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Sustained Monomorphic Ventricular Tachycardia During Imatinib Therapy in Blast-Phase Chronic Myeloid Leukemia: A Case Report from a Resource-Limited Setting

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Case Report

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Abstract

Background: Imatinib has substantially improved the treatment of chronic myeloid leukemia (CML) and is generally associated with a favorable cardiovascular safety profile. Nevertheless, serious arrhythmias occurring during treatment may present a diagnostic challenge, particularly in patients with advanced disease and multiple potential contributing factors.

Case presentation: A 45-year-old woman with CML in blast phase developed sustained monomorphic ventricular tachycardia (VT) during treatment with imatinib. Bone marrow examination showed 66% blasts, and her clinical course was complicated by severe anemia and metabolic abnormalities. During the arrhythmic episode, her heart rate was approximately 198 beats/min and blood pressure was 93/61 mmHg. She remained conscious, with palpable pulses and preserved peripheral perfusion. The ventricular tachycardia persisted despite two intravenous amiodarone loading doses and subsequently converted to sinus rhythm following synchronized electrical cardioversion. No recurrence was documented during the subsequent observation period. The event occurred in the setting of advanced malignancy and other potential clinical contributors, making its cause difficult to determine.

Conclusion: This case illustrates the challenge of evaluating ventricular tachycardia in a patient with advanced CML receiving imatinib. A treatment-related arrhythmia is a possibility, but the clinical context includes several competing contributors and does not establish a definitive cause. Assessment of serious arrhythmias during cancer treatment should consider medication exposure, the underlying disease, and potentially reversible clinical factors.

Key Clinical Message

Tyrosine kinase inhibitors (TKIs), including imatinib, have been associated with rare but potentially serious ventricular arrhythmias. Patients receiving TKI therapy may benefit from baseline cardiovascular assessment, ECG monitoring when clinically indicated, attention to potential drug interactions, and prompt evaluation of new cardiac symptoms or arrhythmias.

Introduction

Chronic myeloid leukemia (CML) is a blood cancer driven by the BCR::ABL1 fusion gene, which forms when parts of chromosomes 9 and 22 exchange places in a process known as the Philadelphia chromosome translocation [1]. This fusion produces an abnormal protein with persistent tyrosine kinase activity, leading to uncontrolled growth of myeloid cells. CML is relatively uncommon, with European population-based studies reporting approximately 0.7-1.0 new cases per 100,000 people each year. It is more frequently diagnosed in adults and occurs more often in men than in women [1]. The introduction of tyrosine kinase inhibitors (TKIs), which target the abnormal BCR::ABL1 protein, has greatly improved treatment outcomes and allowed many patients to live for years with the disease [1, 2].

The burden of CML differs across countries and regions. In Ethiopia, the estimated incidence of CML was reported to be 1.98 per 100,000 population in 2017 [3]. Although epidemiologic estimates help describe the distribution of CML, they do not fully capture the clinical challenges faced by individual patients or the demands of managing the disease in different healthcare settings.

TKIs have changed the outlook for patients with CML, but treatment still requires attention to potential complications. Imatinib was one of the first drugs to target BCR::ABL1 and has generally demonstrated a favorable cardiovascular safety profile compared with some other TKIs, although cardiovascular risks differ among agents [2, 4–6]. Sustained ventricular tachycardia is a potentially life-threatening arrhythmia, and its occurrence during imatinib therapy is uncommon and may be difficult to interpret in patients with advanced malignancy and multiple competing risk factors. This report describes sustained monomorphic ventricular tachycardia occurring during imatinib therapy in a patient with blast-phase CML and no previously diagnosed cardiac disease. The case highlights the challenges of evaluating serious arrhythmias in patients with advanced malignancy, where medication exposure, metabolic abnormalities, and disease-related factors may all contribute to arrhythmogenesis.

Case Presentation

History

A 45-year-old woman was referred for assessment of progressive abdominal enlargement and swelling of both lower limbs. She reported that the abdominal swelling had been present for about two months and had gradually become more noticeable. During the same period, she had become increasingly fatigued and had lost her appetite and weight. She also described a dry cough, reduced tolerance for physical activity, shortness of breath on exertion, rapid breathing, and episodes of palpitations. She did not report orthopnea or paroxysmal nocturnal dyspnea in the initial history. Joint and muscle aches were also documented. In the days before the first emergency assessment, the cough and breathlessness had become more prominent. She reported no history of diabetes mellitus, hypertension, or known cardiac or renal disease, and no drug allergies were reported.

