SARCOIDOSIS VASCULITIS AND DIFFUSE LUNG 2024; 41 (1): e2024004 DOI: 10.36141/svdld.v41i1.15027

Incidence, management and prognosis of new-onset sarcoidosis post covid-19 infection

Oliver Vij[1], Mrinalini Dey[2,3], Kirsty Morrison[4], Koushan Kouranloo[5,6]

[1]Guy's Hospital, London, UK; [2]Institute of Life Course and Medical Sciences, University of Liverpool, Liverpool, UK; [3]Centre for Rheumatic Diseases, King's College London, London, UK; [4]BMA House -British Medical Association, London, UK; [5]School of Medicine, University of Liverpool, Merseyside, UK; [6]Department of Rheumatology, East Surrey Hospital, Redhill, UK

Abstract. *Background and aim:* SARS-CoV-2 infection has been linked to hyperinflammation in multiple organs with a potential mechanistic link with resulting autoimmunity. There have been reports of many inflammatory complications following COVID-19, including sarcoidosis. A literature review on new-onset sarcoidosis following COVID-19 is lacking. We evaluated potential associations between COVID-19 and development of new-onset sarcoidosis. *Methods:* Articles discussing biopsy-proven sarcoidosis after confirmed COVID-19 infection, published 1956 until April 2023, were included. All article types were deemed eligible except opinion and review articles. *Results:* A pooled total of 15 patients with new-onset diagnosis of sarcoidosis after COVID-19 infection were included, 45.5% female, mean age 46.1 years (standard deviation 14.7) at onset of sarcoidosis. Patients were from: Europe (n=11); North America (n=2); South America (n=1); Asia (n=1). The mean time between COVID-19 infection and diagnosis of sarcoidosis was 56.3 days, although this ranged from 10 to 140 days. Organ systems predominantly affected by sarcoidosis were: pulmonary (n=11); cutaneous (n=3); cardiac (n=2); ocular (n=1); systemic (n=1) (with overlapping features in certain patients). Sarcoidosis was treated as follows: glucocorticoids (n=8); azathioprine (n=1); cardiac re-synchronisation therapy (n=1); heart transplant (n=1). All patients were reported to have survived, with one requiring intensive care admission. *Conclusions*: Our result suggests there is a potential link between COVID-19 and new-onset sarcoidosis. The potential mechanism for this is through cytokine mediated immune modulation in COVID-19 infection. Obtaining a tissue sample remains key in confirming the diagnosis of sarcoidosis and this may be delayed during active COVID-19 infection.

Key words: COVID-19, SARS-CoV-2, autoimmune disease, sarcoidosis

Introduction

The COVID-19 pandemic resulted in significant morbidity and mortality (1). Although it is primarily a respiratory disease, there are various extrapulmonary manifestation of COVID-19 now reported (2). It is well recognised that SARS-Cov-2 infection is linked to hyperinflammation and there is a potential mechanistic link to autoimmunity (3-5).

There have been reports of many inflammatory complications following COVID-19 infection and it has been linked with new-onset autoimmune connective tissue disease (6,7).

Theoretical links have been made between COVID-19 and sarcoidosis pathophysiology, suggesting they share common cellular pathways involved in immune response and cell death (8,9). Indeed, there have been several reports of new-onset sarcoidosis following infection with SARS-Cov-2. However, a systematic review of the literature is lacking.

Received: 19 July 2023
Accepted: 25 October 2023
Correspondence:
Dr Koushan Kouranloo, MBchB, MRCP (London), FHEA
E-mail: k.kouranloo2@liverpool.ac.uk

We evaluated the potential association between COVID-19 and the development of new-onset sarcoidosis. Our objectives were to investigate the incidence, management and prognosis of new-onset sarcoidosis post COVID-19 infection.

Methods

This systematic review was undertaken in accordance with the Cochrane Handbook and reported as per the Preferred Reporting Items for Systematic Review and Meta-Analyses (10,11).

The protocol was developed and registered in the PROSPERO database of systematic reviews (CRD42023430311)(12). The review question was: What is the incidence and management of new-onset sarcoidosis after COVID-19 infection?

Population

We included adults with biopsy-proven sarcoidosis with the "intervention" as COVD-19 and related terms. We excluded patients that developed new-onset sarcoidosis without prior SARS-Cov-2 infection or patients who had flares of existing sarcoidosis following SARS-Cov-2 infection.

Outcome

Outcomes were basic demographics, clinical and investigation findings and treatment administered following new-onset sarcoidosis after SARS-CoV-2 infection.

