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# ECG Changes in COVID-19 Infection


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| *Aim* | To evaluate changes in 12-lead ECG in patients with coronavirus infection. |
| *Material and methods* | This article describes signs of electrocardiographic right ventricular «stress» in patients with COVID-19. 150 ECGs of 75 COVID-19 patients were analyzed in the Institute of Cardiology of the National Medical Research Centre for Therapy and Preventive Medicine. The diagnosis was based on the clinical picture of community-acquired pneumonia, data of chest multispiral computed tomography, and a positive test for COVID-19. ECG was recorded both in 3–6 and in 12 leads. Signs of right ventricular (RV) stress, so-called systolic overload (high R and inverted $T_{V1–3}$ and TII, III, aVF), and diastolic overload (RV wall hypertrophy and cavity dilatation; complete or incomplete right bundle branch block) were evaluated. |
| *Results* | The most common signs for impaired functioning of the right heart include emergence of the RV P wave phase (41.3%), incomplete right bundle branch block (42.6%), ECG of the $S_IQ_{III}T_{III}$ type (33.3%) typical for thromboembolic complications, and signs of RV hypertrophy, primarily increased $S_{V5–6}$ (14.7%). These changes are either associated with signs of RV myocardial stress (16%) or appear on the background of signs for diffuse hypoxia evident as tall, positive, sharp-ended T waves in most leads (28%). |
| *Conclusion* | A conclusive, comprehensive assessment of the reversal of hemodynamic disorders and electrocardiographic dynamics in patients with COVID-19 will be possible later, when more data become available. |



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| *Keywords* | Electrocardiography; COVID-19; right ventricular stress; left ventricular hypertrophy; ischemia |
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When managing patients with coronavirus disease [1], ECG recording is recommended due to the need to monitor the QT duration. There is a risk of fatal rhythm disorders, such as ventricular tachycardia and ventricular fibrillation, when using azithromycin and chloroquine to treat COVID-19, which causes the QT prolongation [2]. It is recommended that three standard leads, or even one monitor lead, be recorded to measure the QT interval (to avoid prolonged contact with the patient) [3]. The aim of the study is to evaluate changes in 12-lead ECG in patients with the coronavirus disease.

## Material and methods

The study included the first 75 patients with COVID-19 admitted to the Cardiology Research Institute of the Russian National Cardiology Research Center in April 2020. The diagnosis was based on the clinical presentation of community-acquired pneumonia, chest multislice computed tomography, and a positive COVID-19 test.

## ECG recording in the coronavirus disease

The ECG laboratory of the Cardiology Research Institute analyzed 150 ECG records of 75 patients with COVID-19. ECG was recorded on Russian EASY RCG digital devices with the remote transmission. The devices used automatic ECG analysis software:

a) to measure the duration of waves and intervals and the amplitudes of the atrioventricular complex;
b) to perform ECG analysis with the automatic generation of conclusions for the main electrocardiographic syndromes.

The recorded ECGs were transferred to a tablet, where they were graphically represented, and the automatic ECG analysis data was generated. Thus, the doctor could acquire all the ECG data and automatic syndrome-based conclusion at the patient's bedside.
The ECG was transferred from the tablet to the ECG storage server installed in the "clean" area of the ECG laboratory via wi-fi and the doctors' workplaces. After the doctor had checked the calculated variables and automatic conclusions, the ECG was sent to the electronic chart.
In the Cardiology Research Institute, ECG was registered in both 3-6 and 12 leads. The standard 12-lead ECG allowed us to perform routine analysis


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of the atrioventricular complex's initial morphology with the diagnosis of various electrocardiographic syndromes (atrial and ventricular hypertrophy, myocardial infarction, ischemia, myocardial damage, metabolic changes, ventricular conduction disorders) and evaluate the ECG changes.

The advantage of 12-lead ECG (compared to the recommended limb leads) is also the more accurate measurement of the QT and QTc intervals [3]. The QTc interval measured in a limb lead is shorter than in a 12-lead ECG.

