Lessons learned from the management of a case of acute synchronous cardio cerebral infarction in a resource-poor setting

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Abstract

Introduction

Cardio-cerebral infarction (CCI), the simultaneous occurrence of acute ischemic stroke (AIS) and acute myocardial infarction (AMI), is infrequent and poses a management challenge for physicians. There is a paucity of available evidence for the best management approach of this rare clinical entity.

Case Presentation

A 59-year-old female with uncontrolled type 2 diabetes mellitus, hypertension, hyperlipidemia presented 6 ½ hours following sudden right-sided hemiparesis, hemisensory loss, right upper motor neuron facial nerve palsy, aphasia, vertigo, and vomiting. She also had complained of vague anterior chest pain since 12 hours earlier and had worsened to a sudden agonizing chest tightness with autonomic symptoms within a few minutes following the acute neurological event. On admission to the tertiary care center, she had vitals of; pulse rate 79/ minute, blood pressure – 182/100 mmHg, respiratory rate – 22/ minute, SpaO2 – 95%, was confused with GCS of 14/15, and had right-sided long tract signs with aphasia. NIHSS was calculated to be 12. Urgent electrocardiogram (ECG) revealed acute inferior-posterior and right ventricular ST-elevation myocardial infarction (STEMI), a non-contrast CT (NCCT) of the brain revealed acute ischemic stroke in the right centrum semiovale with multiple lacunar infarctions in the right temporoparietal region. With the patient presenting out of the thrombolytic window for AIS and in the absence of facilities for thrombectomy for AIS in the hospital, the patient's subsequent management was taken with discussions involving the cardiology and neurology teams. The risk of secondary cerebral hemorrhage was presumed to be high from peri-PCI antithrombotic therapy or thrombolysis for AMI. Hence patient was managed conservatively, initially with a single antiplatelet (aspirin), a statin, meticulous multiparameter monitoring, oxygen supplementation, anti-anginal, blood pressure, and diabetic control. With a subsequent NCCT brain taken at 48 hours showing small extension of the size of the infarct, and with an improvement of the level of consciousness, dual antiplatelets (addition of clopidogrel) and LMWH therapy sequentially started and continued while actively monitoring for cerebral complications. The disease was later complicated with hypotension due to right ventricular infarct, which was fluid responsive, and ischemic hepatitis requiring N-acetyl cysteine therapy. Though the patient did not receive the ideal management and was offered the best possible management at that point, she showed a favorable outcome. 2D echocardiogram showed moderate systolic dysfunction without ventricular clots, and modified Rankin score was 3 on discharge. Early outpatient secondary PCI and passive physiotherapy-based neurorehabilitation were arranged.

Conclusion

CCI management should be individualized according to the clinical circumstances and the vascular territories involved in each organ. The selection of treatment is limited by the safety of antithrombotic

therapy against the extent of cardio cerebral injury and the availability of choices for endovascular interventions. Further evidence on management needs to build up for a better clinical outcome.

Introduction

The term cardio-cerebral infarction (CCI) was first introduced in 2010 to describe the simultaneous occurrence of acute ischemic stroke (AIS) and acute myocardial infarction (AMI) either at the same time (simultaneous or synchronous) or one following the other (metachronous) (1). It is an infrequent occurrence compared to isolated AIS or AMI, poses a management challenge for physicians, and carries an increased mortality risk (2). The therapeutic time window of both conditions is narrow and different, and the use of antiplatelets, anticoagulants, peri-percutaneous coronary interventional (PCI) ant-thrombotic agents, and the recommended doses of fibrinolytic agents for the acute management of AMI may increase the risk of hemorrhagic conversion of AIS. At the same time, a delay in treatment can result in permanent, irreversible damage to the infarcted area of the heart and brain, making CCI a challenging medical emergency to manage, particularly in a setting with limited resources. (3, 4). Furthermore, the rarity of the entity limits the availability of robust evidence for management. The presented case and the review of relevant literature explore the possibilities of CCI management.

