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ARTIKEL PENELITIAN

Hypokalemia Induced Arrhythmia during Pregnancy in Rural Area - A Case Report

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ABSTRACT

Hyperemesis gravidarum is a severe form of nausea and vomiting during pregnancy that leads to persistent fluid and electrolyte imbalances, with hypokalemia being among the most clinically significant complications. Hypokalemia disrupts normal cardiac membrane excitability and can precipitate lethal arrhythmias, including ventricular tachycardia, ventricular fibrillation, and torsade de pointes — rhythms that carry a substantial risk of sudden cardiac death if not promptly identified and treated. These life-threatening arrhythmias arise from hypokalemia-induced hyperpolarization of the cardiomyocyte resting membrane potential, which paradoxically heightens ventricular automaticity and creates a vulnerable electrophysiological substrate susceptible to malignant dysrhythmias. A 33-year-old multiparous woman at 29 weeks of gestation presented to the emergency unit with a three-day history of generalized weakness, fatigue, persistent nausea, vomiting exceeding five episodes per day, diffuse muscle cramping, and abdominal discomfort. On examination, she was tachycardic with an irregular pulse of 100 beats per minute and tachypneic at 24 breaths per minute. The initial electrocardiogram demonstrated ventricular bigeminy, and brief self-terminating runs of non-sustained ventricular tachycardia were subsequently observed — findings that signaled an evolving arrhythmogenic state with potential for progression to fatal ventricular arrhythmia. Laboratory investigations confirmed severe hypokalemia with a serum potassium level of 1.7 mEq/L. Two-dimensional echocardiography revealed no structural cardiac abnormalities with a preserved left ventricular ejection fraction of 60%. The patient was managed with intravenous potassium chloride replacement, empirical magnesium sulfate supplementation, Bisoprolol, and Methyldopa in the Intensive Care Unit. Serial electrocardiograms documented progression from ventricular bigeminy to trigeminy before complete resolution following serum potassium normalization to 3.4 mEq/L. This case demonstrates that hypokalemia secondary to hyperemesis gravidarum can induce lethal ventricular arrhythmias, and that favorable outcomes are achievable through early recognition and systematic management.

Keywords: Arrhythmia, Hypokalemia, Pregnancy, Hyperemesis, Ventricular Ectopy

ABSTRAK

Hiperemesis gravidarum adalah kondisi mual dan muntah yang berat selama kehamilan sehingga menyebabkan ketidakseimbangan cairan dan elektrolit secara terus-menerus dengan hipokalemia menjadi salah satu komplikasi yang paling signifikan secara klinis. Hipokalemia dapat mengganggu rangsangan membran jantung dan dapat memicu aritmia yang mematikan, termasuk takikardia ventrikel, fibrilasi ventrikel, dan ritme *torsade de pointes* yang membawa risiko besar kematian jantung mendadak jika tidak segera diidentifikasi dan diobati. Aritmia yang mengancam jiwa ini muncul dari hiperpolarisasi potensial membran kardiomiosit yang diinduksi oleh kondisi hipokalemia, yang secara paradoks meningkatkan kerja ventrikel dan menciptakan substrat elektrofisiologi yang rentan terhadap kondisi disritmia yang berbahaya. Seorang wanita multipara berusia 33 tahun pada usia kehamilan 29 minggu datang ke unit gawat darurat dengan riwayat lemas selama tiga hari, mual dan muntah melebihi lima episode per hari, kram otot, dan rasa tidak nyaman di perut. Pada pemeriksaan, didapatkan takikardia dengan denyut nadi tidak teratur lebih dari 100 kali dan takipnea. Elektrokardiogram awal menunjukkan gambaran ekg bigeminy ventrikel, kemudian berlanjut menjadi takikardia ventrikel dan diamati temuan yang menandakan keadaan aritmogenik yang berkembang dengan potensi berkembang menjadi aritmia ventrikel yang fatal. Hasil pemeriksaan laboratorium mengkonfirmasi hipokalemia berat dengan kadar kalium serum 1,7 mEq/L. Ekokardiografi dua dimensi menunjukkan tidak ada kelainan struktural jantung dengan fraksi ejeksi ventrikel kiri yang sebesar 60%. Pasien diberikan kalium klorida secara intravena, suplementasi magnesium sulfat empiris, Bisoprolol, dan Methyldopa di Unit Perawatan Intensif. Elektrokardiogram serial mendokumentasikan perkembangan dari bigeminy ventrikel menjadi trigeminy hingga menjadi gambaran normal setelah serum kalium menjadi 3,4 mEq/L. Kasus ini menunjukkan bahwa hipokalemia akibat hiperemesis gravidarum dapat menyebabkan aritmia ventrikel yang mematikan, dan hasil yang baik dapat dicapai melalui pengenalan dini dan penatalaksanaan yang sistematis.