We observed one more specific feature of the automatic calculation of the QRST complex in our study. The duration of QT and QTc differ when these parameters are determined in different leads. Such variances are taken into account in the ATES software. These variables are calculated in 12 leads simultaneously, when the averaged ventricular complexes of each of the 12 leads are superimposed. This technique allows the first and last reference points of the beginning and the end of the repolarization process to be found with greater accuracy.

Disposable electrodes, similar to those used for ECG monitoring, were used to record the chest leads. If possible, the disposable electrodes were left on the patient's skin until the next ECG recording, which significantly saved time for repeated ECG records.

The duration of QTc was calculated using Bazett's formula:

$$QTc = QT/\sqrt{RR}.$$

The detection rate of various electrocardiographic syndromes was calculated based on ECG data recorded at admission.

Parameters showing right heart hypertrophy were evaluated in the analysis of the standard 12-lead ECG [4]:

- signs of right atrial enlargement, namely the amplitude of $P_{II, III, aVF}$ > 2.5 mm $P_{V1}$ > 1.5 mm, Macruz index <1.0 (normal 1.1–1.6);
- signs of right ventricular (RV) enlargement $R_{V1}$ ≥7.0 mm, $S_{V1}$ < 2.0 mm, $qR_{V1}$, depression $S_{V1-3}$ and negative $T_{V1-3}$; $R_{V5-6}$ ≤S (R/S <1), $V_{5-6}$ <5 mm, $S_{V5-6}$ ≥ 7 mm;
- ECG criteria for acute cor pulmonale: $S_IQ_{III}T_{III}$ with $T_{V1-3}$ inversion.; $S_IT_{III}$ or $T_{III}$ and negative $T_{V1-3}$; $S_IQ_{III}T_{III}$ with right bundle branch block (RBBB);
- additional signs of pulmonary embolism (PE) in the form of T-wave inversion in the right thoracic leads; $S_IQ_{III}T_{III}$; transient RBBB; $S_I$, $T_{III}$ or $T_{II}$; P-pulmonale, particularly of a transient nature; transient ST depression in the left precordial leads.

Signs of RV myocardial stress, systolic overload (high R and inverted $T_{V1-3}$ and $T_{II}$, III, aVF), and diastolic overload (RV hypertrophy and dilatation, incomplete or complete RBBB) were also estimated.

Other ECG parameters were evaluated using established criteria for left ventricular hypertrophy (LVH), focal scar, ischemic and metabolic disorders [5].

**Table 1.** The rate of individual ECG syndromes indicating right ventricular stress

| ECG syndrome | Number of cases | |
|---|---|---|
| | n | % |
| Peaked P wave | 31 | 41.3 |
| Typical P-pulmonale | 3 | 4.0 |
| Incomplete RBBB | 32 | 42.6 |
| Complete RBBB | 1 | 3.0 |
| $S_IQ_{III}T_{III}$. | 25 | 33.3 |
| Signs of RV myocardial stress | 12 | 16.0 |
| Signs of RV hypertrophy | 11 | 14.7 |
| Signs of hypoxia (high positive peaked T waves in most leads) | 21 | 28.0 |
| Metabolic changes as hypokalemia | 4 | 5.3 |
| Low-voltage ECG as a manifestation of emphysema effects | 4 | 5.3 |
| Brugada syndrome phenotype type 2 | 3 | 4.0 |

ECG, electrocardiogram;
RBBB, right bundle branch block; RV, right ventricle.