Case Presentation

A 59-year-old Sinhalese female with poorly controlled type 2 diabetes mellitus, hypertension, and dyslipidemia resulting from non-adherence of medication use, presented to a peripheral base hospital in Sri Lanka (primary level of care) with sudden onset right-sided upper and lower limb weakness associated with slurring of speech, mouth deviation, vertigo, and emesis of yellowish content with undigested food particles. On direct inquiry, she complained of an intermittent, vague anterior chest pain over the preceding 12 hours that worsened to agonizing angina pectoris associated with autonomic symptoms, presyncope, and sweating within a few minutes of the acute neurological event. She had no fever, cough, pleurisy, palpitations, dyspnea, orthopnea, paroxysmal nocturnal dyspnea, headache, syncope, or suggestive features for deep vein thrombosis.

Her poor insight about the underlying comorbidities and prevailing social restrictions associated with the COVID-19 pandemic had made her non-adherent to treatments during the previous three months. Nevertheless, she did not have any history of ischemic cardiac or cerebral vascular events. Her family history was negative for any acute premature cardiac or cerebrovascular events. She was a non-smoker, non-alcoholic, consumed a high fat and salt content diet, and led a sedentary lifestyle pattern.

On arrival at the primary level of care, approximately 2 hours from the neurological event, she was ill, and in pain with a temperature of 36 ˚C, of GCS of 13/15, pulse rate of 90/minute with a regular rhythm, blood pressure of 240/130 mmHg, a left-sided pyramidal weakness of grade 3/5 and aphasia. ECG changes were suggestive of an acute inferior ST-elevation myocardial infarction (STEMI). Intravenous morphine 3 mg and metoclopramide 10 mg, supplemental oxygen via face mask was given, and a nasogastric tube

was placed. An infusion of glyceryl trinitrate (GTN) at 10ug/kg was commenced. Subsequently, she was transferred via ambulance to the tertiary care center located 1 ½ hour away for an NCCT brain and further care.

She arrived at the emergency treatment unit of the tertiary care center approximately 6 ½ hours from the neurological event. The physical examination at the tertiary care center presentation revealed a pulse rate of 79 beats per minute (regular), blood pressure of 182/102 mmHg, respiratory rate of 20 cycles per minute, and SpaO2 of 97%. The eye-opening was spontaneous; she obeyed commands; however, she was confused, giving rise to a GCS of 14/15. Bilateral pupils were equal in size (3 mm) and reactive to light. Neurological examination of the limbs revealed spastic hemiparesis with power MRC grade of 3, hyperreflexia, positive Babinski sign of right upper and lower limbs, and a right-side upper motor type facial nerve palsy. Neurological examination of the left upper and lower limbs was unremarkable. The sensorium and the coordination of the limbs were difficult to assess, given the patient's debilitating condition. The initial NIHSS was at 12. The rest of the cardiovascular, respiratory, and abdominal system-related examinations were unremarkable.

A repeated ECG revealed inferior and new additional posterior and right ventricular STEMI (Figure 1). Non-contrast CT (NCCT) of the brain showed a focal hypodense lesion in the right centrum semiovale with multiple lacunar infarctions in the right temporoparietal region (Figure 2), suggesting an AIS.

Opinions of the cardiology team and the neurology team were taken. Thrombolysis and mechanical thrombectomy for AIS was not indicated as the patient presented beyond the recommended time window. Also, an adequate facility for mechanical thrombectomy was unavailable in the institution. Coronary revascularization by the primary percutaneous coronary intervention (PCI) was deemed impossible because peri-procedural heparinization was presumed to carry an increased risk of a secondary cerebral hemorrhage. Therapy with fibrinolytic agents given at doses recommended for thrombolysis in AMI was contraindicated in a setting of a recent stroke.

