Introduction

With increasing frequency, imaging tests are performed in elderly patients; this is due to a progressive increase in life expectancy, which in turn is due to improved living conditions and medical advances.\(^1\)\(^2\) In the elderly, it is often difficult to establish what normality is, or rather, what changes are consistent with the aging process. This is due to the numerous anatomical and physiological changes that occur during the aging process. In clinical practice, the challenge is to determine the extent to which the changes found in elderly individuals are due to the aging process.\(^3\) The objective of the present review was to describe the most common aging-related chest imaging findings.

We conducted a systematic review of the medical literature on the subject, covering the period between 1950 and 2011 and including articles in Portuguese, English, French, Italian, and Spanish. We search the PubMed, LILACS, and SciELO databases, using the search terms “age”, “aging”, “lung”, “thorax”, “chest”, “X-ray”, “radiography”, “pulmonary”, and “computed tomography”—as well as their corresponding translations—in various combinations. We included only original or review articles on aging-related chest imaging findings. In broad terms, aging results in physiological modifications that must be recognized so as not to be erroneously interpreted as pathological.

Keywords: Aging; Thorax; Lung; Diagnostic imaging.

Abstract

In the elderly (conventionally defined as individuals ≥ 60 years of age), it is often difficult to establish what normality is, because of the numerous anatomical and physiological modifications that occur during the aging process. As a result, the greatest challenge is to differentiate between the normal aging process and the onset of disease. Healthy elderly people commonly present borderline findings on chest imaging. We systematically reviewed the medical literature on the subject, covering the period between 1950 and 2011, including articles in Portuguese, English, French, Italian, and Spanish. We searched the PubMed, LILACS, and SciELO databases, using the search terms “age”, “aging”, “lung”, “thorax”, “chest”, “X-ray”, “radiography”, “pulmonary”, and “computed tomography”—as well as their corresponding translations—in various combinations. We included only original or review articles on aging-related chest imaging findings. In broad terms, aging results in physiological modifications that must be recognized so as not to be erroneously interpreted as pathological.

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studies have established the age at which this finding is first seen, this is known to be due to aging-related muscle mass loss, becoming more pronounced with age.\(^4\)-\(^{10}\) However, there are no objective criteria for the diagnosis of this imaging finding. Another common finding is costal cartilage calcification, which is seen as small islands of compact bone tissue or as nodules, being mistaken for solitary pulmonary nodules in some cases.\(^1\)

The spinal column is another site where age-related degenerative changes are common. The main changes are osteoporosis and spondylosis. The term spondylosis refers to degenerative changes of the spinal column, including reduced intervertebral space, bone sclerosis adjacent to the intervertebral discs, and marginal vertebral osteophytes. In general, vertebral osteophytes are more commonly seen on the right side of the spinal column, which is due to the presence of the descending aorta on the left side. The association of pronounced dorsal kyphosis with a more convex sternum contributes to the so-called “barrel chest” deformity, a phenotypic configuration of the chest in elderly individuals. When combined, these parietal changes cause stiffening of the chest wall, having an unfavorable impact on respiratory mechanics.\(^{11}\)-\(^{13}\)

Barrel chest is an imaging finding that is typically (but not exclusively) seen in elderly individuals. The differential diagnosis should be made primarily with COPD. The diagnosis of COPD should be based on other findings, such as pulmonary emphysema, bronchial wall

### Aging-related changes in radiological findings in the chest wall

One of the most common imaging findings in the aging chest wall is a reduction in the thickness of the parietal muscles, particularly when compared with that of those in younger individuals, a change that can be easily seen on CT scans (Figure 1). This reduction is one of the major causes of increased pulmonary transparency on chest X-rays in the elderly. Although no

![Figure 1 - CT Images. In A, a 25-year-old individual, and in B, an 86-year-old individual. Note the difference between the two in terms of the thickness of the parietal muscles. Note also the liposubstitution (hypodense areas) of the longissimus thoracis in B (arrows).](image-url)
thickening, and bronchiectasis.\(^{14,15}\) In patients with COPD, age is a factor that certainly contributes to disease progression. However, the individual roles of age and COPD in barrel chest cannot be determined by imaging tests.

