Original Article: Lung Surgery

A case series of pneumothorax, pneumomediastinum and surgical emphysema in coronavirus disease 2019 (COVID-19)

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Background: The coronavirus disease 2019 (COVID-19) pandemic has been ongoing for nearly 18 months now and whilst randomized trials identify appropriate treatments, observational data increases knowledge around the real-life effects of COVID-19. Air leak in the context of acute lung injury is not a new phenomenon and usually associated with ventilation-induced lung injury. Air leaks (pneumothorax and pneumomediastinum) in the context of COVID-19 are being increasingly described. We sought to add to the literature by performing a local case review.

Methods: Northumbria Healthcare NHS Trust serves a population of approximately 600,000 in the North East of the United Kingdom. The records of all COVID-19 inpatients between March 2020 till January 2021 were analyzed. Local Caldicott approval was granted. Basic demographics and outcomes were collected. Descriptive statistical methodology was applied.

Results: Thirty-two air leaks were identified out of 2,827 inpatients, giving an incidence of 1.1%. The patients were predominantly male, elderly and with a higher-than-normal body mass index (BMI). There was no relation to previous respiratory disease and ventilation. Air leaks occurred late, signifying progressive lung injury. Fifteen deaths occurred in this specific cohort.

Conclusions: Air leaks are rare but carry significant morbidity and mortality. Knowledge of this evidence can thus enable patient centered decisions about prognostication. Analysis of large data sets will shed further light on this association.

Keywords: Coronavirus disease 2019 (COVID-19); pneumothorax; pneumomediastinum; surgical emphysema

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Introduction

Coronavirus disease 2019 (COVID-19) is a multi-system disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Since the World Health Organization declared a pandemic in early 2020, there have been cases nearly 126 million cases globally and 3 million deaths at the time of writing (1). Typical radiological findings include bilateral, multi-lobar, posterior, peripheral, and basal ground-glass opacity (GGO) with or without consolidation (2). Atypical features are often pleuro-parenchymal or mediastinal associated with air leaks (pneumothorax and/or pneumomediastinum and/or surgical emphysema) (2). The largest case series by Martinelli *et al.* showed the development of pneumothorax in approximately 1% of

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COVID-19 inpatients. Mechanical ventilation and previous lung disease were not risk factors and the authors cautioned against nihilism (3). Mortality is not seemingly ascribed to air leaks (3). Observational case series through rigorous searches of health data sets can capture the true incidence of, and inform local as well as global practice. We thus sought to add to the existing literature by performing a local case review. We present the following article in accordance with the STROBE reporting checklist (available at https://asj.amegroups.com/article/view/10.21037/asj-21-14/rc).

Methods

Materials and methods

All COVID-19 inpatients in Northumbria Healthcare NHS Foundation Trust in the North East of England from 1st March 2020 until 31st of January 2021 were identified. All chest X-rays (CXR) and chest computed tomography (CT) reports were searched for "surgical emphysema" and "pneumothorax" and "pneumomediastinum". Positive reports were identified and independently verified. Demographics and outcomes were collected. Informed consent was not required due to the nature of this anonymized retrospective analysis and nationwide provisions for the use of confidential patient information without consent for COVID-19 purposes. Caldicott approval was granted from Northumbria HealthCare NHS Foundation Trust (RPI-1279). The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013).

Statistical analysis

Basic statistical methodology was applied. Continuous variables are presented as mean (± range) and categorical variables as percentages where appropriate.

Results

During the defined period which spanned first and second waves, out of 2,827 inpatients with COVID-19, 32 (1.1%) patients with air leaks were identified. Mean age was 63 years (range, 29–91 years); 27 (84%) were male; all were white Caucasian except one (East Asian). Seventeen were ex-smokers, 12 never smokers and 2 were current smokers. There was no documented marijuana smoking. Comorbidities were treated chronic obstructive pulmonary disease (COPD) (4), asthma (5) and hypertension (5). Most patients did not have pre-existing documented lung disease, although 13 (41%) were smokers. None of those patients had enough accumulated pack years to develop COPD. One patient had a previous pneumothorax. Median clinical frailty score (CFS) was 2 (range, 1–6), mean body mass index (BMI) was 27.6 kg/m^2 (range, 18.5–46.7 kg/m^2), mean height 1.7 m (range, 1.55–1.9 m). Mean number of days to development of air leak was 13 (range, 1–120). Fifteen patients have died. The inspired fraction of oxygen (FiO_2) at the time of air leak development was 0.21 (air) in 7 patients, 0.24 in 1, 0.35 in 2, 0.4 in 1 and 0.9 (15 liters via non-rebreathe bag) in 3. Seventeen (53%) patients were on continuous positive airways pressure (CPAP) or mechanical ventilation. Ten patients were on CPAP with positive end expiratory pressures (PEEP) between 10 and 12 centimeters (cm) of water. Seven patients were on mechanical ventilation (one via a tracheostomy) on PEEPs between 12 and 16 cm and FiO_2 ranging from 0.75 to 1. The annexed supplementary material summarizes the air leaks and respective outcomes (Table S1). *Table 1* below summarizes the outcomes according to the type of air leaks

