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Celiac disease and lung: a monocentric retrospective study

Oriane Charpentier

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UNIVERSITÉ CLERMONT AUVERGNE
UFR DE MÉDECINE ET DES PROFESSIONS PARAMÉDICALES

THÈSE D'EXERCICE
pour le
DIPLOME D'ÉTAT DE DOCTEUR EN MÉDECINE
par

CHARPENTIER Oriane

Présentée et soutenue publiquement le vendredi 27 mai 2022

MALADIE COELIAQUE et POUMON :
Une étude monocentrique et rétrospective

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TABLE DES MATIÈRES

INTRODUCTION	19
PATIENTS AND METHODS	19
RESULTS	23
Characteristics of the patients	23
HRCT scan analysis	24
Confounding factors analysis	27
DISCUSSION	29
CONCLUSION	33
REFERENCES	34
Annexe 1	37
Annexe 2	38
Annexe 3	39

LISTE DES TABLEAUX ET FIGURES

TABLE

TABLE 1. PATIENT CHARACTERISTICS.....	25
TABLE 2. HRCT SCANNER ANALYSIS.	26
TABLE 3. CT SCAN ANOMALIES ACCORDING TO PATIENT CHARACTERISTICS.....	28

FIGURES

FIGURE 1. FLOW-CHART.	23
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LISTE DES ABRÉVIATIONS

CD: celiac disease

ILD: interstitial lung disease

IBD: inflammatory bowel disease

PFT: pulmonary function test

COPD: chronic obstructive pulmonary disease

FEV1: forced expiratory volume in 1 second

HRCT: high-resolution computed tomography

UH: university hospital

CPP: Comité de Protection des Personnes

TLC: total lung capacity

FVC: forced vital capacity

DLCO: diffusing capacity for carbon monoxide

MPR: multiplanar reformations

MIP: maximum intensity projection

MinIP: minimum intensity projection

ATS: American Thoracic Society

ERS: European Respiratory Society

MCA: multiple correspondence analysis

MGUS: monoclonal gammopathy of undetermined significance

HLA: human leucocyte antigen

UIP: usual interstitial pneumonia

ILA: interstitial lung abnormalities

PA: pulmonary arterial

ADM: adenomely

α 1AT: alpha 1 antitrypsin

BMI: body mass index

BPCO: bronchopneumopathie chronique obstructive

EFR: explorations fonctionnelles respiratoires

TDM: tomodensitométrie

TVO: trouble ventilatoire obstructif

TVR : trouble ventilatoire restrictif

CELIAC DISEASE and LUNG: a monocentric retrospective study

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ABSTRACT

Background: Lung involvement specifically linked to celiac disease (CD), such as bronchial and bronchiolar (asthma, COPD) or sarcoidosis, has been suggested in the literature but without any well-conducted descriptive study to measure the association except for pulmonary hemosiderosis in paediatric population. No study analysing lung involvement on HRCT scanning has been conducted in a population of adult patients with CD.

Objective: To determine if there is a link between CD and specific lung involvement found on chest HRCT.

Methods: Monocentric retrospective study conducted in patients followed for CD and underwent chest HRCT at the time of diagnosis or during follow-up, analysed by two independent and double-blinded radiologists.

Results: Two hundred and forty-two patients were screened. $n =$ Thirty-seven patients fulfilled inclusion criteria and had chest HRCT without drug induced interstitial lung disease (ILD), hypersensitivity pneumonitis, history of thoracic radiotherapy or infectious lung disease. The three most common involvements found were nodules ($n = 24$, 64.9%), then emphysema ($n = 10$, 27%; including $n = 7$, 18.9% smokers), bronchial thickening ($n = 10$, 27%), and bronchiectasis ($n = 9$, 24.3%). Nodules, bronchial thickening and bronchiectasis were independently linked to celiac disease, contrary to emphysema which was associated with COPD $p = 0.019$, tobacco exposure $p = 0.018$ and men, $p = 0.009$. None had relationship with well-follow gluten free diet. Clinical findings during CT scan and symptoms were cough ($n = 12$, 32.4%), dyspnoea ($n = 8$, 21.6%), inspiratory crackles ($n = 5$, 13.5%), snorted ($n = 3$, 8.1%) and wheezing ($n = 2$, 5.4%). Pulmonary function tests (PFT) were analysed in 8 (21.6%) cases, including 5 with obstructive ventilatory disorder, 2 with restrictive ventilatory disorder and 4 with a diffusion impairment.

Conclusion: CD is an independent risk factor of developing tracheobronchial damages and nodules independently tobacco, COPD, pneumonitis history and diet. In patients with CD, a clinical screening of pulmonary symptoms could be interesting and patients may need PFT during the follow-up with chest HRCT if there is an anormal PFT and/or pulmonary symptoms.

INTRODUCTION

Celiac disease (CD) is a chronic multi-visceral autoimmune disease that targets the small intestine in genetically predisposed people, caused by the ingestion of gluten[1]. It is a rare disease (0.7–1.4%)[2], but the prevalence is probably underestimated and increased significantly over the last 50 years[1, 3]. The main manifestations of CD are intestinal (diarrhoea, abdominal pain, weight loss, constipation), but some extra-intestinal manifestations are possible (dermatological, haematological, rheumatological, etc...)[1–3]. The gluten-free diet is the main and essential treatment, but it is difficult to follow across the life. Acknowledgment about lung involvement in CD is scarce contrary to inflammatory bowel disease (IBD) where various pulmonary disorders are described (chronic bronchitis, asthma, interstitial lung disease (ILD), bronchiectasis, bronchiolitis, nodules, tracheobronchial stenosis, pleuritis)[4]. In CD, only pulmonary hemosiderosis in paediatric population is well described[5–7]. However, a statistically significant association between asthma and CD is reported in children[8, 9]. An association with sarcoidosis is suggested[10, 11]. *Ludvigsson and al.* demonstrate an increased risk of having COPD in celiac patients[12], with a significant reduction in airflow (FEV1)[13]. A few case reports of lung damage specifically linked to CD (bronchial, bronchiolar damage, etc.) are described in the literature[14–16]. We conducted a monocentric retrospective study on adult patients with CD and undergone chest HRCT during their follow-up.

