# Cardiac sequelae after COVID-19: results of a one-year follow-up study with ECG monitoring

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SOUHRN

**Cíl:** Vyhodnotit potřebu EKG monitorace u neselektovaných pacientů po prodělané infekci covid-19 a odhadnout riziko rozvoje arytmií a změny variability srdeční frekvence.
**Metody:** Mezi březnem 2020 a lednem 2021 byli pacienti, kteří se zotavili ze symptomatické infekce SARS-CoV-2 prokázané polymerázovou řetězovou reakcí (PCR) (varianty alfa a beta), zařazeni do prospektivní observační kohortové studie CoSuBr (COvid SUrvivals in BRno). Byly provedeny dvě sedmidenní monitorace EKG ve dvou časových intervalech: nejméně šest týdnů po infekci a s ročním odstupem. Vedle demografických parametrů, anamnézy a klinického vyšetření byla hodnocena i variabilita srdeční frekvence (HRV).
**Výsledky:** Studijní soubor tvořilo 74 jedinců, z toho 42 žen (56,8 %). Průměrný věk činil 48 let (rozmezí 21–77 let). Průměrný časový interval mezi nástupem nákazy a první návštěvou dosahoval 105 dní. V době zařazení se u 61 % pacientů projevovaly přetrvávající příznaky, přičemž více než polovina celého souboru (51,3 %) zmínila alespoň jeden příznak možného srdečního původu (dýchací potíže, bušení srdce, nevýkonnost, únava). Nedošlo k žádné změně v průměrné srdeční frekvenci mezi návštěvami (72,7 ± 8 vs. 72,5 ± 8/min; $p$ = 0,756) a nebyla zaznamenána žádná změna výskytu supraventrikulární nebo komorové ektopie. Nebyl zjištěn rozdíl v EKG parametrech mezi symptomatickými a asymptomatickými skupinami. Při roční kontrolní návštěvě po infekci covid-19 nedošlo ke změně variability srdeční frekvence hodnocené pomocí SDNN (V1 vs. V2 156,6 ± 40,6 vs. 156,0 ± 38,0; $p$ = 0,855), rMSSD (V1: 33 ± 13,95 vs. 30,6 ± 12,89; $p$ = 0,175) a trianglu (V1: 28,5 ± 7,8 vs. 29,5 ± 8,8; $p$ = 0,488). Hodnotili jsme přítomnost supraventrikulárních a komorových arytmií a monitorování EKG neprokázalo žádnou významnou patologii.
**Závěr:** Navzdory mnoha informacím o srdečním postižení způsobeném SARS-CoV-2 naše studie nenaznačuje zvýšené riziko vzniku arytmií po covidu-19 v ročním sledování. Na základě našich výsledků se po zotavení z covidu-19 nedoporučuje rutinní monitorování EKG.



*Keywords:*
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ABSTRACT

**Objective:** To evaluate the need for ECG monitoring of unselected patients recovered from COVID-19 and to estimate the risk of development of arrhythmias and heart rate variability change after severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).
**Methods:** Between March 2020 and January 2021 patients who had recovered from PCR-proven SARS-CoV-2 symptomatic infection (alfa and beta variants) were enrolled in a prospective observational cohort study CoSuBr (COvid SUrvivals in BRno) and those with two 7-day ECG monitoring have been analysed.
Demographic parameters, patient history, clinical evaluation, and 7-day ECG monitoring were recorded within two times intervals: the first at least six weeks after infection and a follow-up visit one year later to establish any changes in heart rate variability (HRV).

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**Results:** The study group consisted of 74 individuals, 42 of which were females (56.8%), with 7-day ECG monitoring performed in both time intervals. The mean age was 48 years (range 21–77 years). The mean time interval between the onset of the infection and the follow-up visit was 105 days. At the time of enrolment, 61% of the patients were experiencing ongoing symptoms, while more than half of the whole group (51.3%) mentioned at least one symptom of possible cardiac origin (breathing problems, palpitations, exercise intolerance, fatigue). There was no change in the average heart rate between visits (72.7 ± 8 vs. 72.5 ± 8/min; $p = 0.756$), and no difference in the incidence of supraventricular or ventricular ectopy. There was no difference in parameters between symptomatic/asymptomatic groups.
At the one-year follow-up visit after COVID-19 infection, there was no change in heart rate variability evaluated by SDNN (V1 vs. V2 156.6 ± 40.6 vs. 156.0 ± 38.0; $p = 0.855$), rMSSD (V1: 33 ± 13.95 to 30.6 ± 12.89; $p = 0.175$) and triangle (V1: 28.5 ± 7.8 to 29.5 ± 8.8; $p = 0.488$). We evaluated the presence of supraventricular and ventricular arrhythmias, and the ECG monitoring showed no significant pathology.
**Conclusion:** Despite a lot of information regarding cardiac impairment caused by SARS-CoV-2, our study does not suggest an increased risk in developing arrhythmias after COVID-19 in the one-year follow-up. Based on our results, routine ECG monitoring is currently not recommended after recovery from COVID-19.