Kata kunci: Aritmia, Hipokalemia, Kehamilan, Hiperemeis, Ektopi Ventrikel

INTRODUCTION

Nausea and vomiting represent some of the most frequently encountered physiological complaints among pregnant women, with reported prevalence ranging from 70 to 85% of all gestational cases.[1] Although these symptoms are generally considered a normal part of early pregnancy, a subset of patients experience a far more severe and debilitating condition known as hyperemesis gravidarum. This condition is characterized by persistent, uncontrollable vomiting that prevents adequate oral fluid intake and nutritional maintenance, occurring in approximately 0.3 to 2.3% of all pregnancies worldwide.[2] Unlike typical morning sickness, hyperemesis gravidarum poses significant clinical risks that extend well beyond gastrointestinal discomfort, potentially threatening both maternal and fetal well-being if left unmanaged.

The consequences of prolonged and unrelenting vomiting in pregnancy are multifaceted. Affected individuals are at risk for progressive dehydration, deteriorating nutritional status, metabolic disturbances, and clinically significant electrolyte imbalances. Among the most dangerous of these disturbances is hypokalemia, a condition defined by abnormally low serum potassium levels. Potassium is an essential electrolyte whose homeostasis is fundamentally linked to the regulation of cellular membrane excitability, particularly within cardiac and skeletal muscle tissue. When potassium levels fall below the physiological threshold, the electrochemical gradient across cell membranes is disrupted, resulting in impaired neuromuscular function and heightened cardiac irritability.[4]

Despite the recognized association between hyperemesis gravidarum, electrolyte imbalance, and cardiac arrhythmia, cases presenting in rural or resource-limited settings remain particularly challenging to manage. Limited access to advanced diagnostic modalities, subspecialty consultation, and certain laboratory investigations can complicate the timely identification and correction of underlying electrolyte disorders. This case report aims to highlight the clinical significance of hypokalemia-induced ventricular arrhythmia in a pregnant patient managed in a rural hospital setting, underscoring the importance of early recognition, vigilant electrolyte monitoring, and prompt therapeutic intervention in preventing potentially catastrophic maternal cardiac outcomes.

CASE PRESENTATION

A 33 years old multiparous woman presents to the emergency unit at 29 weeks gestation complaining of weakness and fatigue for three days. She also complained of nausea (+) vomiting (+) >5 times. It was also associated with a constant, generalized muscle cramp, relieved by pain killers for three to four hours only. The patient also complained of abdominal pain, which he described as diffuse, gradual in onset, aching in character, and nonradiating. No history of hypertension, diabetes and allergies were found. Her pregnancy had been progressing normally.

Physical examination revealed an afebrile patient with an irregular pulse of 100 beats per minute, blood pressure 140/95 mmHg, and an oxygen saturation of 98%. On respiratory examination, she was tachypneic (24x/m). Rest of the examination was normal. Electrocardiogram showed ventricular bigeminy (Figure 1). Soon after, she developed with short runs of non-sustained VT episode but, unfortunately there's no ecg to show up caused by its autoterminated. Then, She was admitted in the Intensive Care Unit of our hospital.

Laboratory evaluation revealed hypokalemia (1.7 mEq/L; normal 3.5 to 5.3 mEq/L), ketonuria (-), proteinuria (+) and normal liver, FT4, TSH and renal function. Magnesium test was considered but not used because it was not available in our hospital. Urgent two-dimensional echocardiogram showed normal left ventricular size and wall thickness with no segmental wall motion abnormalities and normal systolic and diastolic function with a left ventricular ejection fraction of 60%, TAPSE 24mm. There were no valvular abnormalities. A 12-lead electrocardiogram showed sinus rhythm with average rate of 100 beats/min with a normal axis, PR-interval, and QRS duration with no evidence of chamber enlargement or infarction, with PVC Bigeminy.

The treatment in ICU included adequate oxygenation, MgSO4 40% maintenance, dexametason 6mg/IM from obstetrician Intravenous potassium chloride (50 mEq) was administered three times daily, resulting in resolution of the ventricular arrhythmia, along with supportive treatment including methyldopa 250 mg and bisoprolol 2.5 mg. Over the subsequent days, A 12-lead electrocardiogram demonstrated sinus rhythm with an average heart rate of 95 beats per minute, normal axis, and normal PR interval and QRS duration. There was no evidence of chamber

enlargement or prior infarction; however, ST-segment depression was noted in leads V3–V6, along with premature ventricular complexes in a trigeminal pattern (Figure 2).

Electrolyte abnormalities persisted, with serum potassium remaining critically low at 2.2 mEq/L. Initial management included intravenous isotonic saline and parenteral potassium supplementation. The patient tolerated therapy well, and no further episodes of arrhythmia were documented over the subsequent 48 hours.

Clinical improvement was noted, and the patient was discharged three days later with normalized serum potassium levels (3.4 mEq/L) and normal electrocardiographic findings (Figure 3). Cardiovascular medications were subsequently discontinued.