The initial assessment raised concern for a hematologic malignancy after marked leukocytosis and circulating myeloblasts were identified. The referral information described approximately 25% peripheral myeloblasts, prompting concern for advanced disease and progression of the underlying chronic myeloid leukemia. The patient was also assessed for symptomatic anemia, fluid overload, and possible respiratory infection.

Bone marrow examination on 17 April 2026 showed 66% blasts. BCR::ABL1 testing was positive on 4 May, confirming CML in blast phase. Hydroxyurea and allopurinol were started on 19 April. Imatinib 300-mg oral capsules were prescribed twice daily on 8 May 2026 for an initial one-month course. The VT occurred on 8 September, 123 days after imatinib was initiated. Imatinib was interrupted from 9 to 29 June because of cytopenias and was subsequently restarted. She required repeated hospital

assessments and blood-product transfusions. She was admitted again on 7 September 2026 with fever, gum bleeding, cough, headache, reduced appetite, diarrhea, abdominal pain, and severe lower-back pain radiating to the lower limbs. The back pain was intermittent but at times severe. No preceding trauma or bowel or bladder dysfunction was reported in the available notes. Weakness in both lower limbs progressed during the later admission. She died on 27 September 2026; the death certificate listed septic shock due to a chest focus as the immediate cause and CML as the underlying illness.

Physical Examination

At initial presentation

On arrival, she was tachycardic, with a recorded heart rate of approximately 120 beats/min and blood pressure around 99/63 mmHg. Respiratory rate was about 20 breaths/min, and oxygen saturation was initially recorded at 97%. Examinations documented pallor in several entries, although one early note described the conjunctivae as pink. The cardiovascular examination noted tachycardia, a systolic murmur, and an S3 gallop in one assessment. The chest was initially described as clear with good air entry. Marked hepatosplenomegaly and grade II pitting edema of the lower limbs were present. She was conscious and oriented, and no peripheral lymphadenopathy was documented.

During the later admission

Subsequent examinations varied with the course of the illness. She was described at times as pale and markedly wasted, with persistent hepatosplenomegaly and episodes of oral or gum bleeding. Examination of the back revealed tenderness around the L4–L5 region. Lower-limb power declined from approximately 4/5 to 2/5 bilaterally; sensation remained intact and deep tendon reflexes were 2/4. Later respiratory assessments documented bilateral crackles, tachypnea, and a need for supplemental oxygen by face mask. Peripheral edema recurred. During clinical deterioration, hypotension and a feeble pulse were recorded. She remained conscious in several of the documented assessments.

Investigations

Hematology and marrow assessment

The initial blood counts showed marked leukocytosis, severe anemia, and thrombocytopenia. Across early assessments, the white blood cell count was reported in the range of approximately 186–229 × 10^9/L, hemoglobin was about 6.4–6.9 g/dL, and platelet counts were markedly reduced, with values ranging from approximately 15 to 52 × 10^9/L in the available entries. Peripheral blood examination raised concern for a high blast burden. Bone marrow aspiration showed a markedly hypercellular marrow with suppression of erythroid and megakaryocytic lineages. Blasts accounted for 66% of nucleated marrow

cells. The marrow findings were interpreted as CML in blast phase. BCR::ABL1 testing was reported as positive, with a value of 100% in the record.

During later admissions, the blood counts fluctuated substantially. Severe anemia and thrombocytopenia persisted, with hemoglobin values repeatedly below 8 g/dL and platelet counts sometimes in single digits. Following periods of cytopenia, the white blood cell count rose markedly, reaching more than 100 × 10^9/L in subsequent results.

