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REVIEW ARTICLE

# Ventricular Bigeminy in Anaphylactic Shock: A Rare Presentation with Epinephrine

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## ABSTRACT

***Background:*** Anaphylaxis is a medical emergency with well-characterized cardiovascular manifestations, typically presenting with hypotension and tachycardia. However, ventricular arrhythmias in this context are exceedingly rare and underrecognized.

***Case Presentation:*** We report the case of a 19-year-old male who developed ventricular bigeminy during an episode of anaphylactic shock. He presented with hypotension, urticaria, and bradycardia. ECG revealed ventricular bigeminy. Prompt intramuscular epinephrine led to immediate improvement in blood pressure and resolution of the arrhythmia. A second episode of bigeminy followed, which also responded to epinephrine.

***Conclusion:*** Clinicians should be aware that arrhythmias like ventricular bigeminy can occur in anaphylaxis and may resolve with standard anaphylaxis treatment. Early administration of epinephrine is critical not only to reverse shock but potentially to stabilize cardiac rhythm.

## KEYWORDS

• Anaphylaxis • Ventricular Bigeminy • Arrhythmia • Epinephrine • Emergency Medicine • Refractory Anaphylaxis

## INTRODUCTION

Anaphylaxis is a severe, life-threatening systemic hypersensitivity reaction characterized by rapid onset and multi-organ involvement, most commonly mediated by immunoglobulin E (IgE). Clinical features often include hypotension, bronchospasm, mucocutaneous signs, and gastrointestinal symptoms. Common triggers include foods, medications,

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insect stings, and latex, although idiopathic cases are not uncommon.[1]

Prompt recognition and administration of intramuscular (IM) epinephrine are the cornerstones of treatment.[2] However, cardiovascular manifestations during anaphylaxis can vary significantly. While tachycardia and hypotension are common due to vasodilation and plasma leakage, arrhythmias are less frequently documented. Rare reports include atrial fibrillation, ventricular tachycardia, myocardial ischemia, and even cardiac arrest.[3,4]

Ventricular bigeminy, a form of premature ventricular complex (PVC) occurring every alternate beat, is highly unusual in anaphylaxis and has not been well characterized in existing literature. Its occurrence in the setting of anaphylactic shock may reflect transient myocardial irritability, ischemia, or histamine induced electrical instability. The role of epinephrine in both reversing the arrhythmia and improving hemodynamics in such cases is also underexplored.

We present a rare case of ventricular bigeminy in a young male with anaphylactic shock, which resolved rapidly following administration of IM epinephrine. This case underscores the importance of early epinephrine use not only to reverse circulatory collapse but also to potentially normalize cardiac electrophysiology in anaphylaxis.

## CASE PRESENTATION

A 19-year-old previously healthy male presented to the emergency department with complaints of dizziness, generalized urticaria, and facial swelling that had developed over the past 30 minutes. His sister, a nursing student, reported a home blood pressure reading of 70 mmHg systolic with an unrecordable diastolic pressure. There was no history of recent medication use, allergen exposure, insect bite, food ingestion, or prior allergic reactions.

## PRIMARY SURVEY

- **Airway:** Patent, no signs of angioedema or laryngeal edema.
- **Breathing:** Respiratory rate 18/min, Oxygen saturation 97% on room air, bilateral normal breath sounds.
- **Circulation:** Initial Blood Pressure: 80/60 mmHg, Heart Rate: 130 bpm, ventricular bigeminy on ECG monitor, Cyanosis over hands and tongue, Extremities cold and clammy.
- **Disability:** Glasgow Coma Scale (GCS): E4V5M6, Pupils equally reactive to light, Blood Glucose: 115 mg/dL.
- **Exposure:** Generalized urticaria and erythematous rashes observed.