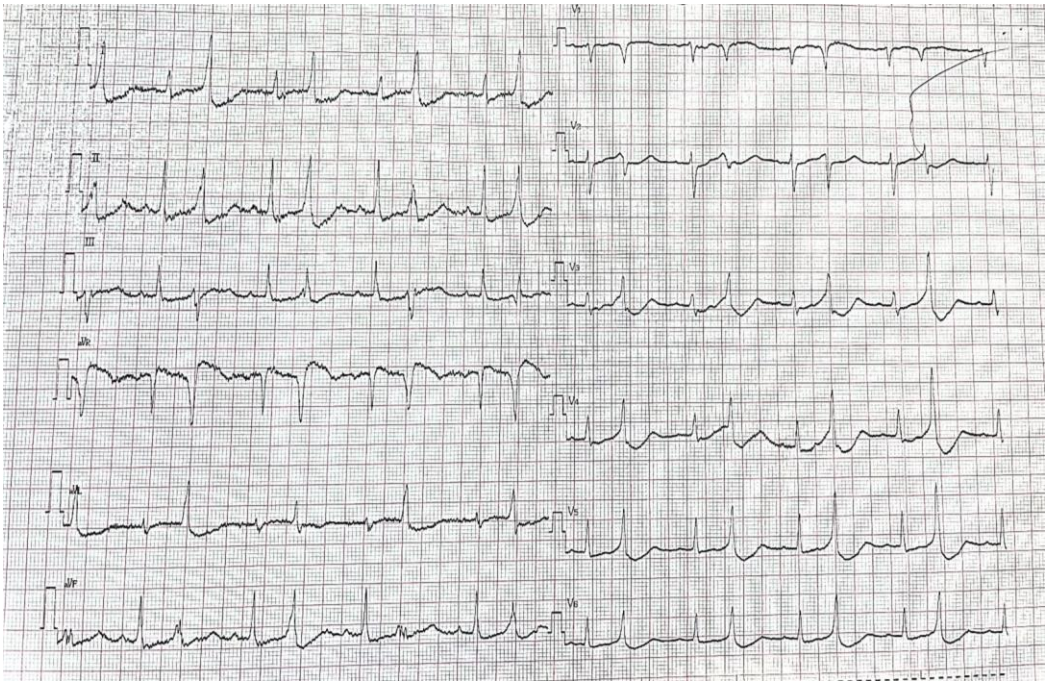


Figure 1. Previous Electrocardiogram (10/23/25) (ECG) Showed Sinus Rhytm, Heart Rate 100 min, Pri 120ms, Qrsd 40ms, No. ST-T Changes, PVC Bigeminy. Potassium: 1.7 mEq

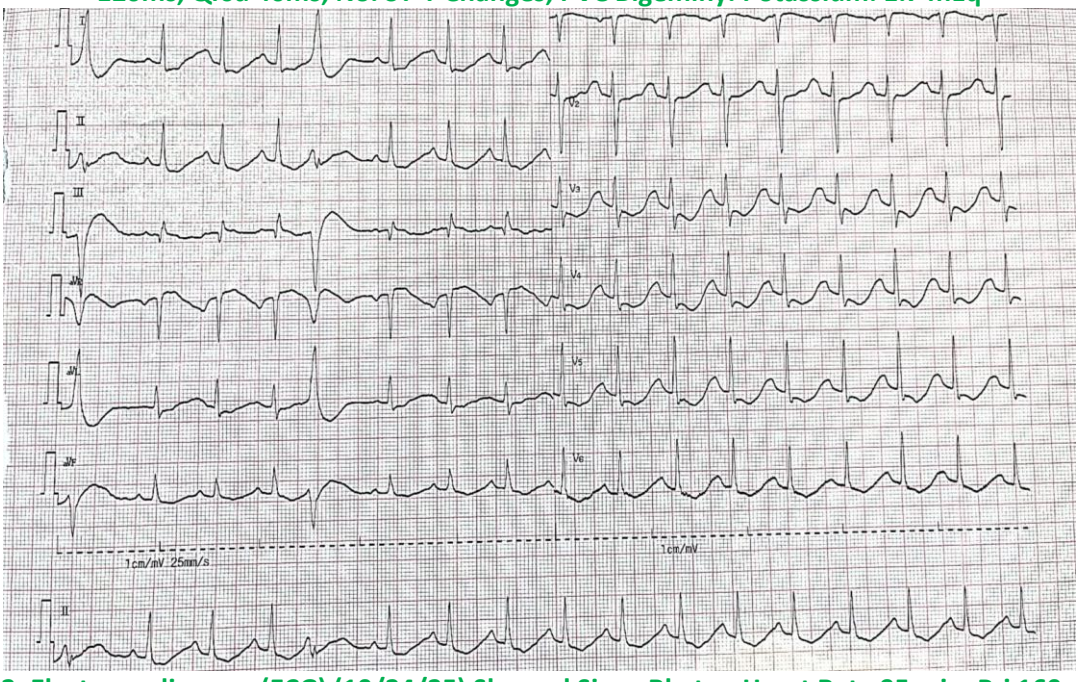


Figure 2. Electrocardiogram (ECG) (10/24/25) Showed Sinus Rhytm, Heart Rate 95 min, Pri 160ms, QRSd 40ms, ST Depression V3-V6, PVC Trigeminy. Potassium: 2.2 mEq/L