Biochemistry and microbiology

The record included elevated lactate dehydrogenase and uric acid during the later illness, alongside low albumin and abnormal liver-associated enzymes. On 8 September, sodium was 119.6 mmol/L and potassium was 3.73 mmol/L; hemoglobin was 7.2 g/dL that evening. Creatinine was 0.5 mg/dL on 7 September and 0.3 mg/dL on 9 September. Calcium was 8.0 mg/dL on 4 and 16 September and 9.0 mg/dL on 21 September. Magnesium was 0.90 mg/dL on 16 September, 1.23 mg/dL on 21 September, and 1.52 mg/dL on 23 September. A bedside glucose (RBS) was 164 mg/dL on the morning of 9 September. Cardiac troponin was less than 0.1 on 15 September, after the VT episode. Blood, urine, and sputum cultures were documented as showing no growth in one assessment. Hepatitis B surface antigen and hepatitis C testing were negative, and GeneXpert testing was recorded as not detected.

Abdominal imaging

Initial abdominal ultrasonography demonstrated hepatomegaly, with the liver measuring approximately 18 cm, and splenomegaly, with the spleen measuring approximately 15.5 cm. A later study described terminal ileal wall thickening over a short segment, with reduced peristalsis and loss of the usual bowel-wall appearance; the radiologic impression suggested typhlitis. Minimal pelvic fluid was also noted at that time. A subsequent ultrasound documented hepatosplenomegaly, a large amount of complex ascites, and dilatation of the inferior vena cava and hepatic veins with reduced respiratory variation, raising concern for elevated right-sided venous pressure or hepatic congestion.

Cardiopulmonary assessment

Echocardiography on 14 April 2026, before the VT episode, showed a dilated, hyperdynamic left ventricle with a reported ejection fraction of 65%, right atrial dilatation, mild tricuspid regurgitation, and an estimated pulmonary artery systolic pressure of approximately 45 mmHg. The inferior vena cava was dilated with less than 50% inspiratory collapse. The report described mild pulmonary hypertension and a hyperdynamic left ventricle; no other structural abnormality was reported.

Chest CT angiography early in the course demonstrated bilateral moderate pleural effusions, bibasilar atelectatic changes, peribronchial wall thickening, ground-glass opacities, and smooth interlobular septal thickening. The findings were considered compatible with an infectious or aspiration-related process,

with a possible contribution from fluid overload. A later CT pulmonary angiogram showed no filling defect in the main, lobar, or visualized pulmonary arteries. It again demonstrated bilateral perihilar and peribronchovascular ground-glass opacities, diffuse smooth septal thickening, pleural effusions, and adjacent compressive atelectasis. The radiologic impression favored pulmonary edema in the setting of anemia, while noting that superimposed pneumonia could not be excluded.

Neurologic and spinal imaging

CT of the thoracolumbar spine was unremarkable. MRI of the lumbar spine demonstrated degenerative changes at L4-L5 with mild spinal canal and bilateral neural foraminal narrowing. Subsequent brain MRI and MR venography showed minimal extra-axial hemorrhage along the posterior fossa convexities, without other major intracranial abnormalities.

Initial treatment

Following the initial hematological assessment, treatment was initiated with hydroxyurea for cytoreduction and allopurinol for tumor lysis syndrome prophylaxis, together with intravenous hydration and supportive care. Antibiotic therapy, including cefepime and vancomycin, was administered for suspected infection. Supplemental oxygen and other supportive measures were provided as clinically indicated.

Imatinib 300 mg orally twice daily was initiated on 8 May 2026 following confirmation of BCR::ABL1 positivity. However, treatment was interrupted from 9 to 29 June 2026 because of cytopenias and was subsequently restarted at the same dose on 29 June 2026. The patient continued to receive imatinib until developing sustained monomorphic ventricular tachycardia on 8 September 2026, approximately 123 days after initial treatment initiation and 71 days after its reintroduction.

Management of cytopenias and recurrent admissions

During subsequent admissions, the patient's clinical course was complicated by persistent anemia and thrombocytopenia, requiring repeated blood-product transfusions and supportive management. She was readmitted on 7 September 2026 with fever, gum bleeding, cough, headache, reduced appetite, diarrhea, abdominal pain, and severe lower-back pain radiating to both lower limbs.