COVID-19 is primarily a respiratory disease with potential extra pulmonary manifestations. Lethality is generally low, and most patients recover within one or two weeks. The COVID-19 pandemic hopefully reached its peak during the last couple of years, during which it left behind not only fatalities but also many patients with ongoing problems. The condition where the symptoms of COVID persist beyond week 4 or beyond week 12 are called "long COVID" and "post-COVID syndrome", respectively.[1]

Approximately 5–10% fail to recuperate fully. Considering the number of people infected worldwide, dealing with post-COVID patients can be challenging for health care systems. The symptoms may affect many organ systems, including the cardiovascular system,[2] and may develop regardless of the severity of acute infection. The care and management of these long haulers is nowadays based especially on the effort to rule out other common causes of the present symptomatology.[3] As the manifestation of post-COVID syndrome is highly varied and different phenotypes have recently been established,[4] an interdisciplinary approach is recommended and physicians across many fields of medicine are involved. This investigation is consuming in terms of time and personnel, is expensive, and the efficacy is not very high. There is thus a need for a comprehensive evidence-based plan to manage post-COVID symptoms. No specific therapy is available so far, and recommendations are mostly guided by our experiences with non-COVID diseases.[5]

Many symptoms of post-COVID syndrome may overlap with those of a cardiovascular origin. Despite data from MRI studies about myocardial damage,[6] the clinical significance of these findings is still unknown. Large epidemiological studies have provided data about higher cardiovascular risk after hospitalisation for COVID-19,[7] but an evidence-based recommendation for single patients, even with ongoing symptomatology, is still based mainly on recommendations from authorities.

To better understand the cardiovascular sequelae of COVID-19, we initiated a prospective observational cohort study called CoSuBr (COvid SUrvivals in BRno). Results of the echocardiographic parameters and biomarkers have been recently published[8] and in these subsequent analyses we were focused on arrythmias and heart rate variability. The aim of our study was to help physicians and their patients with a tool which is commonly available in their practise – 7-day ECG monitoring to estimate the risk of developing arrhythmias during a one-year follow-up after COVID-19 recovery and to detect possible changes in heart rate variability as a marker of autonomic dysfunction.

## Methods

We recruited patients in an outpatient clinic for post-COVID care at least 6 weeks after their recovery from acute symptomatic COVID-19 infection (PCR-verified SARS-CoV-2 from a nasopharyngeal swab). After signing an informed consent form, a clinician interviewed all patients. The demography (age, sex), medical history, and data about the acute phase of COVID-19 infection were collected. The severity of the disease was classified as follows: mild without dyspnea or abnormal chest imaging; moderate for evidence of a lower respiratory tract infection without a decrease of oxygen saturation <94%; severe with a need for oxygen; critical with respiratory failure and/or shock, need for oxygen/artificial ventilation. The need for intensive care, type, and duration of the symptomatology, and treatment were also evaluated. Special interest was paid to ongoing ailments. Two 7-day ECG monitoring was recorded: first at least 6 weeks after acute infection (V1), and the second one a year later (V2). All patients underwent also body plethysmography at the beginning of the trial and those with moderate/severe respiratory impairment have been excluded.

The trial protocol was approved by the institutional review board at University Hospital Brno (08-100620/EK). The data were managed using REDCap electronic data capture tools, and a data analysis was carried out using SAS software. The mean (and standard deviation) was used to present average values for normal data distribution, and the median (and interquartile range) was used to present data of non-normal distribution. Prevalence is reported as the number of and percentage of patients reporting the symptom within the group. A paired t-test or its non-parametric alternative (Wilcoxon test) was used to compare variables for one subject between the visits.