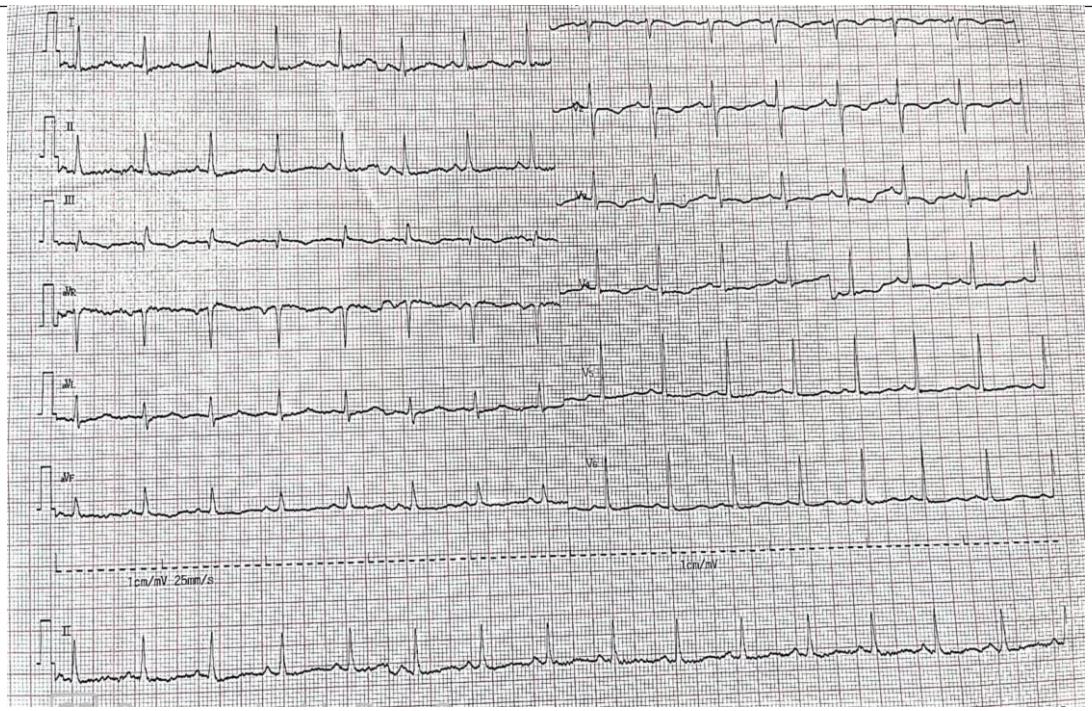


Figure 3. Electrocardiogram (ECG) (10/25/25) Showed Sinus Rhytm, Heart Rate 100 min, normoaxis, Pri 160ms, QRSd 40ms, ST Depression V3-V6 (No. evolution). Potassium: 3.4 mEq/L

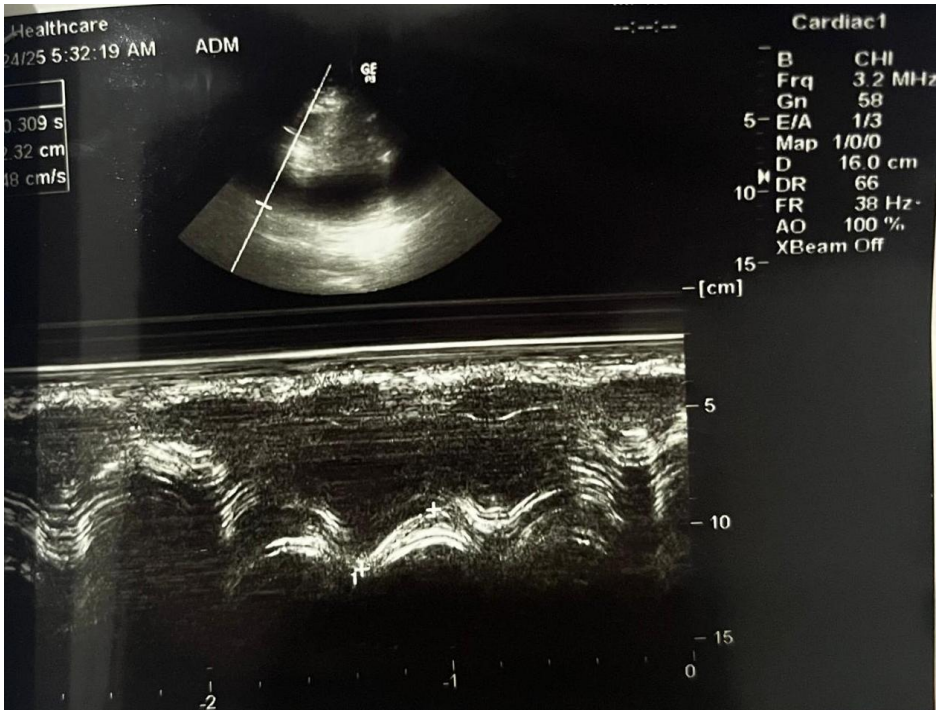


Figure 4. 2D echocardiogram for the normal results

Following the initial stabilization period in the Intensive Care Unit, the clinical team conducted a comprehensive and systematic re-evaluation of the patient's overall condition. At this stage, the management focus shifted from acute arrhythmia control to sustained correction of electrolyte imbalance, optimization of hemodynamic status, and continued fetal surveillance. The patient remained under continuous cardiac monitoring throughout her stay, with serial twelve-lead electrocardiograms obtained on each consecutive day of hospitalization to track the progression of cardiac rhythm changes in response to potassium replacement therapy.