PATIENTS AND METHODS

Population studied. Retrospective study included patients with CD followed in the Internal Medicine, Pulmonology and Gastroenterology Departments at Clermont-Ferrand University Hospital (UH), Clermont-Ferrand, France.

Selection method. We searched the institution's medical record index for patients examined between January 1990 and December 2020 using the diagnostic code 'maladie cœliaque'.

Ethics approval. This study was conducted in compliance with the Declaration of Helsinki principles and is awaiting approval of an Ethic Committee from France; CPP Sud Est n°....

Inclusion criteria. Patients with:

- CD, confirmed by duodenal biopsies, which is the gold standard, interpreted through its progressive phases using the Marsh classification[17, 18]. The Modified Marsh Classification (Oberhuber) broadly itemises those stages through which a normal mucosa (Marsh 0) evolves in becoming ‘flat’ according to increased intraepithelial lymphocytes, crypt hyperplasia and villous atrophy (Marsh 1, 2, 3a, 3b, 3c – Marsh 4 if crypt hyperplasia absent)

And

- Undergone a good quality high resolution chest CT (HRCT) scan at diagnosis or during the follow-up.

Data collected: in patient medical records and medical visits. Data screened (annexe 1) were:

- Sex, age at diagnosis, age at CT scan, respiratory symptoms, pulmonary history, tobacco exposure (weaned or not), inhaled treatments, history of autoimmune disease, IgA deficiency, HLA status, history of bowel lymphoma, and diet during the period of CT scan.

- Serological status of CD (gliadin (AGA), deaminated gliadin peptides (DGP), tissue transglutaminase (TG) and endomysium (EMA) antibodies) and histology findings of patients were evaluated at CD diagnosis and at time of the chest HRCT (3 months before or after HRCT).

- The following parameters of pulmonary function tests (PFTs) were also evaluated: total lung capacity (TLC), forced vital capacity (FVC), forced expiratory volume in 1 second (FEV1), FEV1/FVC ratio, and diffusing capacity for carbon monoxide (DLCO). TLC was measured by plethysmography, FVC and FEV1 were measured by spirometry (using a water sealed spirometer); DLCO was obtained by the single-breath method. Data are expressed as percentages of the predicted values and litter. The predicted values for each subject, based on sex, age, height, and weight, were obtained from standard tables. Lung function was considered

abnormal when volumes were <80% or >120% of the predicted values, when FEV1/FVC ratio was <0.7 and/or when DLCO was <70% of the predicted value.

HRCT scan analysis. All chest CT-scanners were analyzed on a dedicated software by the Maincare PACS of the UH of Clermont Ferrand (Change Healthcare Radiology solutions, Version 12.4.1), in an independent and double-blinded process by a resident radiologist (7th semester) (P.T.) and a (senior) radiologist (P.A.) specialized in thoracic imaging. For each analyzed CT-scanner, the two radiologists completed a standardized grid on the RedCap software, consisting of 57 items of imagery data, and 6 items of general data regarding the indication of the exam and its precise technique (annexes 2 and 3). A systematical analyze of the images was realized, consisting of an analyze in the 3 anatomical dimensions of reference in axial, coronal and sagittal multiplanar reformations (MPR); with a « hard » filter for the lung window, with a MPR « soft » filter for the mediastinal window, with a « soft » filter for the lung window in the axial plan with MIP (Maximum Intensity Projection) reconstructions of 5-10 mm, and finally with a « soft » filter for the lung window in the coronal plan with variable MinIP (Minimum Intensity Projection) reconstructions (5-7mm).

Definition of interstitial lung disease. According to the American Thoracic Society (ATS)/European Respiratory Society (ERS) international multidisciplinary consensus classification of idiopathic interstitial pneumonia[19], ILD is characterised by the continuous presence of bilateral parenchymal lung infiltrates on chest HRCT.

Exclusion criteria. Patients who did not respond to the CD's classification criteria or without chest HRCT scan were excluded. Patients with drug induced ILD, hypersensitivity pneumonitis, who had undergone thoracic radiotherapy, or bronchial or pulmonary infectious disease at time of the chest HRCT were also excluded.

Data analysis. Categorical parameters were expressed as frequencies and associated percentages, and continuous data as mean and standard deviation or as median and [25th; 75th

percentiles], according to statistical distribution. The assumption of normality (Gaussian) distribution was assessed using the Shapiro-Wilk test. Agreement between observers was assessed by agreement rate and concordance Cohen's kappa coefficient. The results concerning kappa coefficient were interpreted in relation to the recommendations reported in the literature by Altman[20]: <0.4 : no agreement, $0.4-0.7$: poor agreement, >0.7 : moderate to strong agreement. The relationships between categorical data were analyzed using Chi-squared or Fisher's exact tests. For continuous data, the comparisons between independent groups were performed with Student's t-test or the Mann-Whitney test if the assumptions of the t-test were not met. The equality of variances was analyzed by the Fisher-Snedecor test. Due to sample size, non-parametric tests were often preferred. Finally, a multiple correspondence analysis (MCA) was proposed to: (i) study the relations between the modalities of the variables, and (ii) determine groups of patients sharing very similar characteristics. MCA can be used to perform cross tabulations and can be considered as a useful tool for revealing the relationships among categorical variables. The significance of association as tested by Chi-squared or Fisher's exact tests provides no information regarding significant individual associations between row-column pairs. On the contrary, factorial analyses such as MCA show how the variables are related, and not just if a relationship exists. This approach enables handling a large amount of data in order to unfold hidden patterns. For these analyses, the variables were chosen according to aforementioned univariate results, clinical relevance and statistical distribution. Parameters always present or always absent were not considered and only individuals without missing data were included in this analysis. Statistical analyses were performed using Stata software (version 15, StataCorp, College Station, Texas, US) and R 3.3.3 (<http://cran.r-project.org/>). All the tests were two-sided, with a Type I error set at 0.05.

RESULTS

Characteristics of the patients

Of the 242 patients initially selected, 37 patients with CD were included in this study. Thirty-one patients did not fulfilled the required diagnosis of CD criteria, and one hundred and seventy-one patients were excluded (for reasons including no chest HRCT scan, drug induced ILD, hypersensitivity pneumonitis, history of chest irradiation and bronchial or pulmonary infection at time of the HRCT; Figure 1).