## Results

The whole study group consisted of 106 individuals, while an evaluation with an ECG Holter monitor was performed in 96 of them on V1, and 74 on V2. The analyses performed for the study group consisted of 74 individuals, 42 of which were females (56.8%), with 7-day ECG monitoring performed in both time intervals. The mean age was 48 years (range 21–77 years). The mean time interval between the onset of the infection and the follow-up visit was 105 days. The most prevalent comorbidities were arterial hypertension (n = 15, 20.3%) and dyslipidemia (n = 12, 16.2%); others included obesity, e.g. BMI over 30 (n = 8, 10.8%), hepatic steatosis (n = 3, 4.1%), asthma (n = 4, 5.4%) and renal insufficiency (n = 5, 6.8 %); see **Table 1**. Almost a quarter of the patients (23.0%) required hospitalisation during the acute phase of the disease, with only two of them requiring artificial ventilation; the rest recovered at home with a mild form of the disease. At the time of enrolment, 61% of the patients were experiencing ongoing symptoms, while more than half of the whole group (51.3%) mentioned at least one symptom of possible cardiac origin (breathing problems, palpitations, exercise intolerance, fatigue).

We assessed the presence of supraventricular premature beats (SPBs) and ventricular ectopy (premature ventricular contractions), and the ECG monitoring after the one-year period showed no significant pathology or trend in the incidence of both these events. These data are summarized in **Table 1**.

During the one-year follow-up after COVID-19 infection, there was no change in heart rate variability evaluated by SDNN (V1 vs. V2 156.6 ± 40.6 vs. 156.0 ± 38.0; $p = 0.855$), rMSSD (V1: 33 ± 13.95 to 30.6 ± 12.89; $p = 0.175$) and triangle (V1: 28.5 ± 7.8 to 29.5 ± 8.8; $p = 0.488$). Dividing heart rate oscillations into low-frequency (LF), and high-frequency (HF) bands, we found significant statistical changes between V1 and V2 for LF (718 ± 433.7 to 646 ± 361; $p = 0.024$) and HF (341.5 ± 335 to 268.0 ± 266; $p = 0.032$).

There was no difference in presence of supraventricular premature beats (SPBs), ventricular ectopy (premature ventricular contractions), and heart rate variability between the group with ongoing symptomatology of possible cardiovascular origin and those without these symptoms.

During the one-year follow-up, one patient was diagnosed with asymptomatic paroxysmal atrial fibrillation, and one with second degree atrioventricular block. No patient died during the follow-up period.

The change in heart rate variability, as well as the incidence of ventricular and supraventricular ectopy during the follow-up visit, are summarised in **Table 2**.

**Table 1 – Incidence of supraventricular and ventricular ectopy in visit 1 and 12 months later (V2)**

| | V1 mean (SD) | V2 mean (SD) | *p* |
|---|---|---|---|
| Heart rate | 72.7 (8.5) | 72.5 (8.6) | 0.756 |
| Min. HR | 44.9 (6.2) | 45 (5.2) | 0.982 |
| Max. HR | 154.5 (21.6) | 151.7 (21.2) | 0.186 |
| Afib, flutter | 0 (0) | 0.8 (7.2) | 1 |
| SVT non-sustained | 9.8 (29.8) | 15 (50.6) | 0.800 |
| SPBs | 1142 (4225) | 1278.9 (4262) | 0.790 |
| SPB couplets | 24.9 (98.5) | 23.8 (87.5) | 0.043 |
| PVCs isolated | 482 (2214.5) | 941 (5451.5) | 0.965 |
| PVCs couplets | 2.9 (16.7) | 1.6 (5.3) | 0.674 |
| PVCs bigeminy | 2.3 (11.6) | 16 (116.5) | 0.984 |
| VT non-sustained | 0.2 (0.58) | 0.4 (0.9) | 0.127 |

SBPs – supraventricular premature beats, premature ventricular contractions; SVT – supraventricular tachycardia; VT – ventricular tachycardia.

**Table 2 – Parameters of heart rate variability in visit 1 and 12 months later (V2)**

| | V1 mean (SD) | V2 mean (SD) | *p* |
|---|---|---|---|
| HF bands | 341.5 (334.9) | 268 (266.3) | **0.032** |
| LF bands | 718.8 (433.7) | 646.8 (361) | **0.024** |
| rMSSD | 33 (13.9) | 30.6 (12.8) | 0.175 |
| pNN50 | 10.8 (9.28) | 9.3 (8.7) | 0.255 |
| SDNN | 156.6 (40.6) | 156 (38.2) | 0.855 |
| SDDN index | 59.5 (18) | 55.8 (15.7) | **0.036** |
| Triangle | 28.5(7.8) | 29.5 (8.8) | 0.488 |

HF – high frequency; LF – low frequency.