Search strategy, databases and study selection

To ensure comprehensive coverage, indexing terms (MeSH, applicable to Medline and Cochrane, and Emtree headings on Embase) as well as keyword searching were used.

Medline, Embase and Cochrane databases were searched for articles discussing biopsy-proven sarcoidosis after confirmed SARS-Cov-2 infection. Medline from 1946, Embase from 1974, Cochrane CDSR from 1995, and Cochrane CENTRAL from inception in 1996. Cochrane CENTRAL first began publication in 1996, but its composite nature means that it does not have an inception (start) date, in the way that other traditional biomedical databases do (13).

The search was restricted to English-language articles and those discussing clinical presentation of disease. Eligible articles included: case reports and series, observational studies, qualitative studies and randomised control trials. Patients with flares of existing sarcoidosis following COVID-19 infection were excluded. Patients with likely sarcoidosis following COVID-19 infection but no histological tissue diagnosis confirming sarcoidosis were also excluded.

Full-length articles were uploaded into Rayyan (www.Rayyan.ai) with duplicates removed. Articles meeting inclusion criteria were examined by two authors at abstract and full paper stage. In addition to basic demographics, information was extracted on clinical investigation findings and treatments administered.

Results

Initially, 296 articles were retrieved with 10 ultimately included (Figure 1). 8 case reports, 1 case series and 1 cohort study were included. The cohort study was assessed for risk of bias using the Newcastle-Ottawa quality assessment (Table 1).

A pooled total of 15 patients with new-onset diagnosis of sarcoidosis after COVID-19 infection were included. 45.5% were female with a mean age of 46.1 years (SD 14.7) at onset of sarcoidosis. Patients were from: Europe (n=11); North America (n=2); South America (n=1); Asia (n=1).

The mean time between COVID-19 infection and diagnosis of sarcoidosis was 56.3 days, although this ranged from 10 to 140 days. Organ systems predominantly affected by sarcoidosis were: pulmonary (n=11); cutaneous (n=3); cardiac (n=2); ocular (n=1); systemic (n=1) (with overlapping features in certain patients). The most commonly reported co-morbidities were hypertension, chronic obstructive respiratory disease and ischemic heart disease and 6 patients were reported to have no co-morbidities. None of the patients were reported to have any pre-existing autoimmune conditions.

All patients underwent tissue biopsy demonstrating features consistent with sarcoidosis: lung (n=11); skin (n=2); cardiac (n=2). Computed tomography (CT) of the chest (n=13), positron emission tomography (PET-CT) (n=2) and cardiac magnetic resonance imaging (n=1) were undertaken. PET-CT was undertaken in a case of pulmonary sarcoidosis and in a case of combined cardiac and cutaneous sarcoidosis.

