiologist's perspective. *Radiology* 1996;198:623–35.
- [29] Burns K, Iacono A. Pulmonary embolism on postmortem examination: an under-recognized complication in lung-transplant recipients? *Transplantation* 2004;77:692–8.
- [30] Aris R, Maia D, Neuringer I, Gott K, Killey S, Gertis K. Post-transplantation lymphoproliferative disorder in the Epstein-Barr virus naïve lung transplant recipient. *Am J Respir Crit Care Med* 1996;154:1712–7.
- [31] Dickson R, Davis R, Rea J, Palmer S. High frequency of bronchogenic carcinoma after single-lung transplantation. *J Heart Lung Transplant* 2006;25:1297–301.
- [32] Konec E, Weisbrod GL, Pakhale S, Chung T, Paul NS, Hutcheon MA. Fibrosis of the upper lobes: a newly identified late-onset complication after lung transplantation? *AJR Am J Roentgenol* 2003;181:1539–43.
- [33] Collins J, Hartman MJ, Warner TF, Muller NL, Kazerooni EA, McAdams HP, et al. Frequency and CT findings of recurrent disease after lung transplantation. *Radiology* 2001;219:503–9.
- [34] de Broucker T, Pontana F, Santangelo T, Faivre JB, Tacelli N, Delannoy-Deken V, et al. Single- and dual-source chest CT protocols: levels of radiation dose in routine clinical practice. *Diagn Interv Imaging* 2012;93:852–8.