On day 3 admission, a twelve-lead electrocardiogram was performed and

demonstrated a sinus rhythm with a heart rate of 100 beats per minute, a normal frontal axis, a PR interval of 160 milliseconds, and a QRS duration of 40 milliseconds. Persistent ST-segment depression was observed in leads V3 through V6, without any evolutionary changes compared to the preceding electrocardiogram obtained on the second day of hospitalization. Importantly, the frequency of premature ventricular contractions had significantly diminished, and no further episodes of ventricular bigeminy or trigeminy were identified on continuous telemetry monitoring. This electrocardiographic picture was consistent with ongoing but improving myocardial repolarization abnormalities, reflecting a gradual rather than immediate normalization of the intracellular potassium pool despite the progressive rise in serum potassium levels documented through serial laboratory measurements.

The persistence of ST-segment changes without further evolution was interpreted in the context of the clinical picture as a residual electrolyte effect rather than an ischemic process. This interpretation was supported by the absence of chest pain, the normal echocardiographic findings obtained earlier in the admission, and the temporal correlation between the electrolyte derangement and the onset of ECG changes. No troponin elevation was documented, further excluding acute myocardial injury as a contributing etiology. The clinical team therefore continued the current management strategy without escalation, maintaining intravenous potassium supplementation and close hemodynamic observation.

Over the subsequent 48 hours, the patient's clinical status demonstrated steady and consistent improvement. Her subjective complaints of generalized muscle weakness and cramping, which had been prominent on admission and were attributable to the systemic effects of hypokalemia on skeletal muscle function, progressively resolved as serum potassium levels trended toward the normal range. The nausea and vomiting, which had been the inciting cause of the electrolyte derangement, were effectively controlled through antiemetic therapy and intravenous fluid replacement, allowing gradual reintroduction of oral intake toward the latter part of the hospitalization. The patient's blood pressure, which had been elevated on admission at 140/95 mmHg, responded satisfactorily to Methyldopa therapy and remained within an acceptable range throughout the remainder of her inpatient stay.

Fetal surveillance was maintained throughout the hospitalization in close collaboration with the obstetric team. Continuous cardiotocographic monitoring was performed to assess fetal heart rate patterns and uterine activity. The fetal heart rate monitoring throughout the admission showed no evidence of fetal distress due to maternal hemodynamic or electrolyte abnormalities. Antenatal corticosteroid therapy with dexamethasone had been initiated early in the admission as a precautionary measure given the gestational age of 29 weeks and the potential risk of preterm delivery in the context of the acute maternal illness. This decision was made collaboratively between the cardiology and obstetric teams, reflecting the importance of a multidisciplinary approach in managing medically complex obstetric patients.

On the day preceding discharge, a repeat 12-lead electrocardiogram (ECG) demonstrated complete resolution of premature ventricular contractions, with restoration of a regular sinus rhythm at a rate of 100 beats per minute. The previously documented ST-segment depression in leads V3–V6 showed no further progression and remained unchanged compared with prior tracings.

A concurrent serum potassium measurement revealed a level of 3.4 mEq/L, approaching the normal reference range (3.5–5.3 mEq/L), consistent with effective correction of the underlying total body potassium deficit.

In light of the patient’s clinical stability and normalization of ECG findings, cardiac medications, including bisoprolol, were discontinued, as the arrhythmia was deemed secondary to electrolyte imbalance and had resolved following potassium repletion.

The patient was discharged in stable condition with arrangements for close outpatient follow-up. Discharge recommendations included dietary counseling focused on potassium-rich foods and continuation of oral potassium supplementation for a limited period post-discharge.

DISCUSSION

Hyperemesis gravidarum represents one of the most severe gastrointestinal complications encountered during pregnancy. It is defined as a condition of uncontrolled, persistent vomiting during gestation that results in significant weight loss, profound electrolyte disturbances, and metabolic derangements severe enough to

necessitate hospitalization. The spectrum of complications arising from this condition is broad and can include progressive dehydration, starvation-induced acidosis, marked electrolyte abnormalities, hypokalemia-associated alkalosis, skeletal muscle wasting, ketonuria, and in severe cases, neuromuscular tetany.[3] In the present case, the patient exhibited several of these hallmark features, most notably severe hypokalemia with an initial serum potassium level of 1.7 mEq/L, which served as the primary substrate for the development of significant ventricular arrhythmia.