Subsequently, the patient was transferred to the high dependency unit of a general medical unit for medical management of the acute STEMI and acute ischemic stroke (CCI). The initial clinical decision was to manage with a single antiplatelet (aspirin 75 mg nocte) and a statin (atorvastatin 40 mg nocte) sublingual glyceryl trinitrate 0.5 mg as needed and ISMN 30mg mane, considering the possible risk of intracranial bleeding. For pain management, intravenous morphine 2.5 mg and metoclopramide 10 mg were given as needed. Additional general and supportive care included oxygen supplementation during the initial hours, timely nasogastric enteral feeding, urinary catheterization, and avoidance of constipation with stool softeners. Troponin I level on admission was 57.1 ng/mL, which gradually came down to 26.1 ng/mL by day three. The 2D echocardiogram revealed global wall hypokinesia with an ejection fraction of 35%. The patient was neurologically non-progressing during the first two days of admission, and the repeat brain NCCT scan performed 48 hours after the AIS revealed an extension of the cerebral infarction to the right frontal region (Figure 3).

After discussing with the cardiology team, we started clopidogrel 75 mg nocte and a total of 10 doses of subcutaneous enoxaparin 60 mg 12 hourly along with bisoprolol 1.25 mg mane and enalapril 1.25 mg nocte. With that, the patient had a remarkable clinical improvement. Troponin I level came down to 6.96 ng/mL by day seven of admission. However, after ten days of admission, the repeat ECG showed q waves in previously affected territories, and T inversions in anterior and lateral chest leads (Figure 4).

On day one, the patient's clinical picture was complicated with fluid responsive hypotension, possibly due to right ventricular infarction and ischaemic hepatitis. On admission, the liver enzymes were high; alanine aminotransferase (ALT) of 1067.5 U/L, aspartate aminotransferase (AST) of 1088.9 U/L. Intravenous N-acetylcysteine (NAC) regimen was prescribed at 150 mg/kg with 5% dextrose over 15 mins, followed by 50 mg/kg over 4 hours and 100 mg/kg after that. The levels came down to ALT 226.4 U/L and AST 142.1 U/L by two days, and the NAC infusion was withheld. However, after another two days, the liver enzyme levels were again elevated to ALT 569.9 U/L and AST 180 U/L, and another NAC infusion was started 100 mg/kg 24 hours, and the ALT level came down to 196.4 U/L with an AST level of 66.6 U/L. On admission, the alkaline phosphatase level was 121.5 U/L (44 – 147) with a total bilirubin level of 33.94 µmol/L (1.71 – 20.5), direct bilirubin 24.22 µmol/L and indirect bilirubin 9.7 µmol/L.

On admission, the capillary blood sugar level was 406 mg/dL, with positive urine ketone bodies and was treated with intravenous infusion of soluble insulin 5 units per hour, which was gradually tailed down over a day and converted to soluble subcutaneous pre-meal insulin 10 units three times a day, following which she had a satisfactory glycemic control.

There was initial hematological and biochemical evidence of infection; white cell count was 23.26 × 10^9/L with neutrophil predominance, blood picture showing evidence of bacterial infections, and elevated inflammatory markers; c-reactive protein 119.6 mg/dL and erythrocyte sedimentation rate of 49 mm per hour. However, the urine culture and blood culture showed no growth, and the urine full report only showed 6 to 8 pus cells per high power field. However, there was no clinical evidence of any other infective focus. Nevertheless, a broad-spectrum antibiotic cover with intravenous meropenem 1 g 8 hourly for 7 days was given until the inflammatory markers came down to baseline.

By day seven, the patient had a remarkable clinical improvement. By day five, she could sit on the bed and walk with support by day seven. The nasogastric tube was removed on day five, and oral feeds were commenced as she showed good oral tolerance. The cognitive impairment was minimal, and she was discharged after eleven days of admission with a plan of reviewing at the outpatient clinic. A routine coronary angiogram was arranged to be done in a few months. Outpatient neurorehabilitation was also arranged. Modified Rankin score at discharge was 3.

Discussion

AIS and AMI are life-threatening medical conditions that carry a grave prognosis not addressed promptly (5). The association between both conditions was recognized a few decades ago from several studies (2,

6), and the term “cardio-cerebral infarction (CCI)” was introduced, considering that there exists a possible relationship between both pathologies rather than being just a mere coincidence (1). CCI occurs at the same time (simultaneous or synchronous) or one following the other (metachronous) (1). Most studies are on metachronous CCIs, which are described to have an incidence ranging from 0.9–12.7%, and the occurrence of AIS and AMI are within 72 hours of each other in all these studies (2, 6, 7). Whereas synchronous CCIs are rare with an incidence of 0.009% (8).