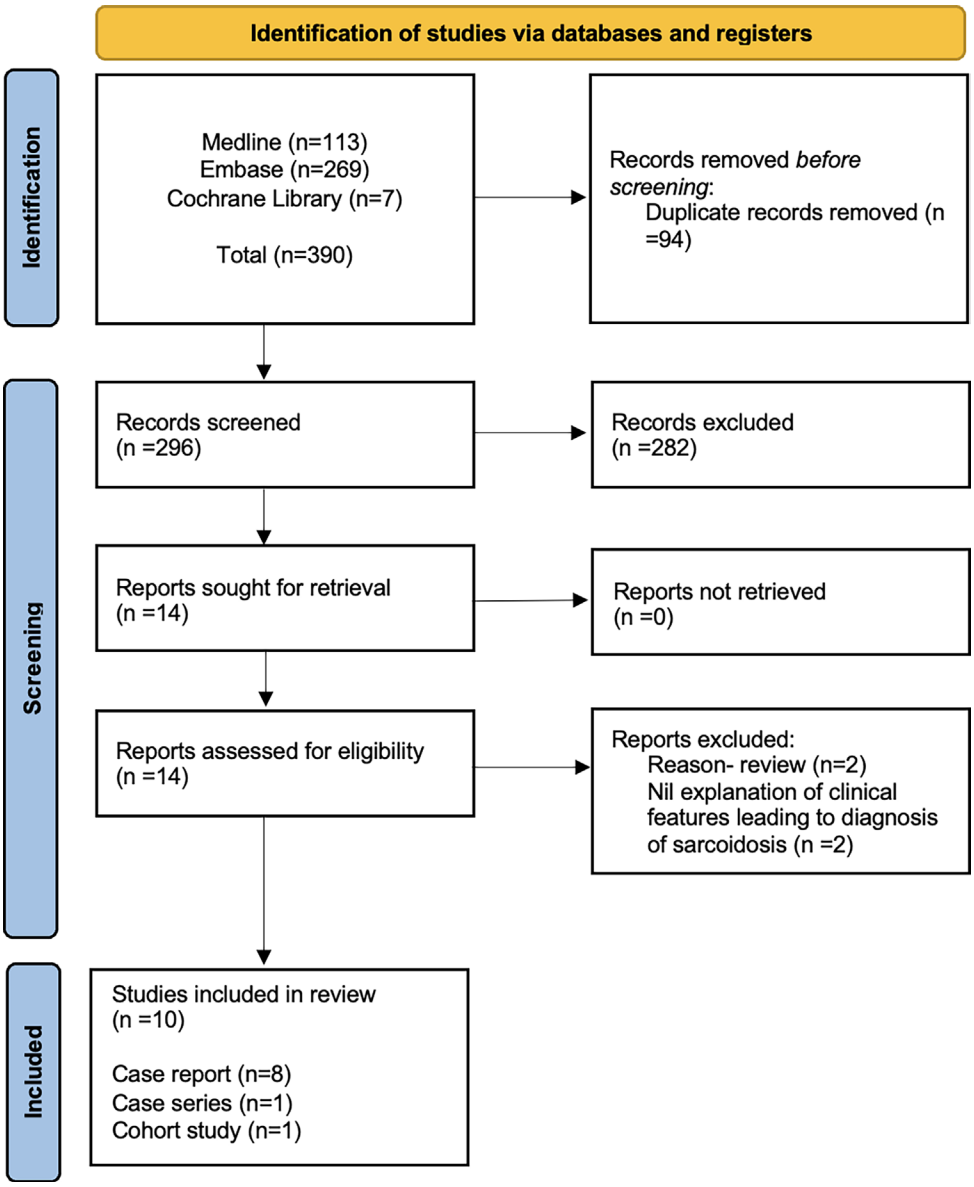

Figure 1. Cochrane Library encompasses library of: systematic reviews; systematic review protocols; controlled clinical trials.

Table 1. Newcastle-Ottawa Quality Assessment for cohort studies.

Study	Selection				Comparability	Outcome			Total (9*)
	Representativeness of exposed cohort (*)	Selection of non-exposed cohort (*)	Ascertainment of exposure (*)	Outcome of interest not present at start of study (*)	(**)	Assessment of outcome (*)	Length of follow-up (*)	Adequacy of follow up (*)	
Jakubec et al. 2022 [14]	*	*	*	-	-	*	*	-	5*

Cardiac magnetic resonance imaging (MRI) was used in investigation of a patient with cardiac sarcoidosis. There was no further mention of exclusion of other differential diagnoses of sarcoidosis in five studies (14-18). Four studies directly mentioned excluding tuberculosis as a differential (19-22).

Sarcoidosis was treated as follows: glucocorticoids (n=8); azathioprine (n=1); cardiac resynchronisation therapy (n=1); heart transplant (n=1). Cardiac resynchronisation therapy and heart transplant were used as treatments for the 2 patients with cardiac sarcoidosis. Azathioprine was used in combination with oral prednisolone in a case of pulmonary and cutaneous sarcoidosis. All patients were reported to have survived, with one requiring intensive care admission. 4 patients reported that their symptoms had greatly improved or resolved following treatment of sarcoidosis, with follow-up of the other patients included in this systematic review not mentioned. These results are summarised in Table 2.

Discussion

This systematic review summarises data on new-onset sarcoidosis following COVID-19 infection. Our findings from the 10 included studies suggest a potential link between COVID-19 infection and new-onset sarcoidosis, although this is not definitive.

Sarcoidosis is an inflammatory disease characterised by granuloma formation affecting many organs but predominantly the lungs (23). The exact pathophysiology of sarcoidosis is yet to be determined, but it is thought to be a combination of genetic, immunological and environmental factors (24).

Despite this unclear mechanism, there have been theoretical links made between the pathogenesis of COVID-19 and sarcoidosis. It has been shown that both diseases share five gene hubs that are both upregulated in disease states (25). Additionally, both diseases share mechanisms that disrupt the renin-angiotensin system (RAS), immune responses and cell death pathways including autophagy (8,9). It is this disruption of the immune response that most of the literature is focused around and thought to underpin the mechanism of COVID-19 induced new-onset sarcoidosis.