Potassium plays an essential role in maintaining the resting membrane potential of excitable cells, particularly cardiomyocytes. Under physiological conditions, the intracellular-to-extracellular potassium concentration gradient is tightly regulated and is essential for the normal generation and propagation of cardiac action potentials. When extracellular potassium levels decline, as occurs in hypokalemia, the resting membrane potential becomes more negative, a phenomenon referred to as hyperpolarization. This hyperpolarized state paradoxically increases cellular excitability by lowering the threshold required to trigger spontaneous depolarization, thereby creating a favorable environment for the emergence of ectopic electrical activity within the myocardium. The result is an increased propensity for the generation of ventricular ectopic beats, which, if sustained or progressive, may evolve into more malignant arrhythmias.[5]

In the present case, the initial electrocardiogram demonstrated ventricular bigeminy, a pattern characterized by the alternating occurrence of a normal sinus beat followed by a premature ventricular contraction. This ECG finding is consistent with the degree of hypokalemia documented on admission and reflects the heightened ventricular irritability that is a well-recognized consequence of severely depressed serum potassium. Shortly after admission, the patient experienced brief, self-terminating runs of non-sustained ventricular tachycardia, which could not be captured on electrocardiographic recording due to their spontaneous resolution. These episodes underscore the clinical urgency associated with severe hypokalemia in the pregnant patient and highlight the potentially life-threatening nature of electrolyte-driven arrhythmias in this population.

On the following day of hospitalization, repeat electrocardiographic monitoring revealed a transition from ventricular bigeminy to ventricular trigeminy, a pattern in which every third beat is a premature ventricular contraction. This progression occurred despite the initiation of intravenous potassium replacement, which can be attributed to the persistence of critically low serum potassium levels at 2.2 mEq/L recorded on the subsequent laboratory evaluation. The continued electrolyte deficit reflected the severity of the underlying depletion and the time required for adequate intracellular replenishment, as total body potassium restoration is a gradual process that cannot be achieved rapidly through intravenous supplementation alone. This observation emphasizes the importance of serial electrolyte monitoring and ongoing potassium replacement rather than a single corrective dose.

The electrocardiographic evolution observed in this patient, from bigeminy to trigeminy and ultimately to resolution, mirrors the clinical trajectory of potassium correction and provides a practical illustration of the direct relationship between serum potassium concentration and ventricular ectopic activity. ST-segment depression noted in leads V3 through V6 on the second and third electrocardiograms further corroborated the ongoing myocardial repolarization abnormalities associated with electrolyte imbalance. These changes resolved in parallel with normalization of the serum potassium, reinforcing the electrolyte-driven etiology of the observed ECG abnormalities rather than a primary ischemic or structural cardiac process.

A two-dimensional echocardiogram was performed to exclude underlying structural heart disease as a contributing factor to the arrhythmia. The results demonstrated normal left ventricular dimensions and wall thickness, preserved systolic and diastolic function with a left ventricular ejection fraction of 60%, a tricuspid annular plane systolic excursion of 24 mm indicating normal right ventricular function, and the absence of any valvular pathology. These findings effectively ruled out

cardiomyopathy, valvular disease, or wall motion abnormalities as alternative explanations for the ventricular arrhythmia, thereby consolidating hypokalemia as the sole etiological factor in this case.

The management approach adopted in this case was appropriately guided by the severity of the electrolyte abnormality and the presence of active arrhythmia. The patient was admitted to the Intensive Care Unit to facilitate continuous cardiac monitoring and ensure prompt recognition of any deterioration in cardiac rhythm. Intravenous potassium chloride supplementation at a dose of 50 mEq administered three times daily was the cornerstone of treatment, targeting gradual restoration of serum potassium to within the normal physiological range. Alongside potassium replacement, magnesium sulfate 40% was administered as a maintenance infusion. This is clinically significant because magnesium and potassium homeostasis are closely interrelated; hypomagnesemia frequently coexists with hypokalemia and can impair the renal tubular reabsorption of potassium, rendering potassium replacement less effective in the absence of concurrent magnesium repletion. Although serum magnesium levels could not be assessed due to the unavailability of the test in the hospital setting, empirical magnesium supplementation was a judicious clinical decision given this well-established physiological relationship.

Bisoprolol, a selective beta-1 adrenergic receptor antagonist, was incorporated into the treatment regimen as an adjunct antiarrhythmic agent. Beta-blockers reduce the automaticity of ectopic ventricular foci by attenuating sympathetic stimulation of the myocardium, thereby decreasing the frequency of premature ventricular contractions. Their use in this context was appropriate and consistent with the management of symptomatic ventricular ectopy in the setting of a reversible precipitant. Methyldopa was concurrently administered for blood pressure management, given the patient's elevated admission blood pressure of 140/95 mmHg, which raised concern for gestational hypertension. Methyldopa remains one of the preferred antihypertensive agents in pregnancy due to its established safety profile for both mother and fetus.

Dexamethasone 6 mg intramuscularly was administered as part of the obstetric management protocol, serving the dual purpose of promoting fetal lung maturity in anticipation of potential preterm delivery at 29 weeks gestation and providing antiemetic benefit, as corticosteroids have been reported to reduce the severity of nausea and vomiting in hyperemesis gravidarum. This decision reflects the necessity of simultaneously addressing both the maternal cardiac emergency and the ongoing obstetric considerations inherent in managing a critically ill pregnant patient.