There are several pathogenic mechanisms of CCIs reported in the literature. One mechanism is the cerebral and coronary vascular embolism from left ventricular mural thrombi formed due to severely hypokinetic ventricular myocardium, especially in anterior or apical wall AMI (9, 10). These thrombi have a significant risk of embolization to the cerebral and coronary circulation (11). Another mechanism is that the sudden hemodynamic compromise in patients with long-standing history of hypertension presenting with AMI may reduce blood flow to water-shed areas of the brain and subsequent infarction, mainly if there is a concomitant failure of blood pressure auto-regulatory mechanisms (1). The third possible mechanism is the extension of an ascending aortic dissection to the coronary ostia and a subsequent extension to the carotid or the vertebral and basilar arteries (2). Another potential mechanism of a CCI is an AIS involving the left insular cortex, whose damage is thought to impair sympathovagal balance resulting in cardiac arrhythmias and wall motion abnormalities (12).

Similarly, adrenergic surge associated with AIS may result in catecholamine-induced myocardial stunning, a common cause of stress-induced cardiomyopathy (Takotsubo syndrome) that may mimic ST-elevation AMI, and in turn, favors the formation of an intracardiac thrombus that may embolize to the cerebral and coronary arteries (13). As in our case, right insular infarctions are thought to cause parasympathetic overactivity and paroxysmal atrial fibrillation, leading to acute coronary embolism (14). We suggest that a plausible mechanism of simultaneous CCI in the reported case was primarily due to an AIS in the right-middle cerebral artery territory, including the right insular region leading to transient arrhythmia and subsequent coronary embolism.

CCI is associated with increased mortality risk and is a management challenge for physicians. Both AMI (especially STEMI) and AIS have a narrow therapeutic time window, and a delayed intervention of one infarcted territory may result in permanent, irreversible morbidity of the other and even death (2). At the same time, antiplatelet therapy, anticoagulants used in coronary intervention, and fibrinolytic for AMI increase the risk for hemorrhagic conversion of AIS, and recommended doses of thrombolytic in AIS increases the risk of cardiac wall rupture in the setting of AMI (3, 15–17). There are no clinical trials or evidence-based guidelines for this therapeutic dilemma, possibly due to its rarity. The consensus on stroke management states that thrombolytic in AIS is relatively contraindicated if there is AMI within the past 3 months; however, American heart association guidelines give only level C evidence for this and recommend further studies (18). Although some studies have reported a higher risk of cardiac rupture with thrombolytic therapy (19), the risk is minimal (approximately 1%), according to several large-scale studies (17, 20, 21). Our patient might have been a possible candidate for thrombolysis with a tissue

plasminogen activator (with an NIHSS of 12) if she had presented within the window period (4.5 hours) for thrombolysis.

Several cases reported in the literature describe variable outcomes to the omission or commission of thrombolysis of endovascular interventions, highlighting the need to individualize treatment in patients presenting with CCI (1, 7, 22, 23). Therefore, the ideal management of simultaneous CCI is a treatment strategy that benefits vascular territories of both organs.

The presentation of AMI is an essential factor in deciding the management of CCI, while STEMI poses the most significant management challenge. Thrombolysis, approved for the acute management of both conditions, has been suggested as the best approach to treating CCI if there is no contraindication, and both presentations are within the time frame for the administration of a thrombolytic (2). However, this has not been studied in clinical trials nor supported by any societal guidelines. The challenge to this management approach is the different dosage and duration of thrombolytic administration recommended to treat acute infarction of these vascular territories (18, 24). There is an increased risk of hemorrhagic conversion of AIS when thrombolytics are administered at higher doses (25), and administration of a lower dose than recommended of a thrombolytic for AMI may be considered under-dosing (8).