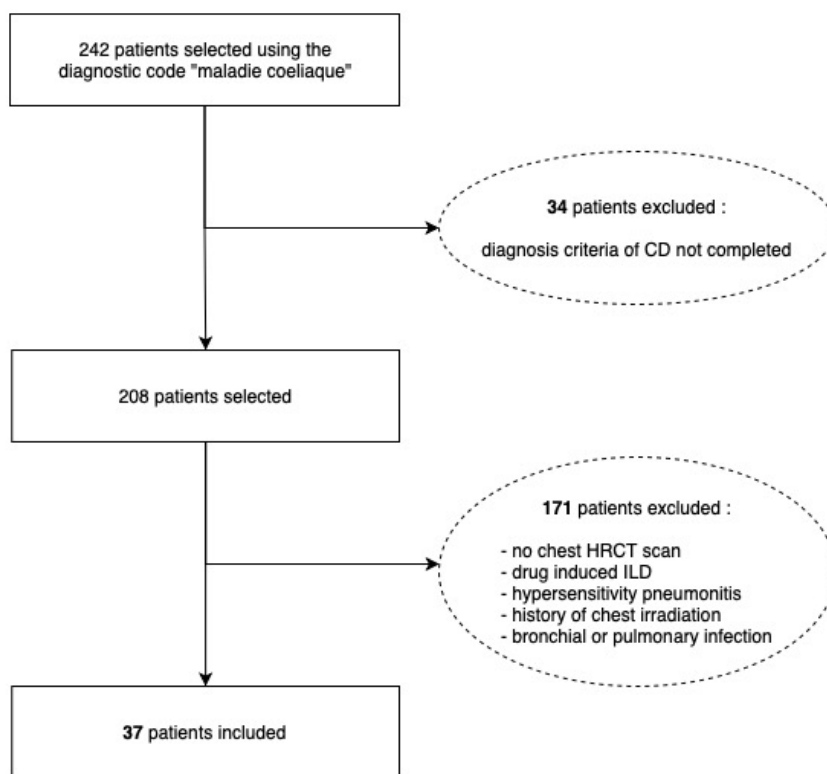


Figure 1. Flow-chart.

The clinical, biological and histological characteristics of the patients are summarised in Table 1. The median age at diagnosis of CD was 53 years (range: 40–66 years) and the median age at the time of chest CT was 64 years (range: 53–71 years). The median follow-up periods before the CT scan was 9 years (range: 4–16 years). Gluten free diet was well-conducted in 62.2% ($n = 23$) of cases. Thirteen patients (35.1%) were smokers (weaned or not), five (13.5%) had chronic obstructive pulmonary disease (COPD) and four (10.8%) had asthma. Four (10.8%) had pneumonitis history but none with tuberculosis. The symptoms or

clinical exam experienced by patients during the HRCT period were: cough ($n = 12$, 32.4%), dyspnoea ($n = 8$, 21.6%), inspiratory crackles ($n = 5$, 13.5%), snorted ($n = 3$, 8.1%) and wheezing ($n = 2$, 5.4%). PFT were performed only in 8 (21.6%) patients and not at the same time of the chest HRCT; 5 had an abnormal FEV1/FVC ratio including 4 with a FEV1 less than 80%, 4 had a DLCO <70%, 2 had a TLC<80% and none with TLC>120%. Among patients with pathological FEV1/FVC, 2 had COPD and 2 had asthma. Among patients with DLCO<70%, 2 had COPD with emphysema and 1 had asthma.

In our population, no patient had sarcoidosis, lupus, psoriasis or IBD. The two predominant dysimmune diseases are Hashimoto thyroiditis ($n = 3$, 8.1%) and monoclonal gammopathy of undetermined significance (MGUS) ($n = 3$, 8.1%, 2 patients with IgG kappa including 1 that progressed to myeloma and 1 patient with IgA kappa fraction). Serological status was missing for 10 patients (27%) at diagnosis and 18 patients (48.6%) at the time of chest HRCT scan. Any was seronegative or IgA deficiency. Histology was missing for 9 patients (24.3%) at diagnosis and 22 patients (59.5%) at the time of CT scan. HLA data were available for only 4 patients (10.8%).

HRCT scan analysis

HRCT scan analysis were found with good agreement between the two radiologists (with a good percentage of agreement and a good kappa correlation coefficient) and summarised in Table 2. The reader 1 was used for the analysis.

Among the 37 patients included, 24 (64.9%) had nodules without suspect aspect like spicular or metastasis of a cancerous mass ($n = 3$). Nineteen patients (51.4%) had a tracheobronchial damage; ten (27%) had bronchial thickening and nine (24.3%) had bronchiectasis. Among patients with tracheobronchial damage, only 4 had PFT and 2 had pathological FEV1/FVC ratio (56.8% and 64.32%) and FEV1 (63% and 75.6%) but both had COPD and tobacco using. Two had pathological DLCO (29% and 33%) but both had COPD with emphysema. One had a normal PFT.

Table 1. Patient characteristics.