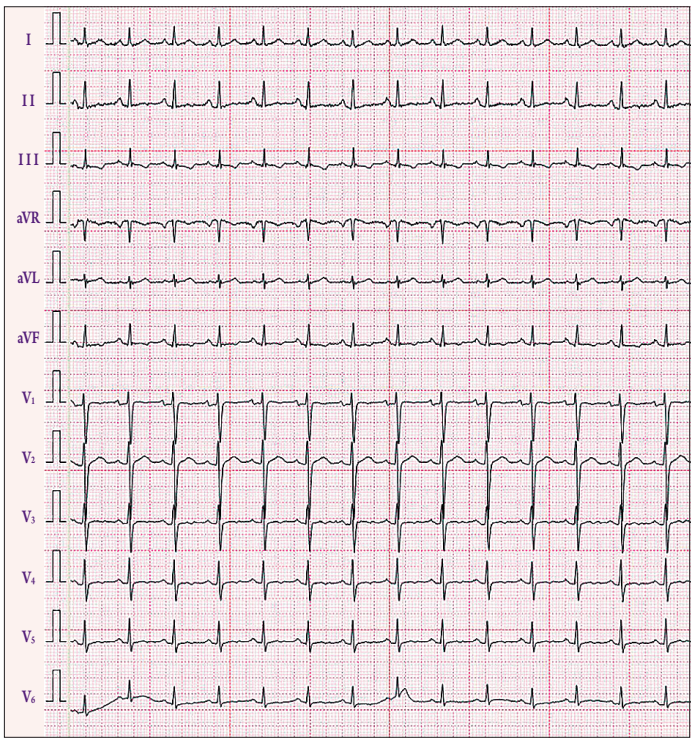
## Results

The pathogenesis of lung damage in the coronavirus disease involves the development of right ventricular stress. Ssigns may appear on the ECG. Table 1 shows the detection rate of various electrocardiographic syndromes that characterize the hemodynamic adjustment of the heart with increased RV load. At the same time, only eight patients with COVID-19 showed relatively normal ECG findings. The remaining 90.6% of patients with COVID-19 showed signs of abnormal ECG changes.

### *Main electrocardiographic parameters of the right ventricular stress*

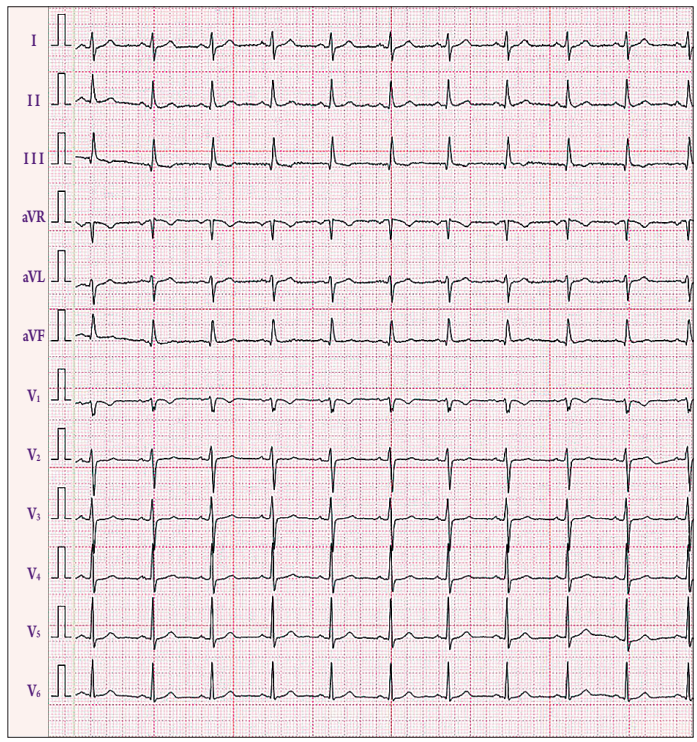
The most common of these were the signs of pulmonary hypertension, namely changes in the atrial component with signs of P-pulmonale. ECG showed peaked P waves, i.e., P with marked atrial phase, in 45% of patients. However, the amplitudes of those changes did not meet the ECG criteria of P-pulmonale. As can be seen in Table 1, only 3

**Figure 1.** Typical changes in the P-wave in a patient with coronavirus pneumonia




A 40-year-old female patient. Suspected coronavirus infection COVID-19 (moderate illness). Bilateral multisegmental pneumonia (without respiratory failure). Chest multislice computed tomography revealed signs of bilateral atypical pneumonia with sites of moderate consolidation; lung tissue lesion of 25–50%.
ECG of 15.04.20: sinus tachycardia, 107 bpm.
Signs of P pulmonale: peaked P waves in leads I, II, III, and aVF; maximum $P_{II}$ amplitude = 2 mm.
Positive phase of PV1 wave up to 1 mm with a normal P wave duration of 104 msec; Macruz index =0.68.