Common imaging findings in the elderly include diaphragmatic bulging due to muscle hypertrophy and dyskinesia in some areas, particularly on the right side, probably caused by the effort of the hemidiaphragm to maintain the anatomical relationship between the lung and the liver.\(^{11,13}\)

**Aging-related changes in radiological findings in the mediastinum**

Approximately 10% of the elderly population presents with changes that are exclusively related to cardiac aging. This select group of individuals is characterized by the exclusive presence of primary findings of cardiac aging, i.e., radiological findings that are not related to common comorbidities in this age group (arterial hypertension, COPD, atherosclerosis, diabetes, and renal failure).\(^{16,17}\)

The most common physiological change related to cardiovascular aging is diastolic dysfunction, which is due to increased left ventricular muscle mass (due to hypertrophy) and age-related changes in the elastic properties of the myocardium.\(^{18,19}\)

The principal radiological features of the “aging heart” include increased myocardial muscle mass and thickness (in particular, increased left ventricular muscle mass) due to myocyte hypertrophy and increased connective tissue matrix (Figure 2); marginal thickening of the heart valves (commonly the mitral and aortic valves) due to fat, collagen, and calcium salt deposition, causing wear of the valve annulus and, consequently, mild heart valve regurgitation in 90% of healthy patients over 80 years of age; and coronary sclerosis, possibly leading to changes in myocardial perfusion.\(^{16,17,20-23}\) Although most of these changes have no clinical significance in healthy patients, they can contribute to decompensation in cases of cardiac overload due to external factors, such as infectious processes.

In most cases, the signs of right heart overload are due to increased pulmonary capillary resistance (as occurs in COPD and mitral valve dysfunction) and generally have a pathological basis; in contrast, the signs of left heart overload (left ventricular hypertrophy) are exclusively associated with cardiac aging in some cases.\(^{22}\)

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**Figure 2** - Cardiac CT scan. In A, reconstruction of an aortic valve during systole, showing thickening of the valve leaflets in an 88-year-old patient with no history of cardiovascular disease. Valve leaflet thickening is a finding that is considered to be characteristic of normal cardiovascular aging. In B, three-dimensional reconstruction of these findings.
Changes in the aorta include elongation and dilation, which are the major factors responsible for the chest X-ray finding of upper mediastinal enlargement in the elderly. In most patients, parietal calcifications of the aorta are most commonly seen in the aortic arch and in the descending portion of the aorta, constituting nonspecific radiological findings. However, in elderly individuals, calcification of the thoracic aorta, heart valves, and coronary arteries indicates a higher risk of cardiovascular diseases. 

### Aging-related changes in radiological findings in the lung parenchyma

During the first two decades of life, the lungs grow and mature. The maximum number of alveoli is reached at approximately 10-12 years of age, and maturation of the respiratory system occurs at approximately 20 years of age in females and at approximately 25 years of age in males.

Decreased lung function is due to imbalances triggered by changes in the ventilation/perfusion ratio in patients at a more advanced age; however, together, these changes account for less than 3% of the total cardiac output, leading to a minimal (6-mmHg) decrease in PaO$_2$ and having no clinical significance unless pulmonary function is affected by an underlying disease. 

One of the major aging-related physiological changes is decreased lung compliance. The two components of the elastic properties of the lung are surface and tissue forces. There is no evidence that the surface-active lining of terminal respiratory units changes its basic mechanical behavior with age. No changes in the quality or quantity of alveolar surfactant have been described, and there is no evidence of changes in type II pneumocyte function. However, changes in the lung parenchyma and chest wall are functionally significant.

Chest wall compliance decreases with age, which is principally due to musculoskeletal limitations, such as vertebral fractures, spondylosis, and progressive loss of respiratory muscle strength. Lung parenchymal compliance normally decreases with age. These changes are generally attributed to changes in lung connective tissue. However, biochemical studies have suggested that the total collagen and elastin content of the lung does not change with age. Rather, collagen becomes more stable because of an increase in the number of intermolecular cross-links. The most widely accepted hypothesis is that lung elastic recoil is lost because of changes in the spatial arrangement of the network of collagen fibers or because of a protein known as pseudoelastin.

Studies of the senescence-accelerated mouse have shown remarkably increased alveolar duct size during the aging process. Enlarged terminal air spaces have also been reported, having been characterized as a relatively homogeneous destruction, with few cellular infiltrates in the alveoli, suggesting that, unlike what occurs in emphysema, air space enlargement was not due to inflammation of the lung parenchyma. 