## INITIAL ECG FINDINGS

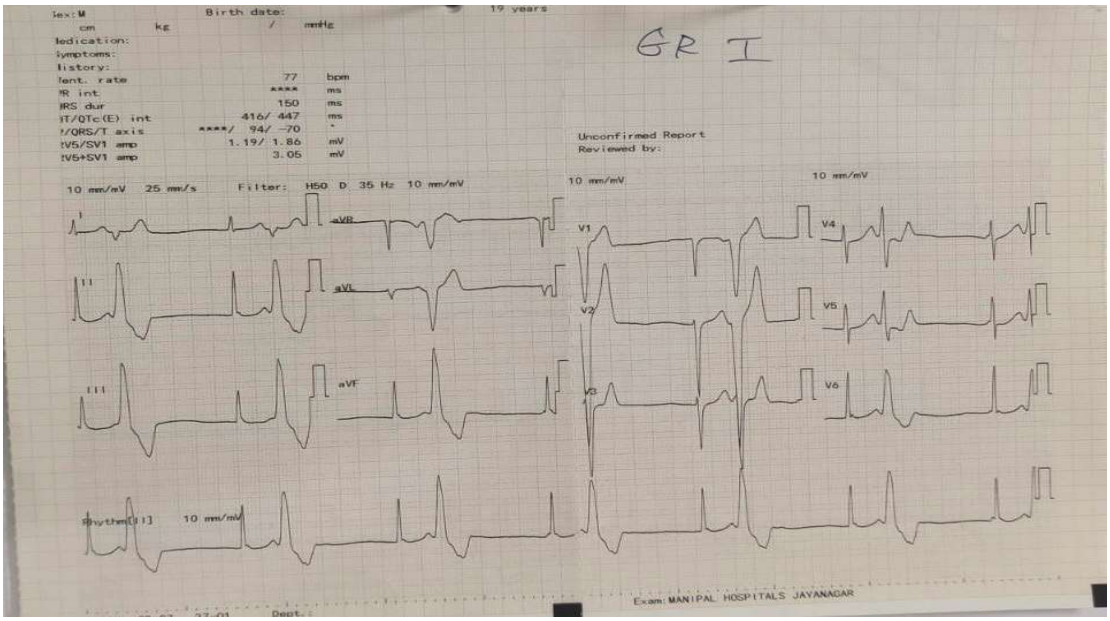



**Figure 1:** Ventricular bigeminy confirmed on 12-lead ECG
(ECG shows regular underlying rhythm with addition of PVC after every beat)

### Management

Given the presence of anaphylactic shock and ventricular bigeminy, the following interventions were implemented:

- Epinephrine 0.5 mg IM administered in the anterolateral aspect of thigh.

### Post-administration:

- ECG: Reverted to sinus rhythm.
- Blood Pressure: Improved to 100/60 mmHg.
- Rashes: Started resolving.

Approximately five minutes later, ventricular bigeminy reappeared. A second dose of epinephrine 0.5 mg IM was