Another management approach is a combined endovascular approach with PCI for AMI and thrombectomy devices for AIS (8). However, the use of adjunctive antiplatelet therapy with PCI poses a significant risk of bleeding with endovascular treatment for AIS (2). There are presently no clinical trials evaluating the safety, outcomes, and the role of dual antiplatelet therapy with endovascular treatment for AIS, but a retrospective study conducted by Broeg-Morvay et al., evaluating the use of aspirin, IV-tPA (tissue plasminogen activator), and endovascular therapy versus IV-tPA and endovascular therapy without aspirin showed no increase in intracranial hemorrhage between the groups. Outcomes at 3 months did not differ (26). However, further trials are needed to assess the safety of antiplatelet therapies with cerebral endovascular procedures.

There is an increased risk of intracranial hemorrhage with the use of antiplatelets, especially with dual antiplatelet therapy along with IV-tPA, shown by several studies, but the use had not adversely influenced the overall clinical outcome (27–29). The increased risk of intracranial hemorrhage with these antiplatelets is likely balanced by the beneficial effect of increased reperfusion or decreased risk of vessel re-occlusion. Tse antiplatelets may not by themselves increase the risk of intracranial hemorrhage but makes one worse if it occurs (28). Therefore, a reasonable approach to the acute management of CCI is a combined treatment of both vascular territories with the administration of a suitable fibrinolytic, followed by PCI if indicated. Intravenous Alteplase (r-tPA) has been previously utilized in one case of CCI, resulting in a favorable outcome (2). However, the utility of intravenous tenecteplase (TNK) has not been explored previously. The absence of subsequent neurological improvement may require a cerebral angiogram and a relevant endovascular procedure. A facility for such is not available in the country at the moment.

Performing an angiogram gives an added advantage of diagnosing an aortic dissection extending to both the coronary ostia and the carotid or vertebral and basilar arteries (2).

Complicating the clinical condition, our patient had an acute liver injury. Elevation of the liver zymes is common in STEMI (30), possibly due to ischemia following a reduced cardiac output (31). Elevated liver enzyme levels in patients with the acute coronary syndrome is a poor prognostic factor (32). In the setting of myocardial infarction, congestive heart failure should always be considered as a possible cause of acute liver failure (33). Our patient showed elevated liver enzyme levels of more than 1000 U/L, where we suspected acute liver injury, possibly due to right ventricular failure following right ventricular STEMI. With treatment of N-acetyl cysteine, the liver enzyme levels were normalized within a few days.

The scarcity of resources for mechanical thrombectomy and low-risk primary PCI n our setting was an important drawback in managing the reported case. Nevertheless, she made a good clinical recovery with initial single antiplatelet therapy with statin and gradual introduction of dual antiplatelet therapy with anticoagulation with LMWH. Further clinical trials and studies are needed to build up the consensus of evidence for the management of synchronous CCI targeting a better clinical outcome. Such trials, however, are essentially limited by the rarity of the entity. At the current time, it is reasonable to justify the management of synchronous CCI to be highly individualized, considering the safety, efficacy, and availability of therapeutic options and utilizing an effective multi-disciplinary approach.

Conclusion

Simultaneous CCI is a rare vascular event involving two vital organs with a poor prognosis and is a therapeutic challenge for physicians. The management of CCI should be individualized according to the clinical circumstances, the vascular territories involved in each organ, the safety of administered medications, and the resources available. Dual antiplatelet therapy with anticoagulation is a possible management option for synchronous CCIs presenting out of the thrombolytic window in a center with limited resources. However, thrombectomy for AIS and PCI for AMI may be ideal in such a situation. Further clinical trials and studies are needed to build up a consensus of evidence-based management of CCI for a better clinical outcome.

Abbreviations

AIS
Acute Ischemic Stroke
AMI
Acute Myocardial Infarction
CCI
Cardio Cerebral Infarction
ECG
Electro Cardio Graph

LMWH
Low Molecular Weight Heparin
NCCT
Non-Contrast Computed Tomography
NIHSS
National Institute of Health Stroke Score
PCI
Percutaneous Coronary Intervention
SpaO2
Arterial Oxygen Saturation
STEMI
ST-elevation Myocardial Infarction.

Declarations

Ethics approval and consent to participate

Not applicable

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials

The authors confirm that the data supporting the findings of this study are available within the article.