	Total n = 37
Median age at CD diagnosis	53 [40; 66]
Median age at chest HRCT	64 [53; 71]
Sex ratio (M/F)	0.4 (10/27)
Tobacco exposure (weaned or not)	13 (35.1%)
Pulmonary history	
COPD	5 (13.5%)
Asthma	4 (10.8%)
Pneumonitis	4 (10.8%)
Pulmonary tuberculosis	0
Respiratory symptoms at chest HRCT	17 (45.9%)
Cough	12 (32.4%)
Dyspnoea	8 (21.6%)
Crackles	5 (13.5%)
Snorted	3 (8.1%)
Wheezing	2 (5.4%)
PFT^a	8 (21.6%)
FEV1/FVC <70%	5 (13.5%)
FEV1 <80%	4 (10.8%)
DLCO <70%	4 (10.8%)
TLC <80%	2 (5.4%)
Immune disease	
Hashimoto thyroiditis	3 (8.1%)
MGUS	3 (8.1%)
Polymyalgia rheumatica	2 (5.4%)
Vitiligo	2 (5.4%)
Type 1 diabetes	2 (5.4%)
Others ^b	7 (18.9%)
Serological status at CD diagnosis	27 (73%)
Tissue transglutaminase antibodies IgA+; IgG+	16 (45.9%); 6 (16.2%)
Deaminated gliadin peptides antibodies IgA+; IgG+	2 (5.4%); 4 (10.8%)
Endomysium antibodies IgA+; IgG+	9 (24.3%); 3 (8.1%)
Anti-gliadin antibodies	9 (24.3%)
Serological status at chest HRCT^c	19 (51.4%)
Tissue transglutaminase antibodies IgA+; IgG+	9 (24.3%); 2 (5.4%)
Seronegative	0
Silent ^d	3 (8.1%)
Histology at CD diagnosis	28 (75.7%)
Marsh 0; 1; 2; 3	0; 1 (2.7%); 1 (2.7%); 26 (70.3%)
Histology at chest HRCT^c	15 (40.5%)
Marsh 0; 1; 2; 3	4 (10.8%); 2 (5.4%); 0; 9 (24.3%)
Refractory sprue ^e	2 (5.4%)
HLA status	4 (10.8%)
DQ2DQ8 +/+; +/-; -/+	1 (2.7%); 3 (8.1%); 0
Bowel lymphoma	2 (5.4%)
Well-follow diet at CT scan	23 (62.2%)

CD: celiac disease; CT: computed tomography; PFT: pulmonary function test; FEV1: forced expiratory volume in 1 s; FVC: forced vital capacity; DLCO: diffusion capacity for carbon monoxide; TLC: total lung capacity; IBD: inflammatory bowel disease; MGUS: monoclonal gammopathy of undetermined significance; HLA: human leucocyte antigen

^aPFT during the follow-up were not realized at the same time of HRCT

^bRheumatoid arthritis n = 1; Scleroderma n = 1; primary Sjogren syndrome n = 1; Horton disease n = 1; Graves' disease n = 1; Spondylarthritis n = 1; Autoimmune neutropenia n = 1

^c3 months before or after HRCT

^dasymptomatic, discovery on family screening or assessment of another autoimmune disease

^efrom CD diagnosis to chest HRCT scan

Table 2. HRCT scanner analysis.

	Reader 1	Reader 2	% agreement (kappa)
CT quality			
Optimal	25 (67.6%)	28 (75.7%)	
Suboptimal	10 (27%)	9 (24.3%)	
Poor	2 (5.4%)	0	
Pattern of interstitial lung disease			
UIP	2 (5.4%)	2 (5.4%)	100 (1.0)
ILA	1 (2.7%)	1 (2.7%)	100 (1.0)
Mosaic lung	2 (5.4%)	2 (5.4%)	100 (1.0)
Tracheobronchial abnormalities	19 (51.4%)	17 (45.9%)	
Bronchiectasis	9 (24.3%)	8 (21.6%)	94.59 (0.84)
Bronchiolectasis	1 (2.7%)	1 (2.7%)	100 (1.0)
Bronchial thickening	10 (27%)	8 (21.6%)	89.19 (0.71)
Mucoid impacts	3 (8.1%)	4 (10.8%)	97.30 (0.84)
Tracheal diverticula	1 (2.7%)	1 (2.7%)	100 (1.0)
Emphysema	10 (27%)	10 (27%)	100 (1.0)
Centrilobular	8 (21.6%)	7 (18.9%)	
Paraseptal	7 (18.9%)	7 (18.9%)	
Panlobular	1 (2.7%)	0	
Bullous	0	0	
Advanced destructive	0	1 (2.7%)	
Left cardiac insufficiency	2 (5.4%)	0	
Mass, nodules or micronodules			
Micronodular syndrome (< 3mm)	1 (2.7%)	1 (2.7%)	100 (1.0)
Mass (> 3 cm)	3 (8.1%)	3 (8.1%)	100 (1.0)
Nodules (3-30 mm)	27 (73%)	25 (67.6%)	96.88 (0.90)
Calcified nodules	6 (16.2%)	4 (10.8%)	
Solitary tissue nodule	3 (8.1%)	3 (8.1%)	
Intrapulmonary lymph node	8 (21.6%)	8 (21.6%)	
Hamartochondroma	1 (2.7%)	1 (2.7%)	
Respiratory disorders	22 (59.5%)	20 (54.1%)	94.59 (0.89)
Subsegmental lamellar atelectasis	17 (45.9%)	14 (37.8%)	
Interstitial abnormalities in contact with a vertebral osteophyte	2 (5.4%)	2 (5.4%)	
Passive collapse on pleural effusion	3 (8.1%)	3 (8.1%)	
Gravity-dependent	6 (16.2%)	7 (18.9%)	
Roll-up/round atelectasis	1 (2.7%)	1 (2.7%)	
Fibro-retractile remodeling/apical caps	6 (16.2%)	4 (10.8%)	94.9 (0.7882)
Pleural lesions	7 (18.9)	7 (18.9)	100 (1.0)
Pleural effusion	6 (16.2%)	6 (16.2%)	100 (1.0)
Pleural thickening	1 (2.7%)	1 (2.7%)	100 (1.0)
Cardiovascular lesions			
Cardiomegaly	14 (37.8%)	11 (29.7%)	
Signs of pulmonary hypertension (PA diameter >29 mm)	5 (14.3%)	5 (14.3%)	100 (1.0)
Pericardial effusion	4 (10.8%)	6 (16.2%)	94.44 (0.77)
Coronary calcifications	15 (40.5%)	16 (43.2%)	
Mediastinal lymph node areas			91.18 (0.74)
A few infracentimetric lymph nodes	2 (5.4%)	5 (14.3%)	
A few ADM of lymphatic stasis or chronic pathology	3 (8.1%)	1 (2.7%)	
Calcified nodes	1 (2.7%)	0	
Pathological ADM	2 (5.4%)	2 (5.4%)	

CT: computed tomography; UIP: usual interstitial pneumonia; ILA: interstitial lung abnormalities; PA: pulmonary arterial; ADM: adenomegaly

Ten (27%) had emphysema, including seven (18.9%) smokers. Among patients with emphysema, only 2 patients had an alpha 1 antitrypsin (α 1AT) assay and they were not deficient. As for the other 8 patients, 6 had a serum protein electrophoresis and none had an α 1 globulin deficiency.