**Figure 2.** SIQIIITIII type changes in ECG




A 28-year-old female patient.
Diagnosis: coronavirus infection COVID-19, PCR+.
Community acquired bilateral multisegmental pneumonia.
Data of computed tomography:
lung tissue lesion of approximately 60%.
ECG of 16.04.20: sinus rhythm, 80 bpm.
Heart electrical axis deviation to the right.
Changes in the atrial component with elements of P pulmonale: peaked waves in lead II; $P_{II}$ amplitude =1 mm.
Macruz index, 0.72. $S_IQ_{III}T_{III}$ type, S up to 5–6.
Negative $T_{V1}$ wave; flattened $T_{V2-3}$.

(4 %) patients had changes in the P-wave amplitude (≥2.5 mm) corresponding to P-pulmonale. Figure 1 shows typical changes in the P-wave in a patient with coronavirus pneumonia.

A similar rate was shown for the signs of diastolic overload, manifested by impaired conduction in the right bundle branch block (45.6%). However, these signs were not classical. The complete block was detected in 1 case, and other patients showed signs of conduction disorders in the form of incomplete RBBB. There were no voltage signs of RV hypertrophy corresponding to the accepted definition of diastolic overload.

The signs of incomplete RBBB also do not meet standard criteria. Most often, there was no rSR pattern in V1-2, but high SV5–6 was detected. The QRS complex prolongation of more than 90 ms indicates the conduction delay. Deep SV5-6 can be associated not only with incomplete RBBB but also with RV enlargement.

$S_IQ_{III}T_{III}$ can be considered the third and rather specific sign of ECG changes (Figure 2). It is an electrocardiographic sign of PE. The prevalence of this ECG feature in patients with viral pneumonia was relatively high (33%).

In 16% of patients with COVID-19, the signs of RV myocardial tension presented as changes in the ventricular complex end part in the right thoracic leads were observed. This can be considered as the fourth type of ECG changes.

Voltage signs of RV hypertrophy in the form of the Sokolow-Lyon index $\Sigma R_{V1}S_{V5} \geq 105$ mm are rare. The most common sign is an increase in the amplitude of SV5-6 ≥7 mm.

Various combinations of these signs were detected in most cases, which can be combined and titled "electrocardiographic signs of right ventricular stress".

Another ECG feature found in 28% of cases was signs of diffuse myocardial hypoxia in the form of symmetric


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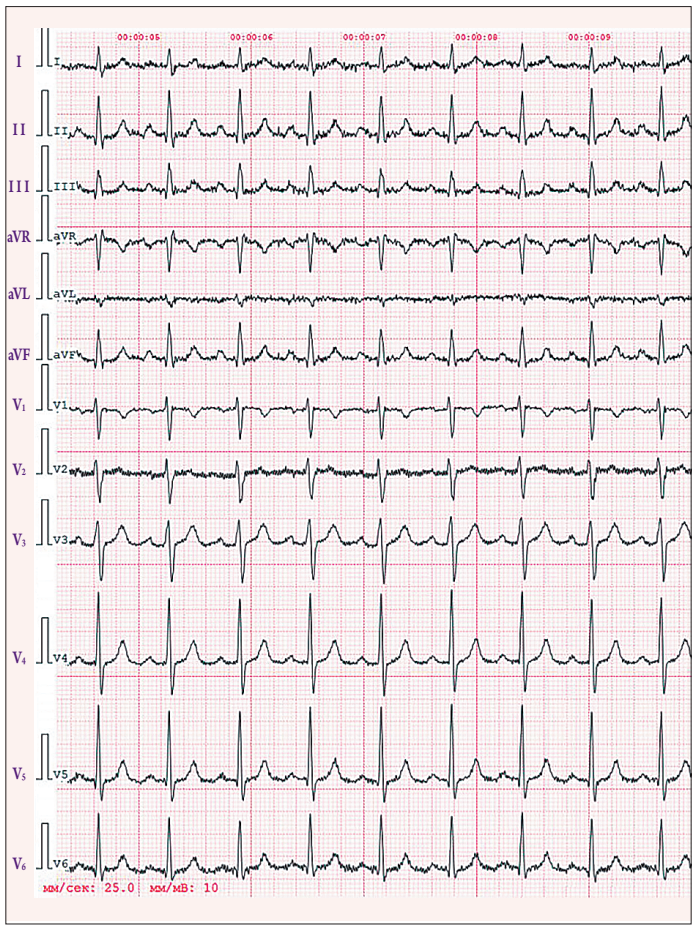
**Figure 3.** Signs of pulmonary hypoxia