## Discussion

Our 7-day study with an ECG Holter monitor is a part of a larger follow-up project (CoSuBr) on the subjects after SARS-CoV-2 infection. Echocardiographic parameters and biomarkers from the whole study group (n = 106) have been published recently.[8] These subanalyses of ECG Holter were done only for the subset of participants (n = 74), where both recordings were available.

A majority of our patients had a mild course of the acute disease, which is the same proportion of COVID-19 disease's severity globally. Our population represents a wide spectrum of ages. More than half of the study group (61%) fulfilled the criteria of post-COVID syndrome at the time of enrolment, which is given by the source of our subjects – mainly an outpatient clinic for post-COVID care.

Except for hypertension (20%), none of our patients suffered from cardiovascular disease before the enrolment. The rate of repeated infection was rare – only four patients had recidivism of COVID during the one-year follow-up period. In addition, an analysis of vaccinated and unvaccinated is impossible, because recruitment to the study had nearly been completed by the time the vaccine became available.

Despite the high rate of symptomatic patients in our study group, the incidence of clinical significance arrhythmias was rare, and their distribution was in the oldest tenth of our study group. One patient was diagnosed with *de novo* paroxysmal atrial fibrillation (female, 73 years old, dilated left atrium), another with atrioventricular block with an indication for implantation of a pacemaker (male, 71 years old; cardiac MRI did not find any signs of myocardial inflammation). We do not suspect these endpoints to be the direct result of SARS-CoV-2 infection. Also the absence of difference in ECG parameters in symptomatic and asymptomatic subjects doesn't support the cardiovascular apparat as organ system responsible for ongoing problems in post-COVID patients. No patients died during the follow-up period.

The impairment of the cardiovascular system during the acute phase of COVID-19 infection has been well described.[9] The impact of hypoxia and cytokine storm during severe inflammatory responses in predisposed individuals can cause a broad spectrum of cardiovascular pathologies, including arrhythmias. In addition, myocardial damage caused by SARS-CoV-2 is suspected for reasons including its spike affinity to the ACE2 receptor binding domain, which plays an essential role in the renin-angiotensin system.[10]

A significant proportion of patients suffered ongoing problems during the weeks and months after acute COVID infection. Common complaints have included persistent fatigue, shortness of breath, brain fog, and also palpitations. Davies et al.[11] in an online survey of a post-COVID population report tachycardia in 61.4% of patients and suspect possible postural orthostatic tachycardia syndrome in almost a third of them. There are also several possible mechanisms for decreased arrhythmic burden after COVID infection: direct cytotoxic injury, antibody or cytokine induced damage, changes in microcirculation, inflammatory cardiac channelopathies and many others.[12,13] During all waves of the pandemic, hospitalisation was high especially in elderly individuals and/or in those with comorbidities. Large epidemiological studies and data from registries brought strong evidence of cardiovascular risk for the post-COVID population between 30 days and one year after acute illness (hazard ratio for dysrhythmias was 1.69),[13] and similar results for any cardiovascular endpoints were described also by Wang et al. (HR 1.552 [1.526–1.578]).[7] The risk was only slightly higher in the non-hospitalised and mainly driven by the severe and critical forms of the disease. An explanation of these findings may be hidden in the frailty of the subjects hospitalised for acute coronavirus infection and the need for admission may only be a marker of lower general health and pre-existing cardiovascular risk. This is in concurrence with the results of our study, when in the early post-acute phase of recovery and one year after there was no change in the incidence of ventricular or supraventricular ectopy. This does not support the theory of SARS-COV-2-induced arrhythmias. Even if our sample is not large, the sequential 7-day ECG monitoring in predefined time frame of one year is robust enough to be able to find possible changes. Considering that in our study population, there were more than 60% of patients with ongoing problems and that previously published analyses of the whole CoSuBr study group did not find dynamics of echocardiographic parameters and cardio markers in the one-year follow-up after COVID-19,[8] we assumed that SARS-COV-2's influence on the heart in the post-COVID period is weak (if at all). We also conclude that the symptoms related to post-COVID syndrome are not of cardiac origin.