administered, which reverted the ECG to sinus rhythm. Blood pressure improved to 130/80 mmHg. No side effects of the drug were noted.

### Supportive Medications

- Hydrocortisone 100mg IV and Pheniramine 50mg IV were administered.
- IV Fluid Bolus: 1 litre of crystalloid (Normal Saline) was given

### Secondary Survey

Unremarkable except for resolving urticaria.

### Investigations:

- **ECG**: Confirmed ventricular bigeminy, which reverted to normal sinus rhythm following epinephrine administration (compare Figure 1 and Figure 2).

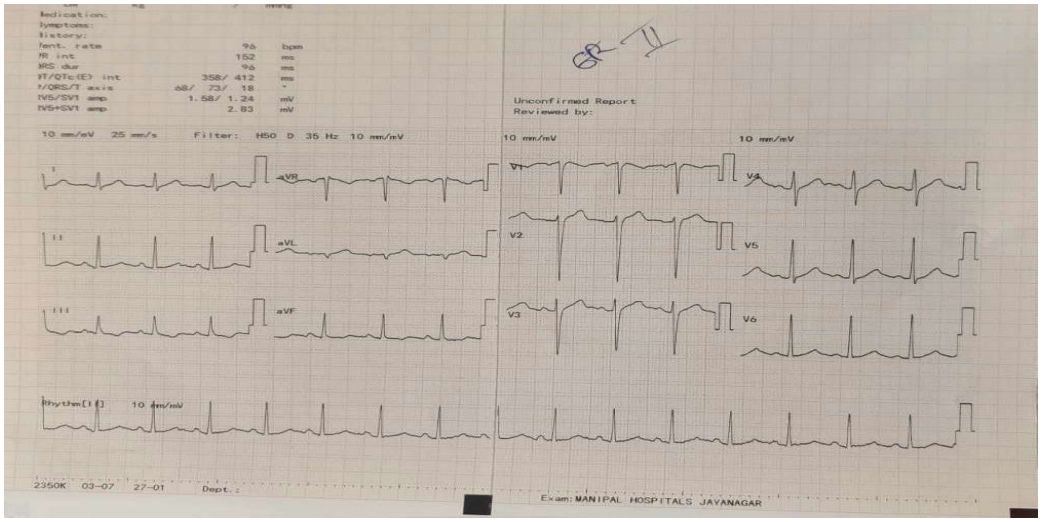
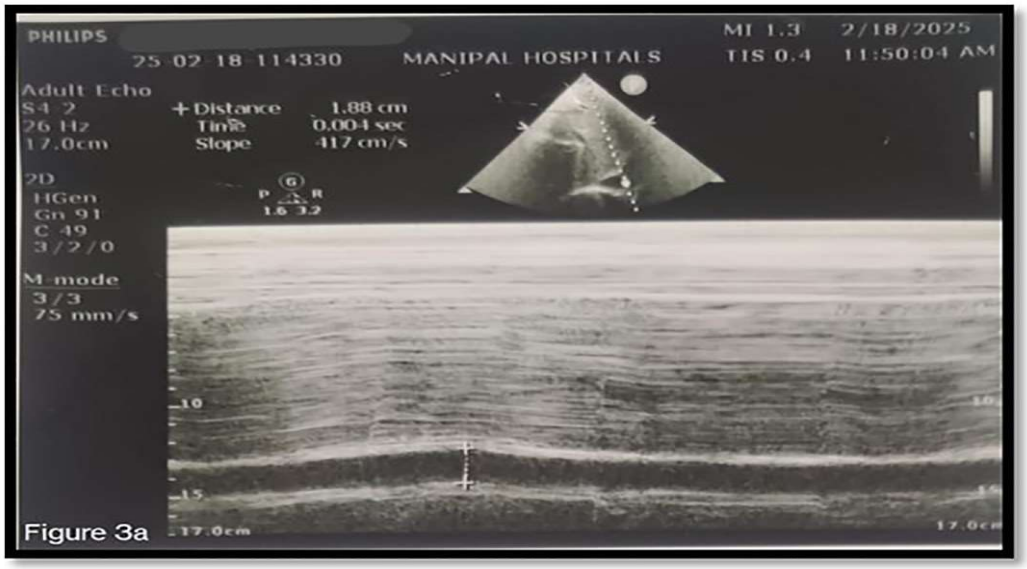
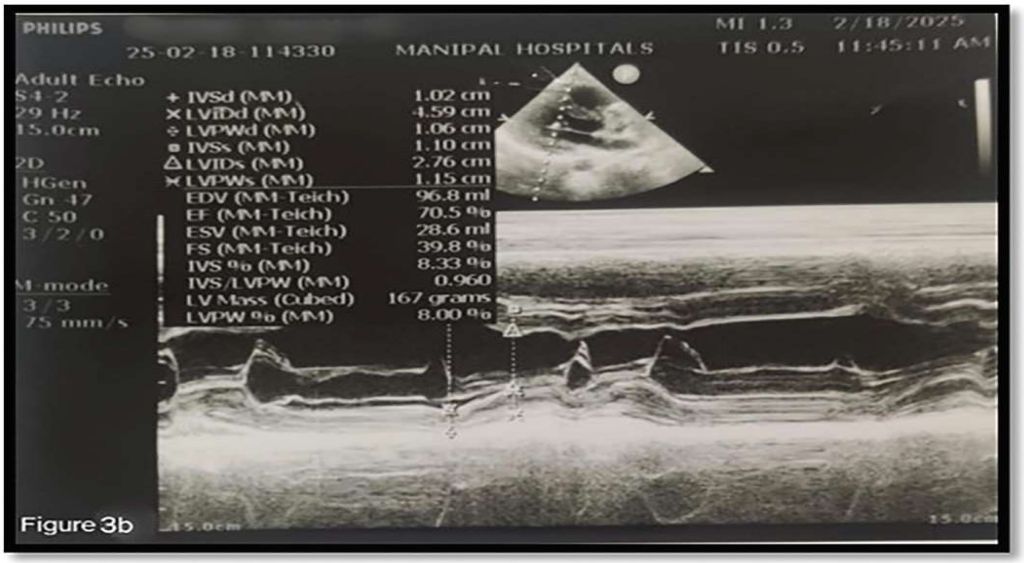