Management during this admission included broad-spectrum antimicrobial therapy with meropenem and vancomycin, supplemental oxygen, transfusion support, analgesia, and close monitoring of fluid balance and urine output. Intravenous furosemide was administered and adjusted according to her volume status. Hydroxyurea was also reintroduced, with dose adjustments in response to rising leukocyte counts. On 8 September 2026, the patient developed sustained monomorphic ventricular tachycardia while receiving imatinib therapy. Following the arrhythmic episode, imatinib was held because of cytopenia and concern about a possible treatment-related cardiovascular adverse event. The VT persisted despite two intravenous amiodarone loading doses and was subsequently terminated by synchronized electrical cardioversion. Metoprolol tartrate 25 mg orally four times daily was ordered on the morning of 9 September. No further episodes of VT were documented during the available follow-up.

Ventricular Tachycardia Episode

On 8 September 2026, the patient developed a regular wide-complex tachycardia that was interpreted by the treating team as sustained monomorphic ventricular tachycardia (VT), with a heart rate of approximately 198 beats/min and a QRS duration of 146 ms. At assessment, her blood pressure was 93/61 mmHg. She remained conscious (GCS 15/15), with palpable pulses, warm extremities, and a capillary refill time of less than two seconds; the contemporaneous assessment described her as hemodynamically stable. No ischemic changes were reported on the ECG.

Intravenous amiodarone was administered as a 150-mg loading dose, followed shortly thereafter by a second 150-mg loading dose. The heart rate remained approximately 198 beats/min, and the ventricular tachycardia persisted despite both loading doses. An amiodarone infusion was subsequently initiated, consisting of 360 mg in 500 mL of D5W over 6 hours, followed by 540 mg in 1,000 mL of D5W over 18 hours. Because the tachycardia persisted despite amiodarone therapy, synchronized electrical cardioversion was performed, resulting in conversion to sinus rhythm.

The post-conversion ECG showed a QT interval of 324 ms and a corrected QT interval of 446 ms. Metoprolol tartrate was started the following morning. Subsequent notes documented resolution of the VT, with no recurrence recorded during the available follow-up. ECG recordings obtained during the VT episode and after conversion are presented in Fig. 1.

Clinical Course and Outcome

Despite hematology-directed treatment and repeated supportive interventions, the patient experienced a complicated course marked by persistent severe anemia and thrombocytopenia, periods of profound cytopenia, and subsequent marked leukocytosis. Recurrent fever and respiratory symptoms prompted treatment for suspected infection. Imaging showed pleural effusions and diffuse bilateral pulmonary changes, with pulmonary edema favored on the later CT angiogram, although concomitant pneumonia could not be excluded. She also developed abdominal findings suggestive of typhlitis, progressive lower-limb weakness in the context of lumbar degenerative changes, and increasing fluid accumulation with ascites.

In the final phase of her illness, she developed worsening respiratory distress and hemodynamic instability despite oxygen therapy, antimicrobial treatment, transfusion support, and other supportive measures. She died on 27 September 2026. The death certificate recorded septic shock attributed to a chest focus as the immediate cause of death, with CML as the underlying illness. The major diagnostic, therapeutic, and clinical events during the patient's course are summarized in Table 1.

Table 1
Timeline of major clinical events, ventricular tachycardia management, and outcome

Timeline	Clinical event	Key findings / management
Day 0	Initial presentation and diagnosis of CML in blast phase.	Bone marrow showed 66% blasts; BCR::ABL1 positivity subsequently confirmed.
Day 2	Cytoreductive and supportive treatment initiated.	Hydroxyurea and allopurinol started, with hydration and supportive care.
Day 19	Imatinib initiated.	Imatinib 300 mg twice daily started.
Days 51–71	Imatinib interruption for cytopenias, then reintroduction.	Twenty-day interruption because of cytopenias; treatment subsequently restarted at the same dose.
Day 123	Hospital readmission.	Admitted with fever, gum bleeding, cough, headache, reduced appetite, diarrhea, abdominal pain, and severe lower-back pain.
Day 124	Sustained monomorphic VT developed during imatinib therapy.	VT occurred approximately 123 days after imatinib initiation and 71 days after treatment reintroduction.
+ 50 minutes	Two intravenous amiodarone loading doses administered.	VT persisted despite treatment.
+ 3 hours	Continuous amiodarone infusion initiated.	Tachycardia remained present.
+ 3–4 hours	Synchronized electrical cardioversion performed.	Successful conversion to sinus rhythm.
Day 124	Imatinib held.	Imatinib held because of concurrent cytopenias and concern for a possible treatment-related cardiovascular adverse event.
Day 125	Post-event management.	Metoprolol initiated; no recurrence of VT documented.
Days 125–142	Subsequent clinical deterioration.	Ongoing supportive care and management of complications.
Day 142	Death	Septic shock due to a chest focus listed as the immediate cause of death; CML listed as the underlying illness.