Sinus tachycardia, heart rate, 95 bpm.
Changes in the atrial component with elements of P pulmonale. Tall, peaked T waves in leads II, III, aVF, V3–6, a sign of diffuse myocardial hypoxia.

peaked T-waves, often with a slight ST depression. When describing ischemic changes in the myocardium, this sign is usually attributed to the manifestations of diffuse myocardial hypoxia associated with coronary insufficiency. However, signs of myocardial hypoxia are possible for insufficient oxygen supply to the myocardium in anemia and pulmonary disorders, as well as for coronary artery disease.

Figure 3 shows changes in the ST–T complex in a 78-year-old patient with mild bilateral pneumonia and confirmed COVID-19.

We should also mention the transient Brugada syndrome phenotype, caused by a body temperature increase. Three similar cases were identified in the data analyzed, one of which is shown in the following illustration (Figure 4).

According to our observations, the development of the Brugada syndrome phenotype could be observed in different leads from V1 to V3. Furthermore, the same patient could have all, as shown in this example.

How does the development of pulmonary hypertension affect the signs of left ventricular disorders in patients with COVID-19? If we consider all the listed electrocardiographic signs of "right ventricular stress", then 6 of 7 patients with signs of LVH had changes in the atrial component in the form of both atria enlargement (n = 3) or P-pulmonale (n = 3). Only one patient with signs of LVH and P-pulmonale showed changes in the RV: incomplete RBBB, $S_IQ_{III}T_{III}$.

Of the 15 cases of ischemic changes in the myocardium in ECG, 9 patients had signs of changes in the right heart. Elements of P-pulmonale were detected in 3 cases of a combination of COVID-19 and coronary artery disease (CAD).Changes in the ST–T complex in the $V_{1-3}$ leads in 4 cases, indicating RV myocardial tension, $S_IQ_{III}T_{III}$ were detected in 2 patients, and incomplete RBBB was found in 2 patients.

Thus, 73% of patients with COVID-19 + hypertension and CAD showed ECG signs of LVH, ischemia, and scarring of the myocardium combined with the signs of changes in the right heart.