Two deaths were directly attributable to a pneumothorax. This is explained in greater detail below. All the pneumothoraces that were large (n=3), according to British Thoracic Society criteria (4), had intercostal drainage. All other pneumothoraces were small.

Discussion

COVID-19 causes diffuse alveolar damage (6). Rupture of any alveolar sacs combined with pleural porosity will lead to pneumothorax. Subsequent air leaks can penetrate into the mediastinum and skin via the Macklin effect, causing pneumomediastinum and surgical emphysema. The Macklin effect, described in 1939, is the movement of air along the sheaths of the pulmonary vasculature from the alveoli into the mediastinum (7). "Lung frailty" and "architectural disruption" seems to be the etiological factor rather than ventilation associated lung injury (5,6,8). The majority of patients were male, had a higher-than-normal BMI and were White Caucasian, reflecting local demographics in the North East of England.

Two deaths were directly attributable to a pneumothorax. One patient was elderly, frail with a CFS of 6, early dementia, an acute cerebrovascular incident. He acquired COVID-19 as an inpatient and developed subsequent respiratory failure. He then developed a large pneumothorax

Table 1 Summary of patient outcomes according to type of air leak

Variables	Isolated pneumothorax	Isolated pneumomediastinum	Pneumomediastinum and surgical emphysema	Pneumomediastinum and surgical emphysema and pneumothorax	Pneumothorax and surgical emphysema	Pneumothorax and pneumomediastinum
Total number	11	7	5	6	1	2
Deaths	3	0	5	5	1	1
Total number on CPAP at the time of leak	2	3	3	2	0	0
Total number on mechanical ventilation at the time of leak	0	0	2	2	1	1
Total amount of oxygen only at the time of leak	9	4	0	2	0	1

CPAP, continuous positive airways pressure.

with significant distress and agitation and was thus palliated. Another patient had a CFS of 6 and end-stage COPD who presented with COVID-19 and a small pneumothorax. He was in significant respiratory failure and was palliated appropriately. Another patient developed a pneumothorax whilst receiving CPAP (ceiling of treatment established on admission) and was successfully treated with a small-bore chest drain, but developed progressive respiratory failure and was palliated. Martinelli *et al.* caution against nihilism in the treatment of pneumothorax (3), and our findings would replicate that. A timely, holistic and overall assessment of the patient is advised.

All patients who developed an isolated pneumomediastinum (n=7) were managed conservatively and are still alive. All were discovered as an incidental finding on CT scans or CXRs. As such, it seems that an isolated pneumomediastinum is not an adverse prognostic development.

Of the 6 patients who developed a combination of pneumomediastinum, surgical emphysema and pneumothorax (unilateral or bilateral), only 1 survived. All had a number of small (12 Fg) and large bore (24 Fg) drains placed either intrapleurally or subcutaneously, and 3 patients with massive surgical emphysema also had subcutaneous incisions made and vacuum dressings placed over. The surviving patient was a 46-year-old who presented with bilateral pneumothorax, surgical emphysema and pneumomediastinum. He was maintained on FiO_2 of 0.4 until acute worsening prompted mechanical ventilation. A 24-Fg drain was inserted which improved the surgical emphysema but respiratory failure worsened significantly in the next 24 hours. He was referred and accepted for extracorporeal membrane oxygenation (ECMO) at a national tertiary center, and survived to discharge after a prolonged inpatient stay. The foregoing perhaps signals that the development of pneumothorax, surgical emphysema and pneumomediastinum is an adverse prognostic sign, unless "lung rest" via ECMO is possible. It should be noted that all other patients were referred for ECMO, but declined.