**Figure 2:** ECG showing sinus rhythm after administration of epinephrine

- **Echocardiogram** (Figures 3a to 3f): Normal, with no structural heart disease.

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× LVIDd (MM) 4.59 cm
÷ LVPWd (MM) 1.06 cm
■ IVSs (MM) 1.10 cm
△ LVIDs (MM) 2.76 cm
✕ LVPWs (MM) 1.15 cm
EDV (MM-Teich) 96.8 ml
EF (MM-Teich) 70.5 %
ESV (MM-Teich) 28.6 ml
FS (MM-Teich) 39.8 %
IVS % (MM) 8.33 %
IVS/LVPW (MM) 0.960
LV Mass (Cubed) 167 grams
LVPW % (MM) 8.00 %
Figure 3b

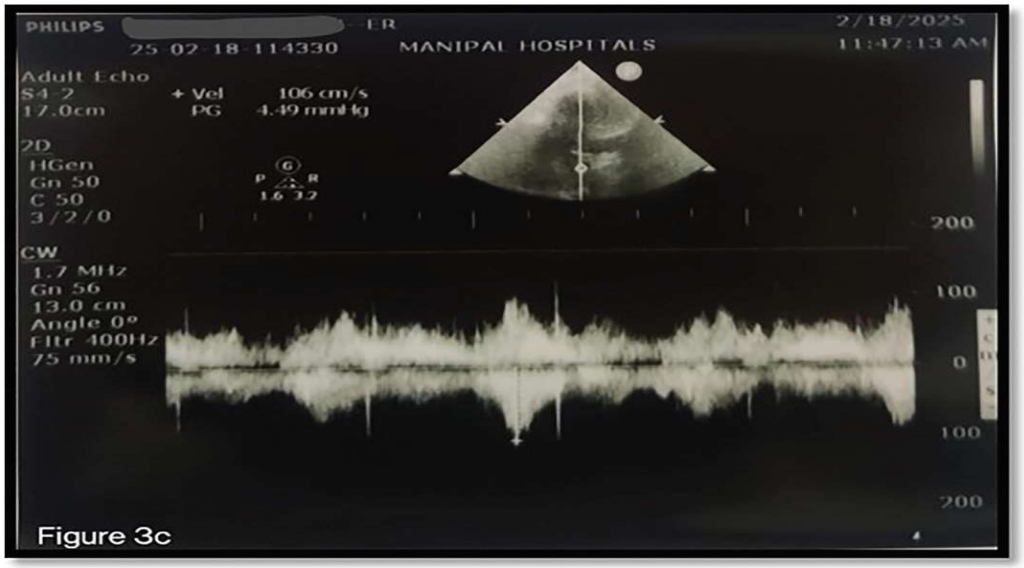
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Figure 3c