Competing interests

The authors declare that they have no competing interests.

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Authors' Contributions

All authors were involved in the management of the patient and generating the concept. All authors made an intellectual contribution and wrote the paper. All authors read and approved the final manuscript.

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Figures

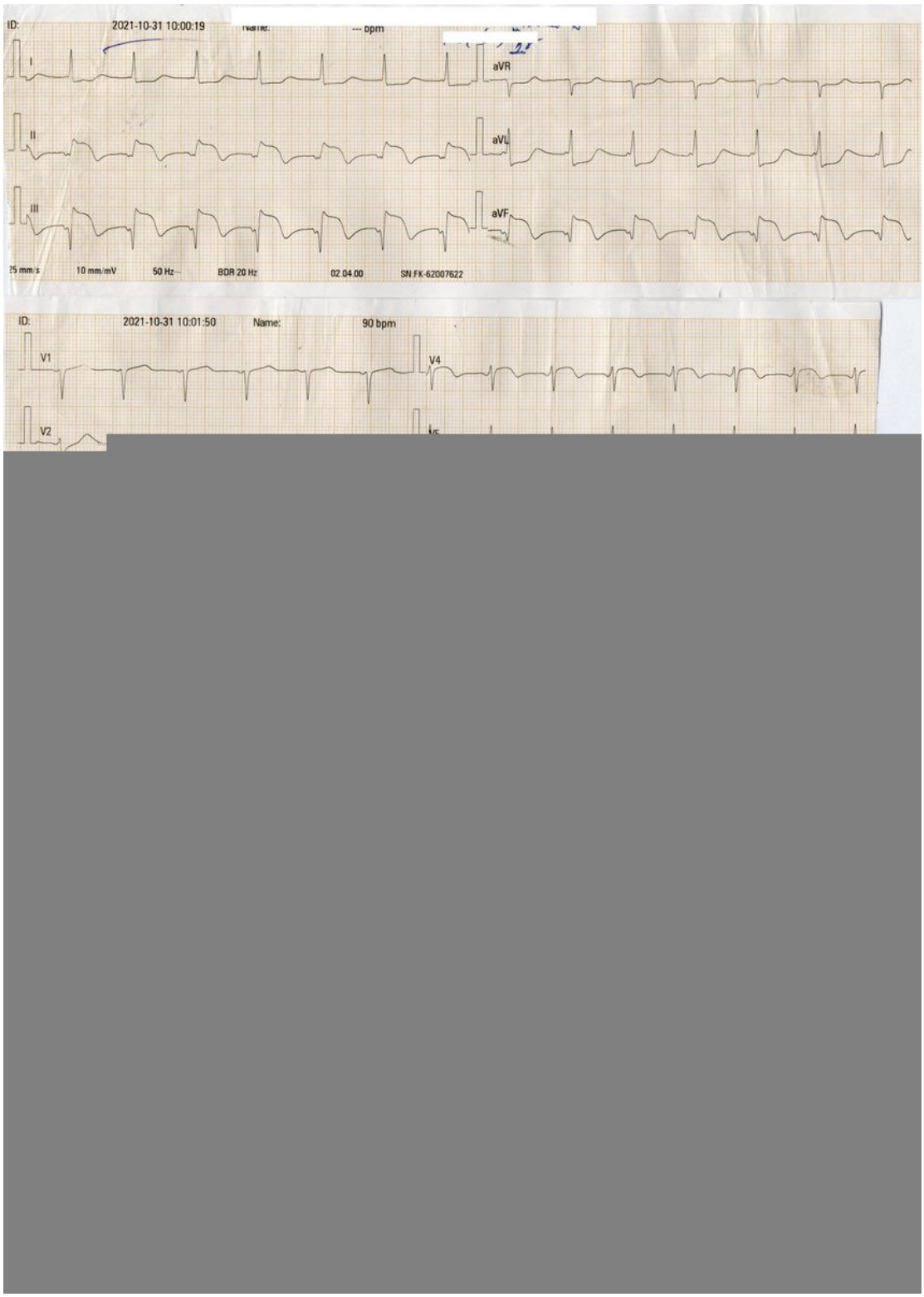

Figure 1

ECG showing ST elevations in II, III, aVF, V7, V8, V9, and V4R leads

Figure 2

NCCT brain showing a focal hypodense lesion in right centrum semiovale with multiple lacunar infarctions in the right parietal region