Three patients (8.1%) had ILD, including usual interstitial pneumonia (UIP) ($n = 2$, 5.4%, both smokers, 74 and 59 years old) and interstitial lung abnormalities (ILA) ($n = 1$, 2.7%).

Six patients (16.2%) had fibro-retractile remodelling or apical caps. Among these patients, 3 had a known pneumonia history.

Pleural lesions were found in seven (18.9%) patients; including effusion, $n = 6$ and thickening, $n = 1$.

In mediastinum, eight (21.6%) had pathological lymph node, found in a context of fortuitous screening ($n = 3$), chest pain ($n = 2$), or for other reason ($n = 3$). Four (10.8%) had pericardial effusion; all were minimal and none were detected in a context of chest pain suggestive of pericarditis or inflammatory syndrome. Among them, 2 were malnourished but not severe denutrition with a body mass index (BMI) $>16\text{kg/m}^2$ (16.2 with serum albumin 38.9g/L and 16.6 with serum albumin 30g/L) and 2 had a BMI between 18 and 25kg/m² (24.4 and 21.5, without known albumin assay).

Confounding factors analysis

After exclusion of confounding factors (tobacco exposure, COPD, pneumonitis history) nodules, bronchial thickening and bronchiectasis were independently linked to celiac disease (There is no statistical relation to tobacco exposure: nodules $p = 0.696$, bronchial thickening $p = 0.444$, bronchiectasis $p = 1.00$; COPD: nodules $p = 0.618$, bronchial thickening $p = 0.597$, bronchiectasis, $p = 0.081$; and pneumonitis history: nodules $p = 0.539$, bronchial thickening $p = 0.557$, bronchiectasis $p = 1.00$). Rheumatoid arthritis and sarcoidosis had no influence on the presence or absence of nodules ($p = 1.00$). Asthma had no influence on the presence or absence of bronchiectasis or bronchial thickening ($p = 1.00$). Autoimmune diseases and

immunodeficiency (IgA deficit, MGUS or autoimmune neutropenia) had no influence on the presence or absence of bronchiectasis (autoimmune diseases, $p = 1.00$; and immunodeficiency, $p = 0.09$). However, bronchiectasis are more frequent in elderly patients ($p = 0.0384$, 74 years [65; 77] vs 59 years [50; 67.5]). On the other hand, emphysema is not associated with CD but secondary to tobacco exposure ($p = 0.018$) and COPD ($p = 0.019$). Emphysema is more frequent in men ($p = 0.009$), but men had significantly more tobacco use (8/10 men vs 5/27 women, $p < 0.001$). Well-conducted gluten free diet had no influence on the presence or absence of these anomalies: nodules ($p = 0.185$), bronchial thickening ($p = 0.425$), bronchiectasis ($p = 1.00$). These analyses are summarised in Table 3.

Table 3. CT scan anomalies according to patient characteristics.

	Bronchiectasis		Bronchial thickening		Emphysema		Nodules	
	Yes	No	Yes	No	Yes	No	Yes	No
Median age at CT	74 [65; 77] ^P	59 [50; 67] ^P	61 [58; 79]	65 [47; 71]	59 [58; 75]	63 [47.5; 71]	61 [58; 74]	61 [50; 74]
Sex ratio (M/F)	3/6	7/21	3/7	7/20	6/4 ^P	3/21 ^P	20/4	5/5
Tobacco								
Yes	3 (8.1%)	10 (27%)	5 (13.5%)	8 (21.6%)	7 (18.9%) ^P	5 (13.5%) ^P	8 (21.6%)	4 (10.8%)
No	6 (16.2%)	16 (43.2%)	5 (13.5%)	17 (45.9%)	3 (8.1%) ^P	17 (45.9%) ^P	15 (40.5%)	5 (13.5%)
COPD								
Yes	3 (8.1%)	2 (5.4%)	2 (5.4%)	3 (8.1%)	4 (10.8%) ^P	1 (2.7%) ^P	3 (8.1%)	2 (5.4%)
No	6 (16.2%)	26 (70.3%)	8 (21.6%)	24 (64.9%)	6 (16.2%) ^P	23 (62.1%) ^P	21 (56.8%)	8 (21.6%)
Asthma								
Yes	1 (2.7%)	3 (8.1%)	1 (2.7%)	3 (8.1%)				
No	7 (18.9%)	26 (70.3%)	9 (24.3%)	24 (64.9%)				
Pneumonitis								
Yes	1 (2.7%)	3 (8.1%)	0	4 (10.8%)			3 (8.1%)	0
No	8 (21.6%)	25 (67.6%)	10 (27%)	23 (62.1%)			21 (56.8%)	10 (27%)
Autoimmune disease^a								
Yes	0	2 (5.4%)						
No	9 (24.3%)	26 (70.3%)						
Autoimmune disease^b								
Yes							1 (2.7%)	0
No							23 (62.1%)	10 (27%)
Immunodeficiency^c								
Yes	5 (13.5%)	6 (16.2%)						
No	4 (10.5%)	22 (59.5%)						
Well-conducted gluten free diet								
Yes	5 (13.5%)	18 (48.6%)	5 (13.5%)	18 (48.6%)	5 (13.5%)	17 (45.9%)	18 (48.6%)	4 (10.8%)
No	2 (5.4%)	9 (24.3%)	4 (10.8%)	7 (18.9%)	3 (8.1%)	6 (16.2%)	5 (13.5%)	4 (10.8%)

COPD: Chronic obstructive pulmonary disease

^arheumatoid arthritis, spondyloarthritis, primary Sjogren syndrome, sarcoidosis, lupus and IBD (inflammatory bowel disease)

^brheumatoid arthritis and sarcoidosis

^cIgA deficit, MGUS (monoclonal gammopathy of undetermined significance) and autoimmune neutropenia

^Pp-value<0.05

DISCUSSION

In our knowledge, this is the largest study showing that CD is an independent risk factor for lung damage in chest CT in cohort of adult CD patients and the absence of relationship with well-conducted gluten free diet. Pulmonary nodules, bronchial thickening, and bronchiectasis seems to be frequent and specific to CD.