The mechanism for a close temporal association between COVID-19 and sarcoidosis is, at present, unclear. Many of the studies we reviewed suggested that COVID-19 was dysregulating the inflammatory response in some way (14,17,20,21). Capaccione et al further suggested this is via cytokine-mediated immune stimulation (22). There is growing evidence to suggest that TH17.1 cells producing high amounts of the TH-17 related cytokines interleukin-17 (IL-17), IL-22 and interferon-γ (IFN-γ) are involved in the pathogenesis of acute sarcoidosis in response to infectious or environmental antigens that could include viral infection (24, 26–28). Interestingly, among the many cytokines shown to be present in the inflammatory response to COVID-19 infection, IL-17 and IFN-γ have been implicated through the upregulation of TH2 and TH1/TH17 cells with a skew towards TH17 (29,30). This mutual cytokine profile between COIVD-19 infection and acute sarcoidosis provides a potential mechanism through which COVID-19 could predispose to sarcoidosis.

Moreover, in silico analysis of SARS-Cov-2 binding proteins has suggested that there are proteins members that contribute to COVID-19 pathogenesis via complement and coagulation cascades that could promote sarcoidosis. This is thought to be via a SARS-Cov-2 specific gene known as open reading frame 8 (ORF8) which upregulated proteins including IL-17 receptor A, growth differentiation factor 15, FK506-binding protein 10 and tissue-type plasminogen activator (PLAT) that are also associated with sarcoidosis pathogenesis (31).

There have been other documented temporal associations between infections and sarcoidosis, specifically *Propionibacterium acnes* (32). Some studies have implicated P. acnes in the pathogenesis of sarcoidosis (33–38). Others suggest that it is not specific for sarcoidosis as it is a normal finding in peripheral lung tissue (39).

A shared pathogenesis between COVID-19 and sarcoidosis is further reinforced as *Propionibacterium acnes* has been shown to stimulate the development of granulomas in murine studies with increasing concentration of cytokine-producing CD4+ cells that generate IFN-γ (40). It has been suggested that P. acnes causes an increased Th1 response in sarcoidosis patients, a response that has also been shown in COVID-19 patients (30, 37). This indicates it is possible for infections to cause granulomas via a similar mechanism of cytokine-mediated immune stimulation. Although this suggests that infectious organisms can cause granulomatous infection, this does not necessarily mean they cause sarcoidosis itself.

Histological evidence of non-caseating granulomas is essential in confirming sarcoidosis (41).

Table 2. Summary of included studies with basic demographics, type of sarcoidosis and summary of treatment and outcomes.

Title	Organ	Time between onset of COVID-19 and sarcoidosis	Relevant blood tests	Treatment of COVID-19	Exclusion of other differentials	Treatment of sarcoidosis	Pre-existing autoimmune condition	Co-morbidities	ITU admission	Outcome
Racil et al. 2023 [19]	Pulmonary	A few weeks	Raised serum ACE	Low-flow oxygen therapy and corticosteroid therapy	Sputum smear and culture ruled out tuberculosis. Histology confirmed sarcoidosis.	Oral prednisolone	None	Unknown	No	Survived
La Placa et al. 2023 [15]	Cutaneous	Not mentioned	Unknown	Unknown	Histology confirmed sarcoidosis.	Unknown	None	Hypertension, hysterectomy for endometrial cancer 7 years previously.	No	Survived
Somboonviboon et al. 2022 [20]	Pulmonary; ocular	10 weeks; 70 days	Serum ACE 20.3 U/L, normal serum calcium	Hydration, cough suppressants and antipyretics	Fungal infection and tuberculosis excluded from both bronchoalveolar lavage (BAL) and lymph node aspiration cultures. Histology confirmed sarcoidosis.	60mg prednisolone daily tapered to 20mg over 1 month and maintained at this dose	None	None	No	Survived
Rodrigues et al. 2022 [21]	Cutaneous; pulmonary	20 days	Unknown	Unknown	Tuberculin test negative. Histology confirmed sarcoidosis.	60mg prednisolone plus 100mg azathioprine OD	None	None	No	Survived
Palones et al. 2022 [16]	Cutaneous	14 days	Serum ACE 57 nmol/ml/min	Unknown	Histology confirmed sarcoidosis.	Inhaled corticosteroids for 1 month	None	None	No	Survived
Jakubec et al. 2022 [14]	Pulmonary (6)	2-6 months; 112 days	Unknown	Oxygen therapy required in 51/98 patients. 1 patient required NIV. 5 patients died.	Histology confirmed sarcoidosis.	Unknown	Unknown	Common amongst entire sample with the most frequent being arterial HTN followed by chronic respiratory disease, diabetes mellitus and cardiovascular diseases.	Unknown	Survived
Capaccione et al. 2022 [22]	Pulmonary	5 months; 140 days	Serum ACE207 U/L, ESR 25 mm/h, calcium 10.6 mg/dL	Hydroxychloroquine and azithromycin. ICU admission with renal replacement therapy. Tracheostomy 3 weeks after ICU admission.	Cultures for fungi, norcardia, actinomyces and acid-fast bacilli negative. Histology confirmed sarcoidosis	20mg oral prednisolone tapered to 10mg OD	None	Prostate cancer, CKD	Yes	Survived