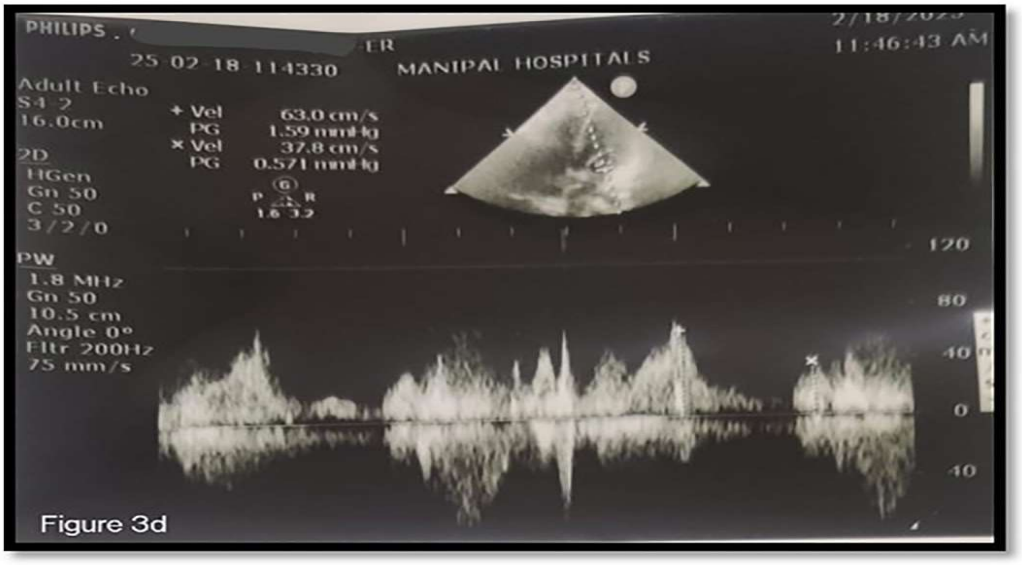
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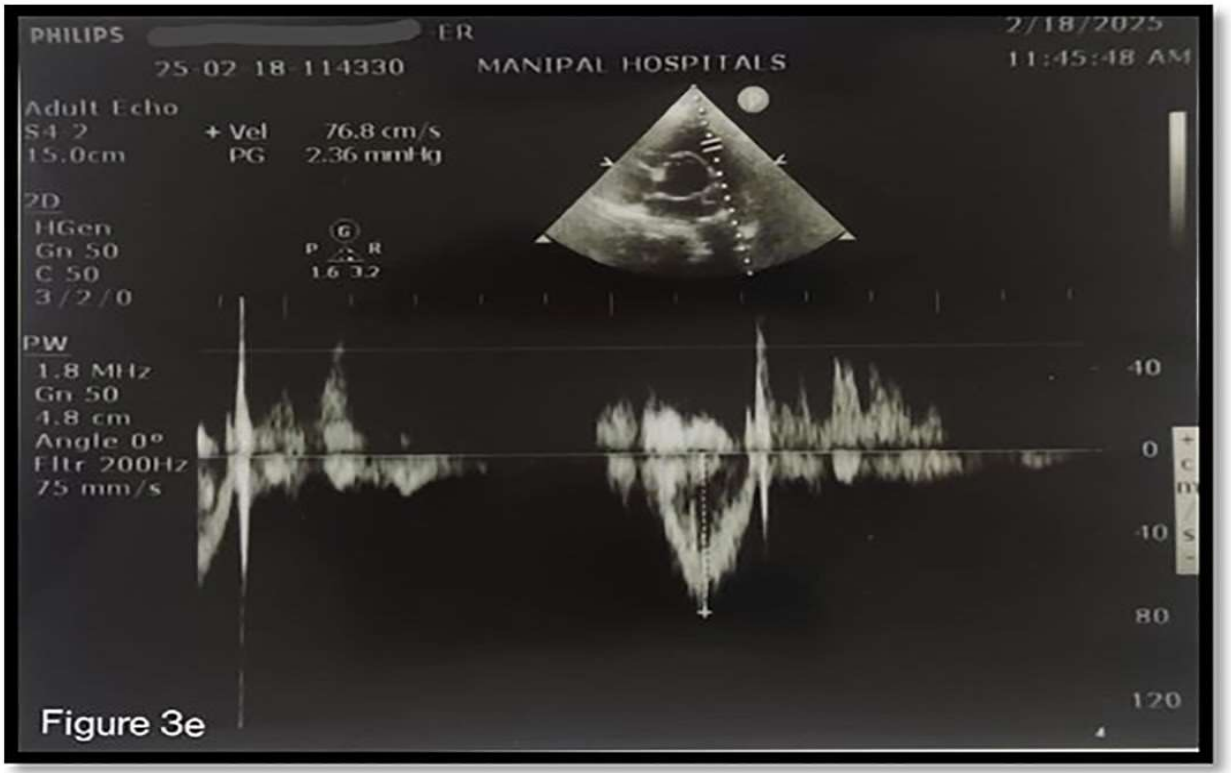
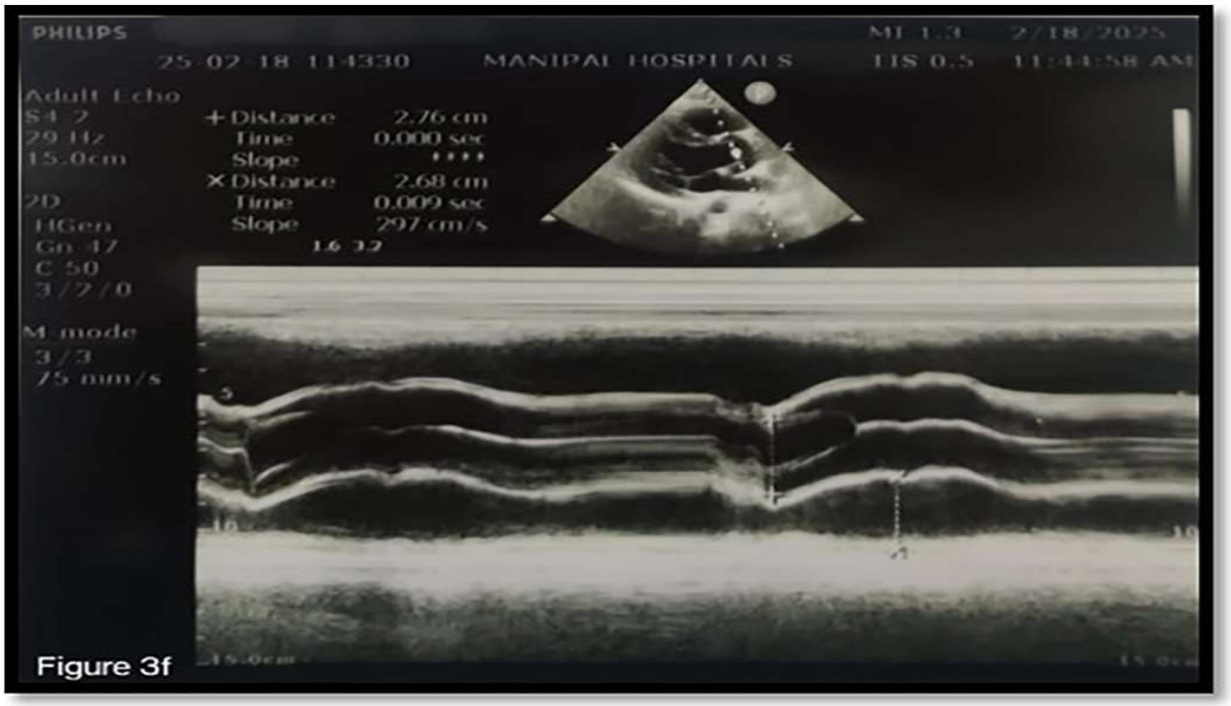
Figure 3d