The clinical outcome in this case was favorable. Following several days of intensive potassium replacement and supportive care, the patient's serum potassium normalized to 3.4 mEq/L and the electrocardiogram demonstrated resolution of ventricular ectopy with restoration of a stable sinus rhythm. The patient was subsequently discharged in stable condition, with cardiac medications appropriately discontinued given the resolution of the arrhythmia upon correction of the underlying electrolyte disturbance. This outcome reinforces the principle that ventricular arrhythmias secondary to hypokalemia are generally reversible when the electrolyte abnormality is identified and corrected in a timely manner.

This case reinforces the well-established principle that ventricular arrhythmias secondary to hypokalemia are largely reversible when promptly recognized and appropriately managed, underscoring the importance of early identification and correction of electrolyte abnormalities.

This case also brings attention to the unique challenges of managing a complex cardiac and obstetric emergency in a rural hospital setting with limited diagnostic resources. The inability to measure serum magnesium levels, the absence of electrophysiology consultation, and the lack of advanced cardiac monitoring infrastructure are realities faced by many healthcare providers in resource-constrained environments. Despite these limitations, the clinical team demonstrated sound judgment in empirically addressing potential coexisting electrolyte

deficiencies and maintaining vigilant monitoring through serial electrocardiography. This case serves as a reminder that thorough clinical reasoning and systematic management can yield excellent outcomes even in the absence of advanced technological resources.

In conclusion, this case emphasizes that hypokalemia secondary to hyperemesis gravidarum is a clinically significant and potentially life-threatening condition capable of precipitating serious ventricular arrhythmias in the pregnant patient. Clinicians managing pregnant women with prolonged vomiting must maintain a high index of suspicion for electrolyte disturbances, particularly hypokalemia, and should incorporate early electrocardiographic assessment and serial electrolyte monitoring as integral components of the clinical evaluation. Prompt and adequate potassium replacement, supported by empirical magnesium supplementation and adjunctive antiarrhythmic therapy where indicated, constitutes the foundation of effective management and is critical to achieving favorable maternal and fetal outcomes.[3]

From an obstetric viewpoint, this case highlights the importance of recognizing hyperemesis gravidarum not merely as a gastrointestinal nuisance but as a potentially life-threatening condition with serious systemic consequences. Hyperemesis gravidarum in this patient manifested at 29 weeks' gestation, representing an atypical temporal profile, as the condition classically peaks between 8 and 12 weeks and resolves by mid-gestation in the majority of cases. Persistence into the third trimester necessitates a comprehensive evaluation for underlying or contributory pathophysiological factors, including multiple gestation, gestational trophoblastic disease, and other maternal or fetal conditions that may sustain or exacerbate refractory nausea and vomiting beyond the first trimester. The presence of proteinuria alongside hypertension at 140/95 mmHg in this patient also raises an important obstetric concern. Although the primary focus of this case centers on the hypokalemia-induced arrhythmia, the concurrent finding of elevated blood pressure and proteinuria at 29 weeks necessitates consideration of superimposed preeclampsia, which carries its own significant maternal and fetal risks. Methyldopa was appropriately initiated for blood pressure control, consistent with current obstetric guidelines favoring its use as a first-line antihypertensive in pregnancy. However, further workup including urine protein quantification, liver enzymes, complete blood count, and creatinine would be warranted to formally evaluate for preeclampsia in this clinical context.[6]

Fetal wellbeing surveillance is a critical component of managing any acutely ill pregnant patient. At 29 weeks gestation, the fetus remains at significant risk of complications associated with preterm birth should maternal hemodynamic instability precipitate preterm labor or necessitate emergency delivery. The administration of dexamethasone 6 mg intramuscularly in this case was a sound and guideline-concordant obstetric decision, consistent with recommendations from major organizations including ACOG and WHO for antenatal corticosteroid administration between 24 to 34 weeks gestation in patients at risk of preterm delivery within seven days. Corticosteroid therapy significantly reduces the incidence of respiratory distress syndrome, intraventricular hemorrhage, and neonatal mortality in preterm neonates. The additional antiemetic benefit of corticosteroids in hyperemesis gravidarum, while a secondary consideration, may have contributed to the improvement of the patient's gastrointestinal symptoms during the admission. Continuous cardiotocographic monitoring throughout the hospitalization was essential not only to assess fetal heart rate reactivity but also to detect any uterine activity that might signal the onset of preterm labor in the context of the acute maternal illness. Maternal electrolyte disturbances, particularly hypokalemia, can theoretically influence uterine contractility given the role of potassium in smooth muscle function, adding further obstetric relevance to prompt electrolyte correction in this population.[7]

The multidisciplinary collaboration between the cardiology and obstetric teams demonstrated in this case represents the ideal model of care for medically complex obstetric patients. Decisions regarding timing and mode of delivery, thresholds for escalation of fetal surveillance, and the safety of pharmacological

agents must all be evaluated jointly to balance maternal cardiac stability with fetal wellbeing. In resource-limited rural settings, this interdisciplinary coordination becomes even more critical given the constraints on available diagnostic and therapeutic modalities.