Lung nodules are a common, usually incidental, finding on chest CT scans. A systematic review of CT screening lung cancer trials noted that a lung nodule was detected in up to 51% of study participants (benign in more than 95% of cases)[21, 22]. This percentage remains below our results (64.9%) and studies about CT scan in the screening of lung cancer include smokers.

In literature, nodules are associated with tobacco, COPD, history of pneumonitis and some autoimmune disease[21, 22]. Emphysema is associated with men, tobacco use, α 1AT deficiency and COPD[23, 24]. Bronchial thickening and non-cystic fibrosis bronchiectasis are associated with tobacco, α 1AT deficiency, COPD and asthma, history of pneumonitis, immunodeficiency (notably humoral deficiency) and autoimmune diseases (rheumatoid arthritis, primary Sjogren syndrome, sarcoidosis, IBD, etc...)[24–29].

To our knowledge, it is the first study excluding these confounding factors (except α 1AT deficiency), showing specific lung involvement related to CD (nodules, bronchial thickening and bronchiectasis). However, we notify a marginal but non-significant difference in bronchiectasis according to COPD ($p = 0.081$) and immunodeficiency ($p = 0.076$).

Tracheobronchial damage and symptoms experienced by patients (cough, crackles, etc...) are also described only in case reports in the literature[14–16]. No previous study analysed nodules specifically linked to CD. The range of pulmonary conditions reported in association with CD is analogous to IBD in which steroid sensitive bronchial inflammation, bronchiectasis and nodules are recognised complications[4]. Lung and bowel are derived from the same embryonic cell line and share a common epithelium with submucosal lymphoid tissue

which is a key component of host mucosal defence. The lung and gut may develop similar inflammatory reactions because of these shared anatomical features. A second possibility, the shared antigen theory, notes that gut and lung epithelia are exposed to the same antigens and this shared exposure may induce similar inflammation in both systems. Immune system activation may induce aberrant migration of the lymphocytes which invade the gut and other organs such as the lungs[4]. We also have reported six patients (16.2%) with fibro-retractile remodelling or apical caps suggestive of lung damage scar including three with known pneumonia history. One had pneumonitis in his story but without fibro-retractile remodelling or apical caps. Splenic atrophy (with asplenia and presence of Howel-Jolly bodies) and selective IgA deficiency are associated with coeliac disease and could predispose to development of recurrent infections[30, 31]. Our patients did not have selective IgA deficiency, but we did not know if they had splenic atrophy or asplenia. Impairment of nasal mucociliary clearance could play a role in the development of frequent lung infections in patients with CD. In another way, *Comba and colleagues* showed an impairment of nasal mucociliary clearance could play a role in the development of frequent lung infections in patients with CD, they showed that nasal mucociliary clearance was significantly prolonged in children with coeliac disease (531 ± 155 s vs 448 ± 80 s ; $p = 0,006$)[32].

On the other hand, emphysema is not significantly associated with CD but associated to COPD and tobacco exposure. Emphysema is more frequent in men, as it is reported in the literature[23], but it may be because they are significantly more smokers than women ($p < 0.001$).

Pleural lesions were found in seven (18.9%) patients (effusion, $n = 6$ and thickening, $n = 1$). It is the first study reporting pleural lesions related to CD, unlike IBD which present pleural manifestations recognized in the literature[4].

We found in our population five patients (13.5%) with COPD and four (10.8%) with asthma. These findings are correlated with others found in the literature[8, 9, 12, 13]. Contrary

to *Collin P and al.*[10] and *Douglas J. G. and al.*[11], we did not find any sarcoidosis in our population. We found no history of tuberculosis either. These findings need to be contrasted with those of *Ludvigsson et al.* who found more tuberculosis in patients with celiac disease ($p < 0.001$)[33].

Eight (21.6%) had pathological mediastinum lymph node. These findings could have a link with mesenteric lymph node cavitation which is an exceptional complication of celiac disease occurring exclusively in adults[34]. But additional studies are needed to confirm the hypothesis.

Finally, we reported four (10.8%) pericardial effusion (all minimal and none with cardiac symptoms or severe denutition). It is correlated to findings of *Riccabona and al.* who reported in a study that 50% of children with celiac disease had asymptomatic and limited pericardial effusion that only detectable with instrument, independently serum protein level[35]. In our knowledge, it is the first study showing pericardial effusion with a significative prevalence ($n = 4$, 10.8%) in a cohort of adult CD patients without severe denutrition ($BMI > 16 \text{ kg/m}^2$). Indeed, severe malnutrition can induce this anomaly[36].

Our study has some limitations. We examined only a small number of patients due to monocentric design and few celiac patients have ever had a chest HRCT scan in their story. The prognosis and severity of disease and degree of pulmonary dysfunction in CT period could not be assessed because of the lack of regular PFT monitoring or repeated low dose CT scan in all patients. Furthermore, PFT monitoring in our study were not performed at the same time of the chest HRCT scan. Respiratory symptoms are not always screened in CD patients due to the lack of data concerning respiratory manifestations of CD in the literature. Moreover, we did not consider all risk factors of lung damage. Indeed, many causes of immunodeficiency are missing, such as oral steroids, HIV status or systematically screening for hyposplenism or asplenia. In addition, only two patients had an $\alpha 1$ AT assay although a significant number had bronchiectasis and emphysema, which are lesions associated with $\alpha 1$ AT deficiency[37, 38]. Yet, an

association between α 1AT deficiency and CD has been reported in the literature[39], even if *Klasen and al* demonstrated the opposite later[40]. However, we tried to compensate for this missing data by analysing serum protein electrophoresis, which did not suggest an alpha 1 anti-trypsin deficiency in these patients. Finally, we have also many missed data regarding the HLA status and serological and histological status at CT, so it is difficult to define a profile of celiac patients more at risk of developing lung damage. Finally, our study does not have a control group (without CD).