The only survivor out of the two patients who developed pneumothorax and pneumomediastinum was a 79-year-old patient whose PCR test was positive was 21 days prior to an acute presentation with chest pain. A CT thorax showed a pulmonary embolus, a small pneumothorax and pneumomediastinum. He was anticoagulated and oxygen saturations remained above 94% on air. He was discharged with no intervention required for his air leak. The other patient who died was on mechanical ventilation.

This is a single centre retrospective study with no control

group, and thus significant limitations exist. For example, 4 patients with COPD, 7 with asthma and 17 ex-smokers having air leaks might sound significant in a cohort of 32, but we cannot infer substantial associations from this.

Conclusions

Air leaks in the context of COVID-19 are rare. Isolated pneumothorax and pneumomediastinum are not adverse prognostic signs, but the development of pneumothorax, surgical emphysema and pneumomediastinum on mechanical or non-invasive ventilation might be. Large data sets must be analyzed to confirm these findings.

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Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://asj.amegroups.com/article/view/10.21037/asj-21-14/rc

Data Sharing Statement: Available at https://asj.amegroups.com/article/view/10.21037/asj-21-14/dss

Conflicts of Interest: Both authors have completed the ICMJE uniform disclosure form (available at https://asj.amegroups.com/article/view/10.21037/asj-21-14/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). Informed consent was not required due to the nature of this retrospective analysis and nationwide provisions for the use of confidential patient information without consent for COVID-19 purposes. Caldicott approval was granted from Northumbria HealthCare NHS Foundation Tru (RPI-1279).

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Supplementary

Tables S1 Full data table of all patients with air leaks, describing demographics, presentation and outcomes