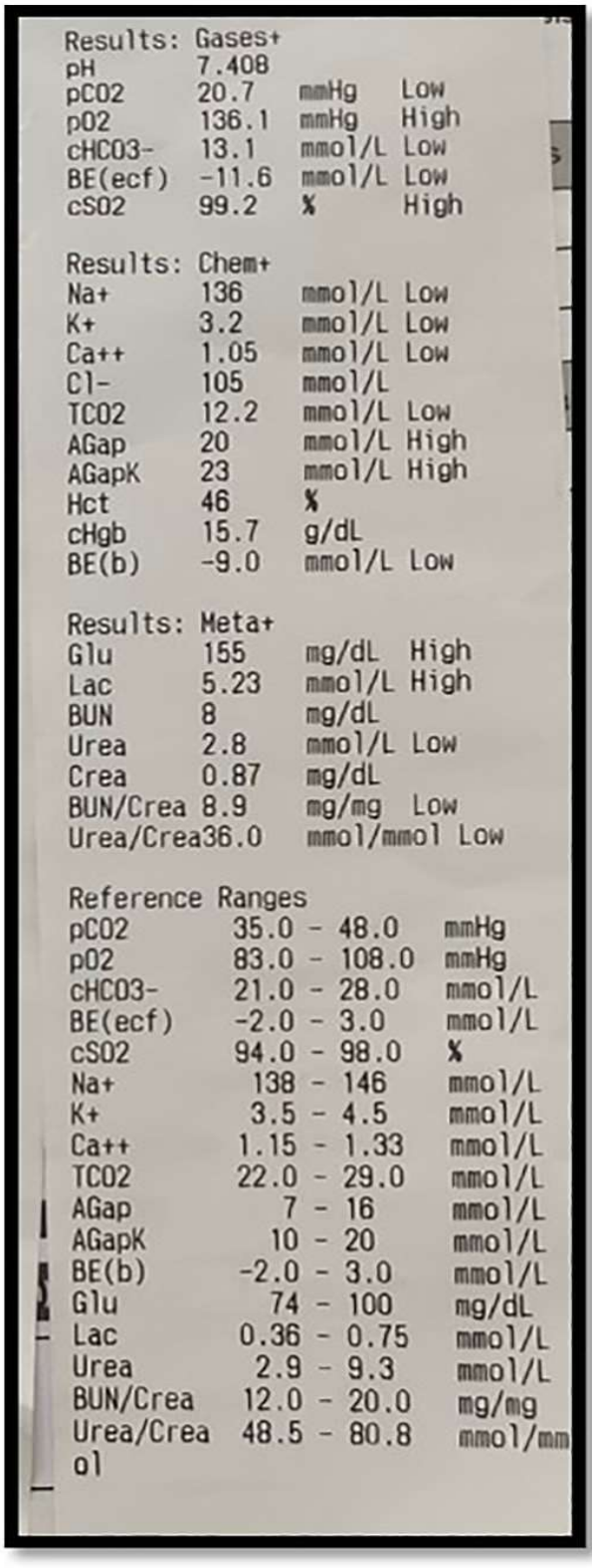



**Figures 3a to 3f:** Two-dimensional Echocardiogram of the Heart

- **Blood gas Analysis:**

Initial Blood Gas **(Figure 4):** Compensated metabolic acidosis with reduction in bicarbonate ($HCO_3^-$) resulting from metabolic acidosis, accompanied by a compensatory decrease in $pCO_2$ through respiratory mechanisms, helping to sustain a near normal pH level was noted. (pH: 7.408, partial pressure of carbon dioxide ($pCO_2$):20.7 mmHg, $HCO_3^-$: 13.1 mmol/L, Base Excess: -11 mmol/L, Lactate:5.23)



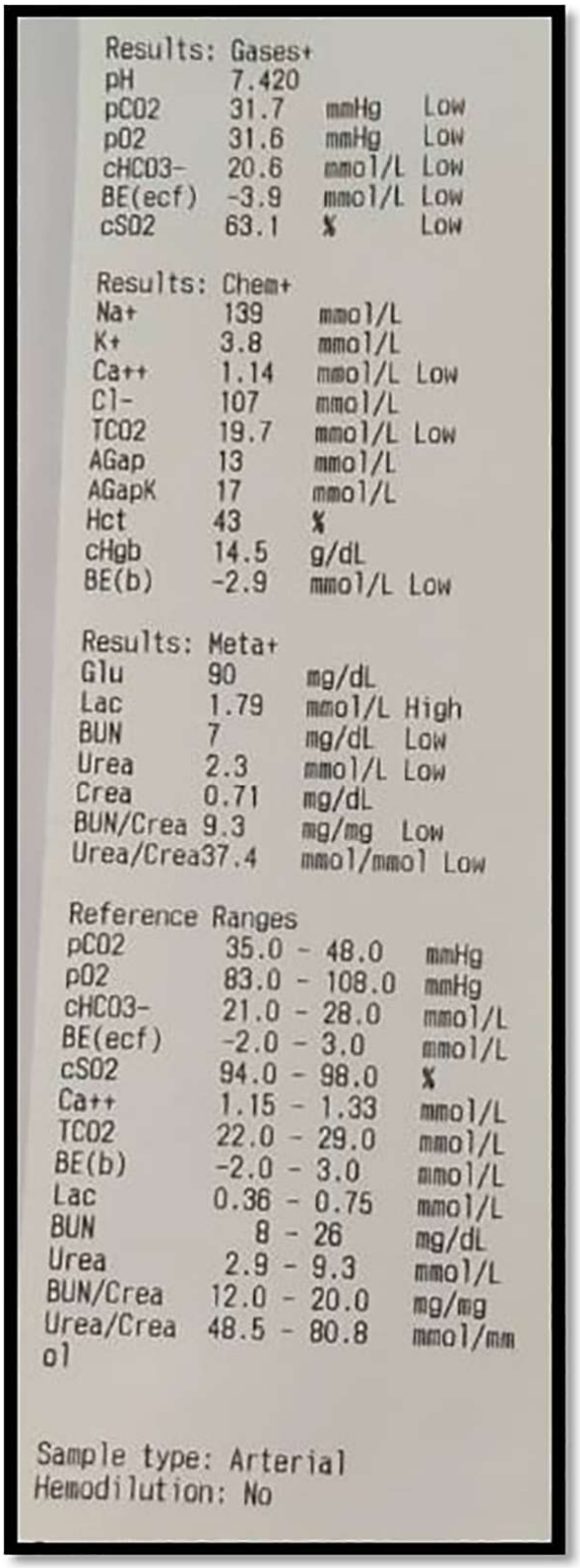
| Results: Gases+ | | | |
|---|---|---|---|
| pH | 7.408 | | |
| pCO2 | 20.7 | mmHg | Low |
| pO2 | 136.1 | mmHg | High |
| cHCO3- | 13.1 | mmol/L | Low |
| BE(ecf) | -11.6 | mmol/L | Low |
| cSO2 | 99.2 | % | High |

| Results: Chem+ | | | |
|---|---|---|---|
| Na+ | 136 | mmol/L | Low |
| K+ | 3.2 | mmol/L | Low |
| Ca++ | 1.05 | mmol/L | Low |
| Cl- | 105 | mmol/L | |
| TCO2 | 12.2 | mmol/L | Low |
| AGap | 20 | mmol/L | High |
| AGapK | 23 | mmol/L | High |
| Hct | 46 | % | |
| cHgb | 15.7 | g/dL | |
| BE(b) | -9.0 | mmol/L | Low |