In conclusion, CD is an independent risk factor of developing tracheobronchial damages (especially bronchial thickening and bronchiectasis) and nodules, independently tobacco exposure, COPD or pneumonitis history, and with no relationship with well-conducted gluten free diet. Small mediastinal lymph nodes and small pericardial effusion are note rare in adult CD adult patients. In adult patients with CD, a clinical screening of pulmonary symptoms could be interesting. This study suggests that a medical attention for dyspnoea, cough and pulmonary auscultation is necessary. PFT during the follow-up with spirometry, plethysmography and DLCO measurement may be required with chest HRCT if there is an anormal PFT and/or pulmonary symptoms. But further controlled and more powerful studies are needed.

CONCLUSION

En conclusion, la maladie cœliaque est un facteur de risque indépendant de développement de nodules et de lésions trachéobronchiques (en particulier les épaissements bronchiques et les bronchectasies), indépendamment du tabac, de la BPCO ou d'antécédent de pneumopathie. La présence de ces anomalies n'est pas modifiée par le bon suivi du régime sans gluten. Cette étude suggère qu'une attention médicale particulière sur la dyspnée, la toux et l'auscultation pulmonaire est nécessaire. La réalisation d'EFR au cours du suivi avec spirométrie, pléthysmographie et mesure de la DLCO semblerait pertinente chez les patients avec signes cliniques et/ou physiques, complétée d'une TDM thoracique *low dose* haute résolution en cas d'EFR anormale et/ou de symptômes pulmonaires. D'autres études à plus grande échelle sont cependant nécessaires pour confirmer ces résultats.

Clermont-Ferrand, le 29 avril 2022

Pr. Pierre CLAVELOU

Doyen – Directeur



Clermont-Ferrand, le 26 avril 2022

Pr. Marc RUIVARD

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Annexe 1.

Age at CD diagnosis	
Age at chest CT scanner	
Sex	Female Male
Tobacco exposure	No Yes
Dyspnoea	No Yes
Caugh	No Yes
Crackles	No Yes
Wheezing	No Yes
Snorted	No Yes
Inhaled bronchodilators	No Yes
Inhaled steroids	No Yes
Pulmonary history	None Asthma Tuberculosis Pneumonia COPD
TLC FVC DLCO FEV1/CVF FEV1	
Autoimmune disease	None Rheumatoid arthritis Lupus Scleroderma Primary Sjogren syndrome Horton Polymyalgia rheumatica Sarcoidosis Hashimoto Graves' IBD Vitiligo Psoriasis Spondylarthritis Type 1 diabetes MGUS Autoimmune neuropenia
Serological status at CD diagnosis	anti-TG IgAlG anti-DGP IgAlG anti-endomysium IgAlG anti-native gliadin
Serological status at chest CT	anti-TG IgAlG anti-DGP IgAlG anti-endomysium IgAlG anti-native gliadin
Seronegative	No Yes
IgA deficiency	No Yes
Silent	No Yes
Histology at CD diagnosis	Marsh 0 Marsh 1 Marsh 2 Marsh 3
Histology at chest CT	Marsh 0 Marsh 1 Marsh 2 Marsh 3
Refractory sprue	No Yes
HLA status	DQ2+/- DQ8+/-
Bowel lymphoma	No Yes
Well-conducted gluten free diet at chest CT scan	No Yes

Annexe 2.

Reason	Acute respiratory distress Chronic respiratory insufficiency Thoracic pain Screening Deterioration of general condition Neoplasia extension assessment Biological inflammatory syndrome Pre-interventional cardiac procedure
Technical	Without injection With injection in the pulmonary arterial phase With injection in the aortic arterial phase With Injection in the mixed arterial phase With portal phase injection Without and with injection in the pulmonary arterial phase without and with injection in the aortic arterial phase Without and with injection in the mixed arterial phase Without and with injection in the portal phase
Technical 2	Decubitus Procubitus Inhale Exhale
Kilovoltage	70KV 80KV 90KV 100KV 110KV 120KV multiKV (GSI)
Cutting thickness (mm)	

Annexe 3.

Exam quality	Optimal Sub-optimal Poor
Reason of poor quality	
<u>Parenchymal anomalies</u>	
Pattern	No UIP NSIP LIP COP RB-ILD DIP
Mosaic lung	Yes No
Cavity syndrome	No Cavity (>3cm) Cyst (<2cm) Pneumatocoles Blebs
Tracheobronchial anomalies	
Bronchiectasis	No Cylindrical Monoliform Cystic Traction
Bronchiolectasis	Yes No
Bronchial thickening	Yes No
Mucoid impacts	Yes No
Tracheal diverticula	Yes No
Emphysema	No Centrolobular Paraseptal Panlobular Bullous Advanced destructive
Left cardiac insufficiency	Yes No
Mass	Yes No
Nodules	No Calcified Tissular Intrapulmonary lymph node Hamartochondroma Mixed Pseudo-nodule
isolated micronodule	Yes No
Micronodular syndrome	Yes No
Respiratory disorders	No Subsegmental lamellar atelectasis Subsegmental lamellar atelectasis Interstitial abnormalities in contact with a vertebral osteophyte Passive collapse on pleural effusion Gravity-dependent Roll-up/round atelectasis
Fibro-retractile remodeling/apical caps	Yes No
<u>Pleural lesions</u>	No

	<ul style="list-style-type: none"> Pleural effusion Pleural thickening Pneumothorax
<u>Mediastinal lesions</u>	
Cardiac	<ul style="list-style-type: none"> Normal Cardiomegaly
Pulmonary arterial diameter	<ul style="list-style-type: none"> Normal <29mm Abnormal >29mm
Pulmonary embolism	<ul style="list-style-type: none"> Yes No Unsuitable exam
Thoracic aorta	<ul style="list-style-type: none"> Normal Abnormally high
Pericardial effusion	<ul style="list-style-type: none"> No Minimal Middle High
Pericardial leaflets	<ul style="list-style-type: none"> Thin Thick inflammatory Thick nodular Calcified
Coronary calcifications	<ul style="list-style-type: none"> Yes No
Mediastinal lymph area	<ul style="list-style-type: none"> Free A few infracentimetric lymph nodes A few ADM of lymphatic stasis or chronic pathology Calcified nodes Pathological ADM
Sus-clavicular/axillary areas	<ul style="list-style-type: none"> Free A few infracentimetric lymph nodes Calcified nodes Pathological ADM Reactional ADM
Thymus	<ul style="list-style-type: none"> Normal Mass Atrophy
Thyroid	<ul style="list-style-type: none"> Normal Normal volume-nodular Atrophic Goiter without nodule Nodular goiter Partial thyroidectomy stigmata Total thyroidectomy stigmata
Diaphragm	<ul style="list-style-type: none"> Normal Hernia Diaphragmatic rupture Diaphragmatic paralysis

(Conseil national de l'ordre des médecins)
SERMENT D'HIPPOCRATE

Au moment d'être admis(e) à exercer la médecine, je promets et je jure d'être fidèle aux lois de l'honneur et de la probité.