Discussion

BCR::ABL1 tyrosine kinase inhibitors (TKIs) have substantially improved outcomes in chronic myeloid leukemia (CML), but their cardiovascular safety profiles differ. Imatinib generally has a more favorable cardiovascular profile than some later-generation TKIs. Nilotinib and ponatinib have been particularly associated with arterial occlusive events, while dasatinib is associated with pleural effusion and

pulmonary arterial hypertension [4–6]. Nevertheless, a generally favorable safety profile does not exclude rare serious arrhythmias. Experimental studies have demonstrated concentration-dependent inhibition of the hERG potassium channel by imatinib, providing a possible biological basis for altered cardiac repolarization, although the clinical relevance of this finding at therapeutic doses remains uncertain [7]. Importantly, the present episode was monomorphic VT rather than torsades de pointes, and the post-conversion QTc was 446 ms; therefore, a QT-mediated mechanism cannot be established.

Evidence linking imatinib to ventricular arrhythmias remains limited. A published case report described ventricular fibrillation in a patient receiving imatinib, followed by another episode during subsequent dasatinib treatment, raising concern about a possible association between BCR::ABL1 inhibition and ventricular arrhythmias [8]. In a large WHO pharmacovigilance analysis, several anticancer agents were associated with disproportionate reporting of long-QT events or ventricular arrhythmias, although such analyses cannot establish incidence or individual drug causality [9]. More recently, a FAERS-based analysis identified cardiac-arrhythmia reporting signals for selected BCR::ABL1 inhibitors, particularly nilotinib, asciminib, and ponatinib; imatinib did not demonstrate a positive disproportionality signal in that analysis [10]. These findings suggest that serious ventricular arrhythmias are not an established common complication of imatinib, but isolated events remain clinically important and warrant assessment.

In this patient, sustained monomorphic VT occurred on 8 September 2026, approximately 123 days after imatinib initiation and 71 days after its restart following a temporary interruption for cytopenias. She was receiving imatinib when the arrhythmia occurred, although the exact time of the last administered dose was not documented. The drug was held after the episode because of concurrent cytopenias and concern about a possible treatment-related cardiovascular adverse event. The temporal relationship, experimental evidence of potential electrophysiological effects, and absence of documented recurrence during the subsequent observation period support considering imatinib as a possible contributor. However, the dechallenge observation is difficult to interpret because synchronized electrical cardioversion terminated the arrhythmia and metoprolol was initiated the following morning. The patient also had advanced blast-phase CML, severe anemia (hemoglobin 7.2 g/dL on the evening of the event), and marked hyponatremia (119.6 mmol/L). Potassium was 3.73 mmol/L, providing no evidence of significant hypokalemia. Magnesium and calcium measurements were obtained after the episode rather than contemporaneously, and the troponin result was also obtained later; therefore, these results cannot reliably exclude abnormalities or myocardial injury at the time of VT. The only available echocardiogram, performed on 14 April 2026 before the arrhythmia, showed preserved left ventricular ejection fraction (65%) but also right atrial dilatation, mild tricuspid regurgitation, and an estimated pulmonary artery systolic pressure of approximately 45 mmHg. Thus, the available findings do not establish a completely normal cardiac substrate. Overall, the association between imatinib and VT is best classified as **possible** according to the WHO–Uppsala Monitoring Centre causality assessment framework [11].

The episode also highlights the importance of prompt rhythm recognition and management in resource-limited settings. The patient presented with a heart rate of approximately 198 beats/min, a QRS duration

of 146 ms, and blood pressure of 93/61 mmHg, but remained conscious with palpable pulses and preserved peripheral perfusion. Despite two intravenous amiodarone loading doses, the VT persisted, prompting synchronized electrical cardioversion, which successfully restored sinus rhythm. Prompt termination of sustained monomorphic VT is appropriate even when initially tolerated because clinical deterioration may occur [12]. In settings where comprehensive cardiac investigations and continuous monitoring may be limited, careful ECG documentation, assessment of potentially reversible contributors, medication review, and timely access to cardioversion are particularly important. In the present case, further evaluation of potential arrhythmic mechanisms was limited by restricted access to advanced cardiac investigations and specialized cardio-oncology services. Baseline and follow-up ECG assessment and monitoring of electrolytes, hemoglobin, renal function, and potential drug interactions may also help identify patients at risk of treatment-associated cardiovascular complications [12, 13].