(*Continued*)

Table 2. Summary of included studies with basic demographics, type of sarcoidosis and summary of treatment and outcomes. (*Continued*)

Title	Organ	Time between onset of COVID-19 and sarcoidosis	Relevant blood tests	Treatment of COVID-19	Exclusion of other differentials	Treatment of sarcoidosis	Pre-existing autoimmune condition	Co-morbidities	ITU admission	Outcome
Bollano et al. 2022 [17]	Cardiac; systemic	1 month; 28 days	Unknown	Unknown	Histology confirmed sarcoidosis.	Pulse-dose steroids for 3 days followed by oral prednisolone tapered down over a year, remaining on 10mg oral prednisolone OD. Cardiac resynchronisation defibrillator was implanted.	None	None	No	Survived
Alonso et al. 2022 [18]	Cardiac	Unclear	Unknown	Unknown	Histology confirmed sarcoidosis.	Immunosuppressive therapy (patient had heart transplant).	None	None	No	Survived
Mihalov et al. 2021 [49]	Pulmonary	Close temporal association	Serum ACE1.27 ukat/L, thymidine kinase 30.6 units/L, complement C3 1.95 g/L	Unknown	Serology for infective causes negative. Blood and urine cultures unremarkable. Histology confirmed sarcoidosis.	20mg prednisolone OD	None	None	No	Survived

This is particularly important in sarcoidosis following COVID-19 infection, as both primarily affect the respiratory system with overlapping clinical and radiological features (42–44). Furthermore, many countries and communities introduced mitigation strategies such as social isolation and quarantine to control COVID-19 infection rates (45). These overlapping features causing diagnostic uncertainty coupled with isolation and quarantine measures in hospitals could have caused delay in confirming sarcoidosis with tissue biopsy potentially delaying appropriate management. This could explain why the time between COVID-19 infection and sarcoidosis diagnosis ranged from 10 to 140 days.

It is worth noting that cases of COVID-19 induced sarcoidosis may be under-reported as sarcoidosis is commonly asymptomatic and most individuals who recovered from COVID-19 infections would not routinely undergo post-infection imaging during the pandemic (46).

Strengths and limitations

This systematic review demonstrates a potential link between the two conditions and posits a potential mechanism for COVID-19 induced sarcoidosis. However, our study included a small number of cases due to the specific condition studied. Therefore, it is important not to infer causality just from these cases, despite the close temporal association.

The possible associated between COVID-19 and sarcoidosis is not certainly not conclusive. COVID-19 infected a large proportion of the population and sarcoidosis could have arisen spontaneously in these patients (47). The diagnosis of sarcoidosis can be complicated, and all the studies included had a tissue diagnosis. Despite this, many infections can cause a granulomatous response that can mimic sarcoidosis (48). It is possible that these cases of sarcoidosis could have been spontaneous, given the large numbers of COVID-19 infections, and the rates of sarcoidosis before and during the COVID-19 pandemic were not evaluated in any of the included studies. More detailed longitudinal studies, on larger cohorts, are required to establish causation.

Conclusion

In conclusion, this is the first systematic literature to examine the incidence, management and prognosis of new-onset tissue-proven sarcoidosis after COVID-19 infection. Our review shows that there is a potential link between COVID-19 and new-onset sarcoidosis. There appears to be a shared cytokine profile between COVID-19 infection and acute sarcoidosis pathogenesis, most commonly with IL-17 and IFN-γ (26–29,31,40). Our results suggest that COVID-19 and other viral infections could lead to immune dysregulation and onset of autoimmune disease.

This association could help to provide further insight into the underlying mechanisms of sarcoidosis. Further studies into their shared pathophysiology may help to guide management of both COVID-19 and sarcoidosis and to better risk stratify patients susceptible to infections.

This manuscript does not contain any studies with human or animal subjects.

All data is available upon request.

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

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