| Results: Meta+ | | | |
|---|---|---|---|
| Glu | 155 | mg/dL | High |
| Lac | 5.23 | mmol/L | High |
| BUN | 8 | mg/dL | |
| Urea | 2.8 | mmol/L | Low |
| Crea | 0.87 | mg/dL | |
| BUN/Crea | 8.9 | mg/mg | Low |
| Urea/Crea | 36.0 | mmol/mmol | Low |

| Reference Ranges | | |
|---|---|---|
| pCO2 | 35.0 - 48.0 | mmHg |
| pO2 | 83.0 - 108.0 | mmHg |
| cHCO3- | 21.0 - 28.0 | mmol/L |
| BE(ecf) | -2.0 - 3.0 | mmol/L |
| cSO2 | 94.0 - 98.0 | % |
| Na+ | 138 - 146 | mmol/L |
| K+ | 3.5 - 4.5 | mmol/L |
| Ca++ | 1.15 - 1.33 | mmol/L |
| TCO2 | 22.0 - 29.0 | mmol/L |
| AGap | 7 - 16 | mmol/L |
| AGapK | 10 - 20 | mmol/L |
| BE(b) | -2.0 - 3.0 | mmol/L |
| Glu | 74 - 100 | mg/dL |
| Lac | 0.36 - 0.75 | mmol/L |
| Urea | 2.9 - 9.3 | mmol/L |
| BUN/Crea | 12.0 - 20.0 | mg/mg |
| Urea/Crea ol | 48.5 - 80.8 | mmol/mm |

**Figure 4:** Blood Gas analysis done on arrival



| Results: Gases+ | | | |
|---|---|---|---|
| pH | 7.420 | | |
| pCO2 | 31.7 | mmHg | Low |
| pO2 | 31.6 | mmHg | Low |
| cHCO3- | 20.6 | mmol/L | Low |
| BE(ecf) | -3.9 | mmol/L | Low |
| cSO2 | 63.1 | % | Low |

| Results: Chem+ | | | |
|---|---|---|---|
| Na+ | 139 | mmol/L | |
| K+ | 3.8 | mmol/L | |
| Ca++ | 1.14 | mmol/L | Low |
| Cl- | 107 | mmol/L | |
| TCO2 | 19.7 | mmol/L | Low |
| AGap | 13 | mmol/L | |
| AGapK | 17 | mmol/L | |
| Hct | 43 | % | |
| cHgb | 14.5 | g/dL | |
| BE(b) | -2.9 | mmol/L | Low |

| Results: Meta+ | | | |
|---|---|---|---|
| Glu | 90 | mg/dL | |
| Lac | 1.79 | mmol/L | High |
| BUN | 7 | mg/dL | Low |
| Urea | 2.3 | mmol/L | Low |
| Crea | 0.71 | mg/dL | |
| BUN/Crea | 9.3 | mg/mg | Low |
| Urea/Crea | 37.4 | mmol/mmol | Low |

| Reference Ranges | | |
|---|---|---|
| pCO2 | 35.0 - 48.0 | mmHg |
| pO2 | 83.0 - 108.0 | mmHg |
| cHCO3- | 21.0 - 28.0 | mmol/L |
| BE(ecf) | -2.0 - 3.0 | mmol/L |
| cSO2 | 94.0 - 98.0 | % |
| Ca++ | 1.15 - 1.33 | mmol/L |
| TCO2 | 22.0 - 29.0 | mmol/L |
| BE(b) | -2.0 - 3.0 | mmol/L |
| Lac | 0.36 - 0.75 | mmol/L |
| BUN | 8 - 26 | mg/dL |
| Urea | 2.9 - 9.3 | mmol/L |
| BUN/Crea | 12.0 - 20.0 | mg/mg |
| Urea/Crea ol | 48.5 - 80.8 | mmol/mm |

Sample type: Arterial
Hemodilution: No

**Figure 5:** Blood Gas analysis after stabilization (done 2-hours after the first)

**Second Blood Gas** (Figure 5): Taken 2 hours after the first one, indicatingimprovement with treatment. (pH: 7.420, $pCO_2$: 31, $HCO_3^-$: 20, Base excess - 2.9, lactate: 1.79)

The comparison of blood gas values (Figures 4 and 5) indicates that anaphylactic shock led to impaired tissue perfusion and metabolic

acidosis. As a compensatory response, hyperventilation reduced $pCO_2$ to maintain acid-base balance. It is, however, important to bear in mind that an elevated lactate level (>4 mmol/L) signifies poor tissue perfusion and shock severity, associated with poor prognosis. Therefore, early recognition of metabolic acidosis to address tissue hypoxia and circulatory failure is crucial to initiate prompt and intensive resuscitation. In the above case scenario, successful shock reversal is reflected in improved blood gas parameters (pH, HCO3–, and lactate levels – Figure 5). If respiratory compensation fails and pH declines further, mechanical ventilation may be required.

- **Complete Blood Count (CBC):**
  - Haemoglobin: 14.5 g/dL
  - Haematocrit: 43% (indicative of polycythaemia)
  - White blood cell count: 12000
  - Platelet count: 178000
- **Electrolytes**
  - Sodium: 139 mmol/l
  - Potassium: 3.8 mmol/l
  - Chloride: 107 mmol/
  - Bicarbonate: 21 mmol/l

### Diagnosis

Anaphylactic Shock with Ventricular Bigeminy – Resolved with Epinephrine.

