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Acute idiopathic pericarditis during a national lockdown to prevent transmission of SARS-COVID-19

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ABSTRACT

Background: Idiopathic acute pericarditis is often presumed to have a viral cause. We hypothesized that if acute viral infection was the cause, the incidence of acute 'idiopathic' pericarditis would decrease during a public health lockdown introduced to prevent the spread of SARS-COVID-19 in New Zealand when acute viral infections decreased by 75% to 99%.
Methods: Hospitalization for acute 'idiopathic' pericarditis during 5 months of the national public health lockdown were compared to 54 months before the COVID-19 pandemic from administrative data.
Results: The hospitalization rate for acute pericarditis was similar before (n = 1364, 24.8 cases/30 days) compared to during the public health lockdown (n = 132, 25.8 cases/30 days), +4% 95 % confidence interval −25 % to +30 % (P = 0.67).
Conclusion: These observations do not support the hypothesis that acute viral infection is the cause for most cases of acute idiopathic pericarditis.

1. Introduction

Over 80 % of cases of acute pericarditis are labeled "idiopathic" after the diagnostic workup. [1,2] When no other cause is identified a viral cause is often presumed.[1,2] However evidence which supports viral infection as the predominant cause of idiopathic pericarditis is indirect. Case reports have associated acute pericarditis with many different viruses.[1,2] In addition symptoms consistent with upper respiratory tract or gastrointestinal infection during the week before presentation with acute pericarditis are common.[3] In contrast to this evidence, case series of hospitalized acute pericarditis patients, which included detailed diagnostic testing to identify the cause, reported evidence for viral infection in only a small proportion of patients.[4–6].

A dramatic change in the population prevalence of acute viral infections occurred during a national public health lockdown implemented to try to eliminate SARS-COVID-19 in New Zealand. Compared to the 5 previous years the national decrease in diagnosed cases for influenza A was 99 %, respiratory syncytial virus 98 %, human *meta*-pneumovirus 92 %, enterovirus 82 %, adenovirus 81 %, parainfluenza virus types 1–3 80 %, and rhinovirus 75 %.[7] We hypothesized that if acute 'idiopathic' pericarditis was caused by respiratory transmitted acute viral infection, the number of cases would be fewer during this lockdown.

2. Methods

National administrative data was used to determine hospital admission rates for a first diagnosis of acute idiopathic pericarditis from July 2015 to December 2019, before the SARS-COVID-19 pandemic, and from May 2020 to September 2020, during the national SARS-COVID-19 public health lockdown in New Zealand. During April 2020, the first full month of the lockdown, acute hospital admissions for other cardiac diagnoses including non-ST elevation myocardial infarction and unstable angina decreased by 30% to 50%, [8] so this month was reported separately.

World Health Organization International Classification of Diseases (ICD-10) codes were used to identify patients with a first primary hospital discharge diagnosis of acute 'idiopathic' pericarditis (code 130.0), 'viral' pericarditis (131.1, excluding all non-viral infective causes), or acute pericarditis 'cause not specified' (130.9) for the New Zealand population (~5.1 million). The national hospitalization rate was then determined for each month, expressed as cases/30 days. Seasonal

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variation was also evaluated.

3. Results

During the 54 months before the SARS-COVID-19 pandemic there were 1364 first diagnoses of acute 'idiopathic' pericarditis, 986 in men (72 %) and 378 in women (28 %). For this period the mean monthly (30 days) hospitalizations for acute idiopathic pericarditis were 24.8 (95 % confidence interval 23.5–26.2), range 15.0 to 37.7. The average number of cases per 30 days during summer months was 23.9, fall 22.6, winter 27.3, and spring 24.9 (p-values by season > 0.05). Rates were similar by year.

During the national public health lock down between 1 May 2020 and 30 September 2020 there were 132 first diagnoses of acute pericarditis. The average 30-day rate for these 5 months was similar to before the SARS-COVID-19 pandemic (25.8 versus 24.8/30 days, +4% 95 % confidence interval −25 % to + 30 %, p = 0.67) (see Fig. 1). In April 2020, the first full month of the lockdown, there were 12 cases, consistent with known decreases in other acute cardiac admissions.[8] When this month was included in the post lockdown analysis, the admission rate for acute pericarditis over 6 months was 23.5 / 30 days (-5%, 95 % confidence interval −36 % to + 23 %; P = 0.56).