CONCLUSION

This case report presents a compelling clinical illustration of severe hypokalemia-induced ventricular arrhythmia in a 29 weeks pregnant patient, arising as a direct consequence of prolonged and intractable vomiting secondary to hyperemesis gravidarum. The clinical course documented in this case, from the initial presentation with ventricular bigeminy and critically depressed serum potassium at 1.7 mEq/L, through the intermediate phase of ventricular trigeminy with persistent ST-segment depression, to the ultimate resolution of all electrocardiographic abnormalities following successful potassium supplementation, provides a clear and instructive demonstration of the direct electrophysiological relationship between hypokalemia and ventricular ectopic activity.

Several important clinical lessons can be drawn from this case. First and foremost, hypokalemia must be consistently considered as a primary differential diagnosis in any pregnant patient presenting with cardiac arrhythmia, particularly in the context of a history of prolonged vomiting or inadequate oral intake. The severity of the electrolyte imbalance documented in this patient, with an initial serum potassium of 1.7 mEq/L, far below the lower limit of normal, reinforces the capacity of hyperemesis gravidarum to produce life-threatening metabolic consequences if not recognized and managed promptly. Clinicians must therefore not underestimate the systemic impact of persistent vomiting in the pregnant patient and should routinely incorporate serum electrolyte assessment as part of the initial evaluation in this clinical context.

Second, this case highlights the critical importance of serial electrocardiographic monitoring in guiding the management of hypokalemia-induced arrhythmia. The evolution of the ECG findings across three consecutive days of hospitalization provided valuable real-time information regarding the adequacy of electrolyte replacement and the trajectory of arrhythmia resolution. Serial electrocardiography is a widely accessible, non-invasive, and cost-effective tool that is available even in resource-limited settings, making it a particularly valuable monitoring modality in environments where advanced electrophysiology services or continuous telemetry infrastructure may not be readily accessible.

Third, the role of empirical magnesium supplementation in this case deserves emphasis. Given the well-established physiological interdependence between potassium and magnesium homeostasis, and the recognized capacity of hypomagnesemia to impair renal potassium reabsorption and thereby perpetuate hypokalemia, concurrent magnesium repletion is a clinically sound and evidence-informed adjunct to potassium replacement therapy. In settings where serum magnesium measurement is unavailable, as was the case in this rural hospital, empirical magnesium supplementation represents a pragmatic and justifiable clinical decision that may significantly enhance the effectiveness of potassium correction.

Fourth, this case underscores the necessity of a multidisciplinary approach in managing medically complex obstetric patients. The concurrent involvement of the cardiology and obstetric teams was essential to addressing both the acute maternal cardiac emergency and the ongoing fetal surveillance requirements inherent to the management of a critically ill pregnant patient at 29 weeks of gestation. The administration of antenatal corticosteroids, the maintenance of continuous fetal monitoring, and the close collaboration between specialties collectively contributed to the favorable outcome achieved in this case.

Finally, this case serves as an important reminder of the clinical realities faced by healthcare providers in rural and resource-constrained settings. Despite the absence of certain diagnostic tools, including serum magnesium measurement and advanced electrophysiology consultation, the clinical team successfully managed a potentially life-threatening obstetric and cardiac emergency through sound clinical reasoning, systematic monitoring, and appropriate therapeutic intervention. This outcome demonstrates that excellent patient care is achievable in resource-limited environments when clinical judgment is applied rigorously and management decisions are grounded in a thorough understanding of the underlying pathophysiology.

In summary, hypokalemia arising from

hyperemesis gravidarum should be regarded as a serious and potentially life-threatening condition with significant cardiac implications. Prompt identification of electrolyte disturbances, early initiation of adequate replacement therapy, vigilant serial monitoring, and a multidisciplinary management approach are the cornerstones of care in such cases. When these principles are applied effectively, complete resolution of hypokalemia-induced ventricular arrhythmia and restoration of normal cardiac rhythm are achievable outcomes, as demonstrated by the favorable clinical course of the patient described in this report.

From an obstetric perspective, this case reinforces that hyperemesis gravidarum should be evaluated and managed comprehensively, with particular attention to its systemic electrolyte consequences rather than its gastrointestinal manifestations alone. Obstetricians must maintain vigilance for the cardiac implications of severe hypokalemia and should incorporate early electrocardiographic evaluation as part of the routine workup in patients presenting with significant vomiting and electrolyte derangement during pregnancy.

Additionally, the coexistence of hypertension and proteinuria in this patient underscores the complexity of obstetric care, where overlapping conditions may require coordinated and simultaneous management. The administration of antenatal corticosteroids at 29 weeks was a vital aspect of care, emphasizing the importance of anticipating preterm delivery in the setting of acute maternal illness.

Ultimately, achieving favorable outcomes in such cases relies on the seamless integration of obstetric and medical expertise, prompt recognition of life-threatening complications, and the consistent application of evidence-based management principles, even within the limitations of a rural healthcare setting.

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