Conclusion

This case describes sustained monomorphic ventricular tachycardia occurring during imatinib therapy in a patient with chronic myeloid leukemia in blast phase and no known history of cardiac disease. The event raises concern for a possible treatment-associated arrhythmia, although the available clinical information must be evaluated alongside the patient's advanced malignancy, severe anemia, metabolic abnormalities, and other concurrent treatments. The case highlights the importance of prompt rhythm recognition, appropriate acute management, systematic assessment of potentially reversible contributors, and careful evaluation of suspected cardiovascular adverse events during targeted cancer therapy.

Abbreviations

ABL
Abelson proto-oncogene
AHA
American Heart Association
BCR
Breakpoint Cluster Region
CML
Chronic Myeloid Leukemia
CYP3A / CYP3A4
Cytochrome P450 3A / 3A4 enzyme
ECG
Electrocardiogram
ESC
European Society of Cardiology
FAERS
Food and Drug Administration Adverse Event Reporting System

FDA
Food and Drug Administration
GBD
Global Burden of Disease
hERG
human Ether-à-go-go-Related Gene
LQT / LQTS
Long QT / Long QT Syndrome
QTc
Heart rate-corrected QT interval
TdP
Torsades de Pointes
TKI
Tyrosine Kinase Inhibitor
UMC
Uppsala Monitoring Centre
VF
Ventricular Fibrillation
VT
Ventricular Tachycardia
WHO
World Health Organization
WHO
UMC-World Health Organization-Uppsala Monitoring Centre

Declarations

Ethics Approval and Consent to Participate

Ethical approval was not required for this single case report at our institution. Written informed consent to participate was obtained from the patient. Supporting documents are available upon request.

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 signed consent form is available for review by the Editor-in-Chief upon request.

Competing Interests

The authors declare that they have no competing interests.

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Author Contribution

N.M.A. contributed to manuscript writing, resource acquisition, and manuscript editing.A.A.W. contributed to patient management, conceptualization, manuscript writing, and resource acquisition. L.T.K. contributed to patient management, conceptualization, manuscript writing, and resource acquisition. A.M.A. contributed to manuscript writing, validation, and investigation. S.K.A. contributed to manuscript writing, validation, and investigation. R.L.L. contributed to manuscript writing, resource acquisition, and manuscript editing.Y.B.K. contributed to manuscript review, editing, and investigation. All authors read and approved the final manuscript

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Data Availability

The data supporting the findings of this case report are available from the corresponding author upon reasonable request. To protect patient privacy and confidentiality, individual-level clinical data are not publicly available.

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Figures

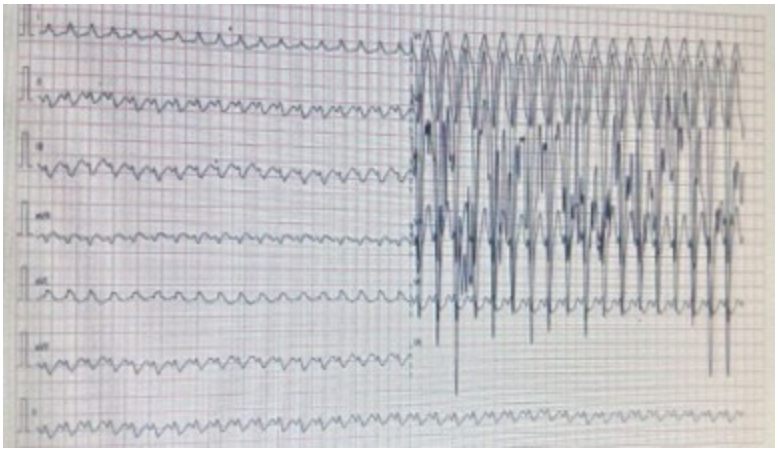


Figure 1

ECG during Ventricular Tachycardia