## DISCUSSION

Anaphylaxis is a systemic, life-threatening hypersensitivity reaction characterized by widespread mast cell and basophil degranulation. The cardiovascular collapse seen in severe cases results from profound vasodilation, increased vascular permeability, and redistribution of intravascular volume.[1] While tachycardia is a common compensatory response, bradyarrhythmias and ventricular ectopy are atypical and may reflect more severe circulatory compromise or direct myocardial involvement.

Ventricular bigeminy, defined as a repeating pattern of a normal sinus beat followed by a premature ventricular complex (PVC), is a rare finding in anaphylaxis and has not been widely reported in existing literature. Its pathogenesis in this context is likely multifactorial. First, **myocardial hypoperfusion**, driven by systemic hypotension, can result in transient ischemia and electrical instability.[2] Second, **histamine and other mediators** released during anaphylaxis can exert direct chronotropic and inotropic effects on cardiac tissue, thereby increasing arrhythmogenic potential.[3] Additionally, the **endogenous catecholamine surge** seen in anaphylaxis may exacerbate myocardial excitability and promote ectopy.[4]

In this case, ventricular bigeminy resolved rapidly following the administration of intramuscular epinephrine, suggesting that the arrhythmia was hemodynamically mediated rather than structural or idiopathic in origin. Epinephrine, through its α1-adrenergic effects, restores systemic vascular resistance and coronary perfusion, while its β1-stimulatory action improves cardiac output and conduction.[5] Although exogenous catecholamines have pro-arrhythmic potential, their net effect in anaphylaxis is often rhythm-stabilizing due to correction of hypoperfusion.

The recurrence of ventricular bigeminy after the initial epinephrine dose, followed by sustained resolution after a second dose, illustrates the dynamic and potentially refractory nature of cardiovascular instability in anaphylaxis. According to current guidelines, **refractory anaphylaxis** is defined as persistent symptoms despite two or more appropriate doses of intramuscular epinephrine.[6] While this case did not meet the strict definition of refractory shock, the arrhythmia recurrence underscores the importance of close hemodynamic and cardiac monitoring during resuscitation.

Few case reports have described ventricular arrhythmias during anaphylaxis, and even fewer have documented bigeminy that resolved with epinephrine alone.[7,8] This highlights a potential gap in the literature regarding arrhythmogenic manifestations of anaphylaxis and supports the incorporation of routine ECG monitoring in patients with bradycardia, hypotension, or atypical presentations.

### Clinical Implications

This case highlights important considerations regarding the cardiovascular manifestations of anaphylaxis and their management:

- **Cardiac arrhythmias may represent an underappreciated component of anaphylaxis.** While hypotension

and airway compromise are the most recognized features, myocardial involvement manifesting as arrhythmias may occur secondary to tissue hypoperfusion, histamine-mediated myocardial stimulation, or catecholamine-induced electrophysiological instability. Continuous ECG monitoring should be considered in all patients presenting with severe anaphylaxis, particularly in those with bradycardia or hypotension.

- **Ventricular ectopy during anaphylaxis may reflect transient, functional myocardial irritability rather than primary cardiac pathology.** In this case, ventricular bigeminy appeared in the absence of structural heart disease and was temporally linked with the hypotensive state. Restoration of sinus rhythm following epinephrine suggests that the ectopy was likely secondary to reversible myocardial hypoperfusion.
- **Epinephrine remains the cornerstone of treatment, even in the presence of arrhythmias.** Although beta-adrenergic stimulation by epinephrine can, in theory, exacerbate arrhythmias, the pathophysiological substrate in anaphylaxis vasodilation and myocardial hypoxia often necessitates its use. In this case, the prompt resolution of ventricular bigeminy with intramuscular epinephrine supports its use as both a hemodynamic stabilizer and an indirect antiarrhythmic agent by improving coronary perfusion and correcting the underlying cause.
- **Early recognition of refractory symptoms is critical.** The recurrence of arrhythmia after initial improvement required timely redosing of epinephrine. This supports current recommendations to repeat IM epinephrine if clinical improvement is incomplete and underscores the importance of close monitoring for evolving or recurrent cardiovascular instability.

Overall, this case reinforces the necessity for vigilant cardiovascular assessment in anaphylaxis, and reaffirms the central role of epinephrine even in cases presenting with bradyarrhythmia or ventricular ectopy as a life-saving intervention.

## LIMITATIONS

Advanced cardiac investigations such as cardiac MRI or Holter monitoring were not performed due to the patient's refusal. Hence, subtle underlying electrophysiological abnormalities cannot be completely ruled out. Additionally, causality between anaphylaxis and the arrhythmia remains presumed but strongly supported by the clinical timeline and therapeutic response.

## CONCLUSION

This case highlights an unusual but clinically significant presentation of ventricular bigeminy during anaphylactic shock in a young, otherwise healthy patient. The arrhythmia resolved completely with intramuscular epinephrine, emphasizing the importance of prompt administration even in the presence of bradyarrhythmia or ventricular ectopy. The case supports the concept that arrhythmias during anaphylaxis may result from reversible myocardial hypoperfusion and histamine-mediated electrophysiological changes. Early recognition, cardiac monitoring, and adherence to anaphylaxis protocols remain essential in ensuring favorable outcomes.

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