Observational and epidemiological studies observed a higher rate of atrial fibrillation,[7] while another study from Italy reported an increase of ventricular tachycardia in heart disease patients with implanted devices and the authors suggest an association with lower physical activity during the pandemic.[14] Significantly higher rates of isolated ventricular premature beats (VPB) and supraventricular premature beats (SVPB) were found in 53.3% and 52.5% of 90 post-COVID athletes, respectively, but no malignant arrhythmias were identified.[15] These data are in concordance with our results and also do not support the theory of a direct relationship between infection and higher risk of arrhythmias during recovery.

Our study group of mostly outpatients with ongoing symptomatology is quite similar to the population enrolled in a Polish long-covid cardiovascular study, which found 6.9% patients with myocardial dysfunction based on echo or MRI.[16] This was not dependent on the severity of the disease course. In their observational study they also found more (p=0.008) heart rhythm disturbances (atrial fibrillation, supraventricular extrasystole, ventricular extrasystole) in patients with vs without myocardial dysfunction after COVID. In contrast to classic postCOVID pattern, patients affected were mostly men of average age 51±13 and also the absence of previous cardiovascular disease was based only on patient's history. No serious arrhythmias and/or conduction disorders were pointed out. They conclude that ECG changes can indicate the possibility of cardiovascular dysfunction. Relationship between changes described and SARS-COV-2 infection remains uncertain.

In post-COVID syndrome patients, mainly in young women, inappropriate sinus tachycardia was described.[17] Moreover, a decrease in physical activity after acute infection, especially in those with ongoing symptomatology, can lead to deconditioning and secondarily to increased heart rate. In our study there was no statistical significant difference in HR between visits. There was only one subject (20-year-old female) fulfilling the condition for inappropriate sinus tachycardia who was successfully treated by Ivabradin. This condition can be related to autonomic dysfunction, which is one of the possible mechanisms of the development of post-COVID symptomatology.[18,19]

Heart rate variability (HRV) from at least a 24-hour measurement is a tool for stratifying cardiovascular risk, when the SDDN values are able to predict morbidity and mortality.[20] In the one-year follow up after COVID-19 infection, our study did not detect any change in heart rate variability as evaluated by SDNN, rMSSD, and triangle (**Table 2**). Dividing heart rate oscillations into low-frequency (LF) and high-frequency (HF) bands, we found significant statistical changes between V1 and V2 for LF and HF. These parameters are mostly affected by breathing rate,[21] which can be a result of the lungs healing and/or regaining conditioning. These parameters also represent possible autonomic dysregulation (HF/LF ratio) and sympathetic/parasympathetic system activity, which was also described after COVID infection.[22] One of them is postural orthostatic tachycardia syndrome (POTS), which has been described mainly in young woman after a mild course of the acute disease and appears to be the most common autonomic phenotype among these patients.[23] Our data for heart rate variability analyses were not obtained under controlled conditions, but come from our subjects under normal living conditions. Based on this, we are not able to express a final statement on HRV.

There are surely limitations of our study. First of all the sample size is too small to be able to make definite conclusion about the risk of arrhythmias in post-COVID patients. Another problem is also the change of immune response of virus naïve population, on which our study has been performed and the nowadays people, who surly underwent several waves of coronavirus infection and the most vulnerable are usually (or should be) vaccinated. There is also a change of virus itself, when all of our patients were enrolled in time when original SARS-CoV-2 or beta variant dominated in the Czech Republic and before current variants or omicron and even delta have been on stage.

## Conclusion

Despite a lot of information regarding cardiac impairment of SARS-CoV-2, our study does not suggest an increased risk in developing arrhythmias after severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in a one-year follow-up, even in a population with a high proportion of ongoing symptomatology. There was no difference in parameters between symptomatic/asymptomatic groups. We have not found any change in heart rate variability during the one-year follow-up, but some findings from our study may suggest autonomic dysfunction after COVID-19. Based on our results, routine ECG monitoring is currently not recommended during COVID-19 recovery.

**Conflict of interest**
None declared.

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**Ethical statement**
The studies involving human participants were reviewed and approved by Ethic Comitee FN Brno, 08-100620/EK.

**Informed consent**
The patients/participants provided their written informed consent to participate in this study.

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