## ECG changes over time in patients with COVID-19

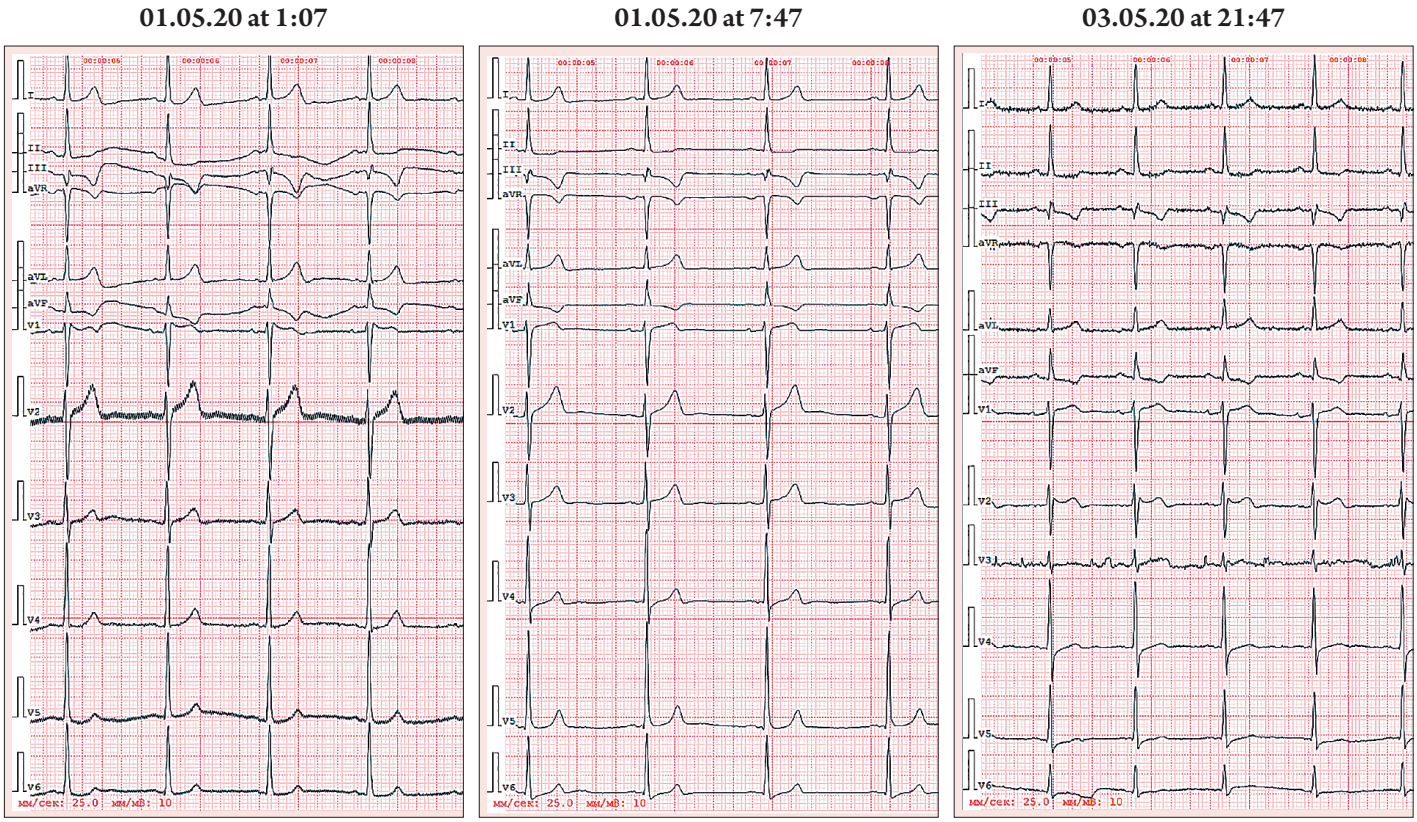
In our study, a small number of patients who initially had no pathological ECG abnormalities showed changes in the atrial component with P-pulmonale, conduction disorders in the right bundle branch, or signs of RV myocardial tension. In almost all cases SV5–6 waves appeared. In patients with initial ECG abnormalities of the right ventricular stress type, these signs progressed by the end of Week 1 of the disease and decreased. All patients taking medications to prolong QT had a significant QT prolongation of up to 500 ms.

It should be noted that the analysis of individual discharge summaries of patients with COVID-19 identified no significant ECG deviations at both admission and discharge. The final full assessment of hemodynamic disorder recovery and electrocardiographic changes in patients with COVID-19 will be available later when a sufficient amount of data is accumulated.

## Discussion

The ECG analysis of Patients with COVID-19 allowed us to identify and describe various electrocardiographic characteristics of acute and subacute cor pulmonale, signs of right heart hypertrophy similar to changes observed in pulmonary arterial hypertension, and signs of systolic and diastolic RV myocardial stress [4]. In this study, we used the general term "electrocardiographic signs of right ventricular stress".

**Figure 4.** ECG of a male 50-year-old patient with confirmed COVID-19 and directional diagnosis of acute coronary syndrome




On the first ECG recorded at admission. Brugada syndrome phenotype type 2 was observed. $ST_{V1}$ saddle-back elevation was the reason for emergency hospitalization due to suspected acute coronary syndrome in COVID-19. The body temperature was elevated to 39°C the day prior to and at admission. In the morning (second ECG), the temperature had decreased and the signs of Brugada phenotype disappeared. In the third ECG (normal body temperature), there were signs of Brugada phenotype type 2 in lead $V_2$.