Turner et al. found that the ratio of lung weight to body weight does not decrease with age in individuals in the 20-60 year age bracket, a finding that suggests little or no lung destruction, or tissue replacement. During the aging process, the alveolar ducts increase in diameter and the alveoli become larger and shallower. After the fourth decade of life, part of the elastic fibers in the respiratory bronchioles and alveoli degenerate, their complacency therefore decreasing. These changes are more pronounced around the alveolar ducts. Consequently, there is alveolar duct dilatation, followed by air space enlargement. This enlargement is remarkably homogeneous, unlike the irregular distribution of air space enlargement in emphysema (Figures 3 and 4).

Morphometric studies have shown a progressive increase in the average distance between air space walls, as well as a decrease in the surface area of air space wall per unit of lung volume. These changes begin in the third decade of life and progress linearly and continuously, resulting in a 25-30% decrease in the surface area of air space wall per unit of lung volume in nonagenarians. Although these changes are histologically different from those seen in pulmonary emphysema, in which there is destruction of the alveolar walls, they result in similar changes in lung compliance. As occurs with pulmonary emphysema, these changes cause a reduction in the supporting tissues around the airways, where there is a trend toward collapse of small (<2-mm) airways and, consequently, changes in airflow. These morphostructural changes in the lung parenchyma constitute an aging-
The extent of the influence of each of these factors has yet to be determined. The initial pathophysiological consequence of these changes is air trapping due to distal airway closure, with a progressive increase in RV. This related phenomenon that has been designated “senile emphysema” (Figure 3). Aging-related parenchymal changes are caused by reduced blood flow from the systemic circulation through the bronchial arteries, as well as by the aforementioned quantitative/qualitative changes in collagen and in lung compliance. The extent of the influence of each of these factors has yet to be determined. The initial pathophysiological consequence of these changes is air trapping due to distal airway closure, with a progressive increase in RV. This

Figure 3 - Three-dimensional CT scan showing (in A) areas of increased small airway volume, known as senile emphysema (blue areas), in an 85-year-old patient. Note the homogeneous distribution of emphysematous areas. In B, three-dimensional CT scan of a healthy 23-year-old patient. Note that there are no areas suggestive of pulmonary emphysema.

Figure 4 - Axial CT scan in a 78-year-old individual (in A) and in a six-month-old child (in B). Note the difference between the two in terms of parenchymal attenuation (denser in the child).
pulmonary hypertension (a clinical manifestation of mild vascular sclerosis) that can redistribute pulmonary flow cranially and be mistaken for early signs of cardiac decompensation.

The major determinants of static lung volumes are chest wall compliance and lung parenchymal compliance. Loss of lung parenchymal compliance and, to a lesser degree, decreased respiratory muscle strength result in an increase in RV (air

mechanism is analogous to that of pulmonary emphysema, with no signs of inflammation and no significant increase in TLC. At the same time, the ventilation/perfusion ratio changes because of a reduction in the number of alveoli with normal gas exchange; this has two pathophysiological consequences: increased physiological dead space and the shunt effect, both of which lead to a decrease in $\text{PaO}_2$.\(^{39,41}\) In addition, there is mild pulmonary hypertension (a clinical manifestation of mild vascular sclerosis) that can redistribute pulmonary flow cranially and be mistaken for early signs of cardiac decompensation.\(^{41-43}\)

The major determinants of static lung volumes are chest wall compliance and lung parenchymal compliance. Loss of lung parenchymal compliance and, to a lesser degree, decreased respiratory muscle strength result in an increase in RV (air

Figure 5 - Asymptomatic 87-year-old female patient. In A, axial CT scan of the left lower lobe showing laminar atelectasis at the lung bases. In B, sagittal reconstruction of this finding.

Figure 6 - Asymptomatic 83-year-old female patient. Axial CT scan taken in the prone position, showing subpleural linear septal thickening (arrows). These radiological findings are indistinguishable from those seen in patients with interstitial lung disease due to other causes.

Figure 7 - Asymptomatic 87-year-old female patient. Axial CT scan with minimum intensity projection reconstruction, a technique that highlights the lowest density areas of the lung parenchyma, showing extensive areas of air trapping. The patient had normal pulmonary function test results.
Likewise, correlation with pulmonary function test results (particularly DLCO) can demonstrate how gas exchange is occurring and guide a conservative approach. Another fact that should be taken into consideration in the elderly is life expectancy and the metabolic need for gas exchange, given that patients whose activity is limited by extrathoracic disease have lower physiological needs. This brings us to the well-known Hippocratic principle of *primum non nocere* (above all, do no harm), which is increasingly true today, given the various choices of procedures and the increase in survival of the population.

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