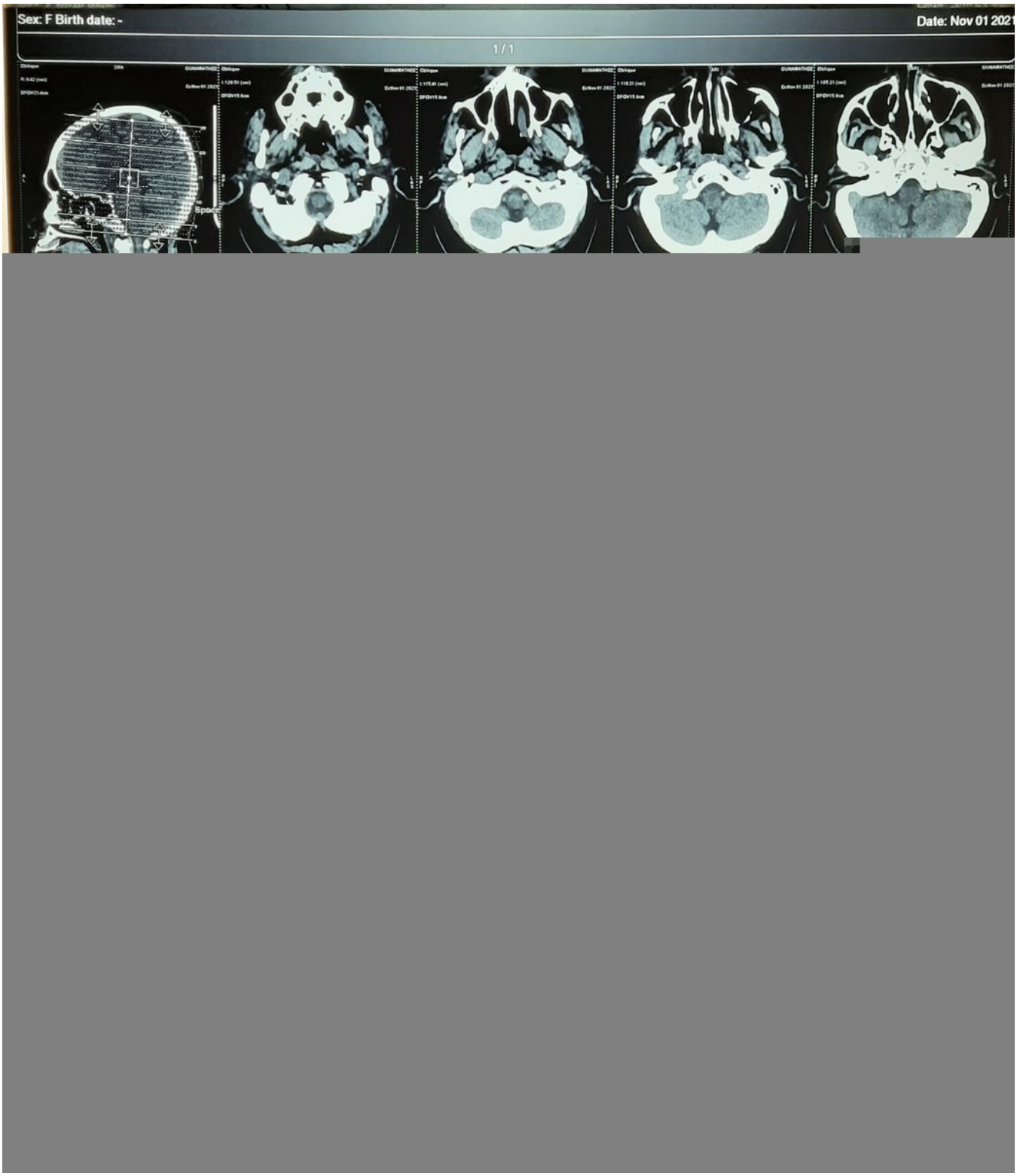

Figure 3

Legend not included with this version

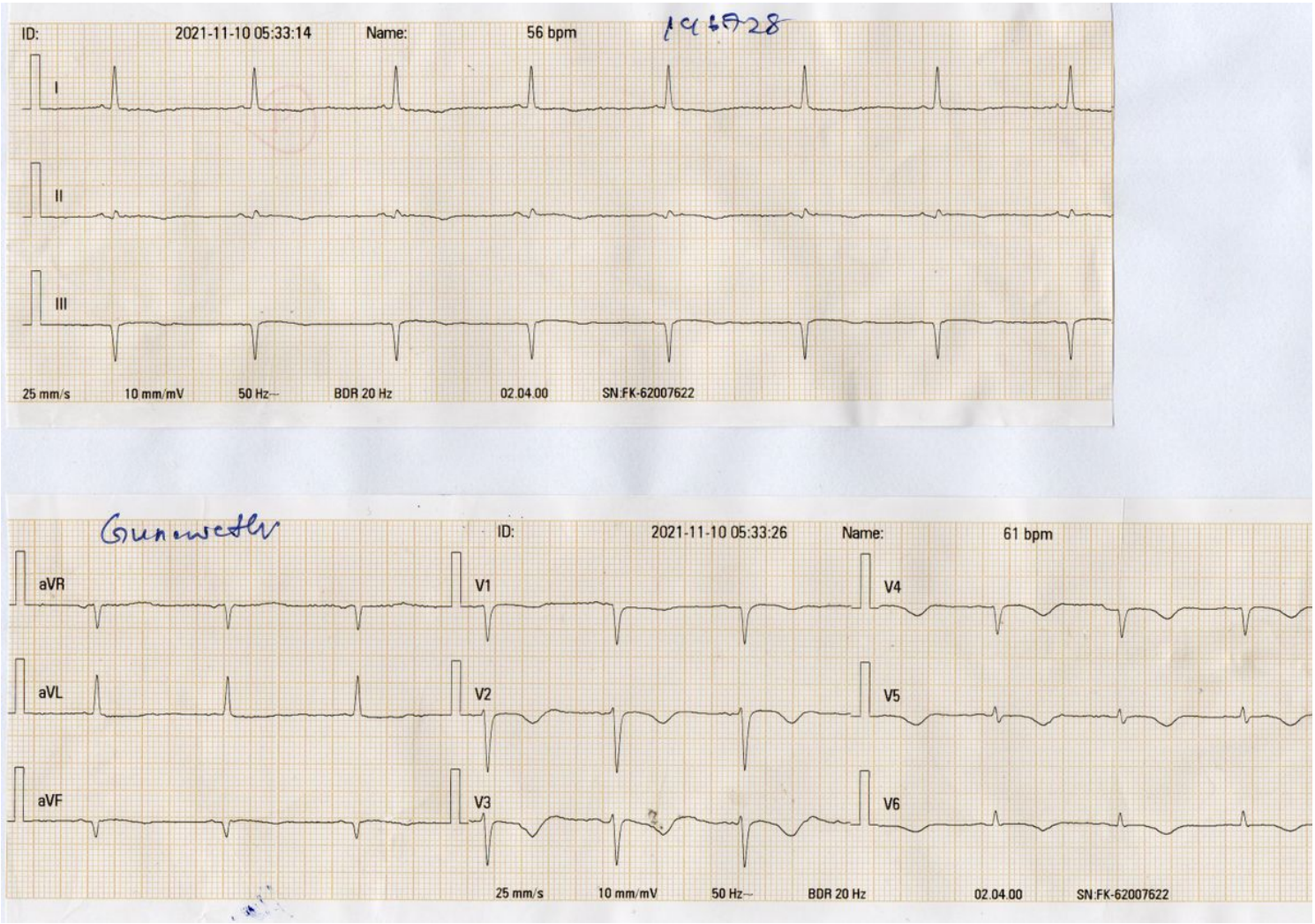

Figure 4

Legend not included with this version

Supplementary Files

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- CAREChecklist.docx