4. Discussion

The hospital admission rate for acute 'idiopathic' pericarditis in New Zealand was similar during a 5 month national public health lockdown compared to the five years before the SARS-COVID-19 pandemic, even though the prevalence of acute viral infections dramatically decreased.[7] If acute viral infection was the most common cause of 'idiopathic' pericarditis, a decrease in the hospitalization rate for acute pericarditis during the lockdown would be expected. We also found no seasonal variation during the previous 5 years. This observation is consistent with a large Danish study, which reported no variation in hospitalization for acute pericarditis by season.[9].

The study has limitations. It included only patients hospitalized for a first episode of acute 'idiopathic' pericarditis, and the diagnosis was based on administrative data using ICD-10 codes. The severity and causes of acute pericarditis may differ between patients admitted to hospital and managed in primary care. In this study hospitalization rates for acute pericarditis remained relatively stable over the 5 years before, and for most of the lockdown, suggesting that, on average decisions on hospitalization of patients did not change substantially. Data on screening for possible viral causes was not included. This is not currently recommended routinely in clinical practice guidelines because the diagnostic yield is low. [2] SARS-COVID-19 [10] and mRNA vaccines for SARS-COVID-19 [11] have both been associated with acute pericarditis, but during the public health lockdown there were few cases of SARS-COVID-19 in New Zealand, and mRNA vaccines had not yet been introduced. During April 2020, the first full month of the lockdown, hospitalizations for all cardiac admissions decreased related to changes in systems of care. However, the admission rates for acute pericarditis, as well as other cardiac diagnoses, were similar to the long-term average from May to September 2020.[8].

Recurrent pericarditis occurs in 15% to 30% of patients after the first episode. Both first and recurrent acute pericarditis are more common in younger men, both respond to colchicine treatment, [12] and in both the trigger is usually not identified. [1] Recurrent pericarditis may be an autoinflammatory disease, characterised by an excessive activation of the innate immune system, without an extrinsic stressor. [13] Recurrent pericarditis shares clinical and genetic features with Interleukin-1-mediated systemic autoinflammatory diseases such as Familial Mediterannean Fever, which also respond to colchicine and anti-Interleukin-1 agents.[13] The current study does not exclude the possibility that viral infection, which may be remote, was the trigger for auto-inflammation in some patients.

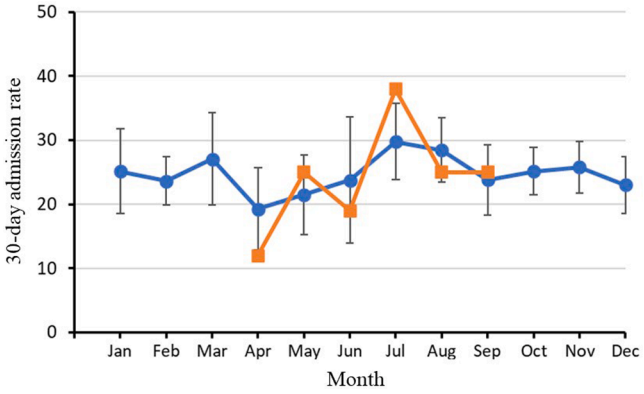

Fig. 1. Average national 30-day admission rate for acute idiopathic pericarditis and 95% confidence interval before the SARS-COVID-19 pandemic from 2015 to 2019 (blue line), and during and after the SARS-COVID-19 lockdown and closing of national borders from April-September 2020 (orange line). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

These observations support the view that the diagnosis "idiopathic acute pericarditis" should not be considered to have a "presumed viral cause", unless there is direct evidence of viral infection. Further research is needed to improve understanding of the pathophysiology of acute idiopathic pericarditis.

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CRediT authorship contribution statement

Anne Langsted: Conceptualization, Formal analysis, Writing – original draft. **Nicola C. Edwards:** Writing – review & editing. **Tom Pasley:** Writing – review & editing. **Ralph A.H. Stewart:** Conceptualization, Data curation, Methodology, Project administration, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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