Age, years	Sex	Ethnicity	Smoking	Co-morbid	CFS	BMI	Ht	Ptx/PMN	Symptoms	Ward/ICU	Oxygenation (FiO_2) at the time of Ptx	Treatment	Outcome	Days from admission to Ptx, pnm	Mode of diagnosis
91	M	White	Ex	Stroke, IHD	6	25.7	1.61	Right Ptx	Cough, fevers, dyspnoea	Ward	0.9	Con	Death	12	Change in condition
75	M	White	Ex	AAA repair	1	25	1.55	Left Ptx	Cough fever	Ward	0.24	Con	Alive	12	Incidental
65	M	White	Ex	COPD, CCF	2	29.8	1.72	Right Ptx	Lethargy, loss of appetite	Ward	CPAP, 0.6	12 Fr	Alive	13	Change in condition
85	M	White	Ex	COPD, AF, BP	4	18.5	1.8	Left Ptx	Cough, fever	Ward	0.9	Con	Death	22	Change in condition
67	M	White	Current	Previous stroke, type 2 DM, emphysema and combined pulmonary fibrosis, previous ptx and pleurodesis	6	23	1.65	Right Ptx	Dyspnoea	Ward	0.28	Con	Alive	1	Seen on admission X-ray
85	M	White	Never	IHD	4	23.2	1.74	Left Ptx	Dyspnoea, pain	Ward	0.21	Con	Alive	1	Admission CTPA
54	M	White	Ex	–	2	–	–	Right Ptx	Cough, pain, haemoptysis	Ward	0.21	Con	Alive	1	Admission CTPA
56	M	White	Never	–	2	22.5	1.82	Left Ptx	Cough	Ward	0.35	12, 20, 24 Fr	Alive	29	Change in condition
54	M	White	Never	–	0	–	–	Right Ptx	Chest pain, cough	Ward	0.21	Con	Alive	120	X-ray
82	M	White	Never	Asthma, hypertension	1	24.3	1.75	Right Ptx	Cough, fever, confusion, SOB	Ward	CPAP, 0.6	12 Fr	Dead	12	Change in condition
32	M	White	Never	–	1	–	–	Right Ptx	Cough, fever	A and E	0.21	Con	Alive	1	Admission Cxr
50	M	White	Never	Asthma	1	24.4	1.9	PMN	Dyspnoea	A and E	0.21	con	Alive	1	Found on CTPA
57	F	White	Never	Asthma	1	23.2	1.7	PMN	Dyspnoea	Ward	0.28	con	Alive	5	Found on CTPA
61	F	White	Ex	–	1	32.7	1.67	PMN	Dyspnoea, fever	ICU	CPAP, 0.65	con	Alive	10	Found on CTPA
49	M	White	Ex	ALD, asthma	2			PMN	Dyspnoea, fever	ICU	CPAP, 0.5	con	–	5	Incidental on cxr
63	M	White	Ex	COPD, asthma	1	27	1.84	PMN	Cough, fevers, dyspnoea	ICU	CPAP, 0.3	Con	Alive	29	Incidental
50	M	White	Never	Asthma	1	26.3	1.9	PMN	Dyspnoea	Ward	0.4	con	Alive	5	Clinical change
76	M	White	Ex	CKD, RA, T2DM, liver fibrosis, UC	2	25.6	1.78	PMN	Fever, dry cough	Ward	0.9	Con	Alive	16	Inpatient CTPA-incidental
61	M	White	Never	–	1	34.1	1.72	PMN and SE	Cough, dyspnoea	Ward	CPAP, 0.9	con	Dead	10	Clinical change
76	F	White	Never	Asthma, CKD, BP, OSA	4	36.2	1.62	PMN and SE	Dyspnoea, cough	Ward	CPAP, 1	con	Dead	4	–
49	M	White	Never	IHD, type 2 DM	2	34.5	1.7	PMN and SE	Chest pain and dyspnoea	ICU	MV	con	Dead	12	Clinical change
74	M	White	Never	BP, diabetes	2	33	1.8	PMN and SE	Dyspnoea, dry cough	ICU	MV	Con	Dead	10	Change in condition
80	M	White	Never	Type 2 DM, CKD	2	26.1	1.59	PMN and SE	Fell down stairs, fever	Ward	CPAP, 0.55	Con	Dead	13	Change in condition
68	M	White	Never	–	2	22.8	1.82	Left Ptx and PMN	Dry cough	ICU	MV	12 Fr	Dead	5	Change in condition
79	M	White	Current	–	2	20.9	1.7	Left Ptx and PMN	Pain	Ward	0.21	Con	Alive	21	Found on CTPA
63	M	White	Ex	Type 2 DM	1	31.1	1.7	Right Ptx and SE	Dyspnoea	ICU	MV	Con	Dead	8	Found when intubated
57	M	White	–	–	1			Left Ptx, PMN and SE	Cough	Ward	CPAP, 0.75	24 Fr, SC and 12 F	Dead	6	Clinical change
76	M	White	Ex	Lung cancer	2	24.8	1.58	Right Ptx, PMN and SE	Facial swelling	Ward	0.21	Con	Dead	1	Seen on admission X-ray
51	M	White	Ex	Aortic stenosis, BP	1	–	–	PMN, SE and bilateral Ptx	Dyspnoea, fever	Ward	Cpap, 0.5	2 SC drains	Dead	5	Clinical change
29	F	White	–	Obesity, pregnant	1	46.7	1.72	PMN, SE and bilateral Ptx	Fever, dyspnoea	ICU	MV	2 24Fr, 2 SC	Dead	3	Cxr on icu post intubation
57	F	White	–	–	2	34.2	1.64	PMN, SE and bilateral Ptx	Cough, chest pain	ICU	MV	12 F tried, 24 F, then 12F	Dead	24	Clinical change
46	M	Asian	Ex	Previous pleural TB	1	23.3	1.66	PMN, SE and bilateral Ptx	Cough	Ward then ICU-required ECMO	0.35	24 Fr, sub cut	Alive	1	Seen on admission X-ray

IHD, ischaemic heart disease; CCF, congestive cardiac failure; COPD, chronic obstructive pulmonary disease; AAA, abdominal aortic aneurysm; AF, atrial fibrillation; BP, blood pressure; DM, diabetes mellitus; Ptx, pneumothorax; PMN, pneumomediastinum; SE, surgical emphysema; ICU, intensive care unit; CKD, chronic kidney disease; RA, rheumatoid arthritis; UC, ulcerative colitis; CTPA, computed tomogram pulmonary angiogram; CXR, chest X-ray; TB, tuberculosis; MV, mechanical ventilation; CPAP, continuous positive airways pressure; CKD, chronic kidney disease; Fr, French gauge; SC, subcutaneous; ECMO, extra-corporal membranous oxygenation.

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