In COVID-19 studies, QT prolongation is observed when antimalarial quinidine-based drugs are used [2]. The electrophysiological mechanism of quinidine involves direct action on the cellular transmembrane action potential. Quinidine suppresses the automatism of diastolic depolarization (phase 4) of pacemaker cells. This is due to decreased sodium ion transport from the extracellular space into the cell during diastole. Quinidine increases the duration of action potential and effective refractory period, reducing potassium release from cells to the extracellular space during repolarization. These effects of quinidine are used to treat arrhythmia [6].

In quinidine therapy, ECG shows QT prolongation, ST depression, negative T-waves, and U-wave amplitude increase. These signs do not require discontinuation of quinidine, although the dosage should be reviewed. The increase in QRS duration by 25% compared to the initial level or QT prolongation by more than 50% requires the discontinuation of quinidine. Frequent polymorphic ventricular extrasystoles, polymorphic ventricular tachycardia or repeated attacks of ventricular flutter fibrillation, and sudden death are the most dangerous complications of quinidine intoxication. Hypotension is another clinically significant complication [6].

Brugada syndrome is a genetic disorder with various pathological ECG changes which increase the risk of ventricular tachyarrhythmias and sudden death. Typical ECG changes are reduced to the right bundle branch pseudoblock and persistent ST elevation in $V_{1-2}$.

The ventricular myocardium consists of three electrophysiologically different cell layers: epicardial cells, endocardial cells, and M-cells. ST-elevation and T-wave inversion in the right precordial leads are thought to be caused in Brugada syndrome by the alterations and shortening of the potential of epicardial and possibly M cells [7-9]. The end of the ventricular repolarization process, localized in the RV outflow tract, changes the RV voltage gradient, leading to the ST elevation [10]. This substrate may suggest the development of local re-entry and ventricular tachycardia.

Local conduction deceleration in the RV outflow tract area due to sclerotic changes or mechanical effects associated with increased pulmonary pressure can lead to RBBB-type extension and splitting of the ventricular complex in $V_{1-2}$.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Brugada syndrome is not associated with structural changes in the heart. However, postmortem histological examination of the heart, both in experimental mice and in clinical cases, may reveal structural, and microscopic abnormalities in the RV outflow tract, as well as local inflammation and fibrosis [11-13].

A reasonable assumption would be that the RV outflow tract is affected in patients with COVID-19 with right ventricular stress, accompanied by pulmonary hypertension. This is not only one of the causes of impaired conduction in the right bundle branch but can also lead to the Brugada syndrome phenotype. According to some data, these transient ECG changes may be a manifestation of concealed Brugada syndrome, which may be accompanied by fatal ventricular arrhythmias [14].

RBBB is the world's third-largest pathology in pneumonia caused by coronavirus disease. The presence of RBBB in the enlarged RV is associated with its diastolic overload. High pulmonary vascular resistance caused by damages to bronchioles and alveoli can lead to the dilation and hypertrophy of RV [7].

Changes in the $S_IQ_{III}T_{III}$ ventricular complex require a separate discussion. This symptom complex is usually detected in PE. The possibility of significant thromboembolism is confirmed by reported clinical findings [3-7]. Venous and arterial thromboembolism, or simply vascular thrombosis due to disproportionate inflammation, hypoxia, immobilization, and diffuse intravascular coagulation, seem to develop in every COVID-19 patient. A third of patients with moderate to severe lung damage have $S_IQ_{III}T_{III}$ syndrome. Based on this sign, thrombotic changes can be suspected in patients with COVID-19 in both large and small pulmonary vessels.

Myocardial damage associated with coronary artery thrombosis in COVID-19 is of particular importance.

In the study [15], elevated troponin levels were associated with adverse COVID-19 outcomes. However, the differential diagnosis of elevated troponin levels in COVID-19 is broad and includes non-specific myocardial damage, impaired renal function (leading to troponin accumulation), myocarditis, PE, and type I and II myocardial infarctions [15].

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