Mon premier souci sera de rétablir, de préserver ou de promouvoir la santé dans tous ses éléments, physiques et mentaux, individuels et sociaux.

Je respecterai toutes les personnes, leur autonomie et leur volonté, sans aucune discrimination selon leur état ou leurs convictions. J'interviendrai pour les protéger si elles sont affaiblies, vulnérables ou menacées dans leur intégrité ou leur dignité. Même sous la contrainte, je ne ferai pas usage de mes connaissances contre les lois de l'humanité.

J'informerai les patients des décisions envisagées, de leurs raisons et de leurs conséquences.

Je ne tromperai jamais leur confiance et n'exploiterai pas le pouvoir hérité des circonstances pour forcer les consciences.

Je donnerai mes soins à l'indigent et à quiconque me les demandera. Je ne me laisserai pas influencer par la soif du gain ou la recherche de la gloire.

Admis(e) dans l'intimité des personnes, je tairai les secrets qui me seront confiés. Reçu(e) à l'intérieur des maisons, je respecterai les secrets des foyers et ma conduite ne servira pas à corrompre les mœurs.

Je ferai tout pour soulager les souffrances. Je ne prolongerai pas abusivement les agonies. Je ne provoquerai jamais la mort délibérément.

Je préserverai l'indépendance nécessaire à l'accomplissement de ma mission. Je n'entreprendrai rien qui dépasse mes compétences. Je les entretiendrai et les perfectionnerai pour assurer au mieux les services qui me seront demandés.

J'apporterai mon aide à mes confrères ainsi qu'à leurs familles dans l'adversité.

Que les hommes et mes confrères m'accordent leur estime si je suis fidèle à mes promesses ; que je sois déshonoré(e) et méprisé(e) si j'y manque.

SERMENT D'HIPPOCRATE

En présence des Maîtres de cette FACULTE et de mes chers CONDISCIPLES, je promets et je jure d'être fidèle aux lois de l'Honneur et de la Probité dans l'exercice de la Médecine.

Je donnerai mes soins gratuits à l'indigent et je n'exigerai jamais un salaire au-dessus de mon travail. Admis dans l'intérieur des maisons, mes yeux ne verront pas ce qui s'y passe, ma langue taira les secrets qui me seront confiés et mon état ne servira pas à corrompre les mœurs ni à favoriser le crime.

Respectueux et reconnaissant envers mes MAÎTRES, je rendrai à leurs enfants l'instruction que j'ai reçue de leurs pères.

Que les HOMMES m'accordent leur estime si je suis fidèle à mes promesses. Que je sois couvert d'OPPROBRE et méprisé de mes confrères si j'y manque

MALADIE COELIAQUE et POUMON : une étude monocentrique et rétrospective

Résumé :

CONTEXTE : L'atteinte pulmonaire spécifiquement liée à la maladie cœliaque (MC), telles que les atteintes bronchiques et bronchiolaires (asthme, BPCO) ou la sarcoïdose, a été suggérée dans la littérature mais sans réelle étude descriptive bien menée pour mesurer l'association, à l'exception de l'hémosidérose pulmonaire dans la population pédiatrique. Aucune étude analysant l'atteinte pulmonaire à la tomodensitométrie (TDM) haute résolution n'a été menée dans une population de patients adultes atteints de MC.

OBJECTIF : Déterminer s'il existe un lien entre la maladie cœliaque et des atteintes pulmonaires spécifiques retrouvées au scanner.

METHODE : Étude rétrospective monocentrique menée chez des patients suivis pour une MC et ayant bénéficié d'une TDM thoracique au moment du diagnostic ou au cours du suivi, analysée par deux radiologues indépendants et en double aveugle.

RESULTATS : 242 patients ont été dépistés. 37 patients remplissaient les critères d'inclusion et avaient une TDM thoracique sans PID induite par médicament, PHS, pneumopathie radique ou pneumopathie infectieuse. Les trois atteintes les plus fréquemment retrouvées étaient les nodules (n = 26, 70.3%), puis l'emphysème (n=10, 27% ; dont 7 fumeurs), le syndrome bronchique (n=10, 27%), et les bronchectasies (n = 9, 24.3 %). Les nodules, le syndrome bronchique et les bronchectasies étaient indépendamment associés à la maladie, contrairement à l'emphysème qui était associé à la BPCO (p = 0.019), au tabac (p = 0,018) et au sexe masculin (p = 0 ;009). Aucun n'était lié au bon suivi du régime sans gluten. L'examen clinique au scanner et les symptômes étaient la toux (n = 12, 32.4 %), la dyspnée (n = 8, 21.6 %), les crépitants (n = 5, 13.5 %), les ronchi (n = 3, 8.1 %) et les sibilants (n = 2. 5,4 %). Les EFR ont été analysées dans 8 (21.6 %) cas, dont 5 avec un trouble ventilatoire obstructif (TVO), 2 avec un trouble ventilatoire restrictif (TVR) et 4 avec un trouble de diffusion.

CONCLUSION : La MC est un facteur de risque indépendant de développer des lésions trachéobronchiques et des nodules, indépendamment du tabac et du bon suivi du régime sans gluten. Chez les patients atteints de MC, un dépistage clinique des symptômes pulmonaires pourrait être intéressant et les patients pourraient bénéficier d'une EFR lors du suivi, complétée par une TDM thoracique s'il existe une EFR anormale et/ou des symptômes pulmonaires.

Mots-clés :

- Maladie cœliaque
- Poumon
- Pneumopathie interstitielle diffuse
- Bronchectasie
- Syndrome bronchique
- Nodule