KICKOFF KITE SCIENTIFIC FORUM

Acute Decompensated HFrEF with Atrial Fibrillation

KITE Heart Failure Program

KITE

Know the Patient
Interpret the Evidence
Translate into Practice
Elevate Outcomes

Dr Qurban Hussain Khan

Assistant Professor, Cardiac Electrophysiology
Medical Superintendent
Rawalpindi Institute of Cardiology

www.qurbankhan.com

30 July 2026 • Pearl Continental Hotel, Rawalpindi

SESSION MAP

We will follow the KITE format

K

Know the Patient

Clinical phenotype, red flags, context

I

Interpret the Evidence

ECG, imaging, biomarkers, differential

T

Translate into Practice

Emergency actions, GDMT, discharge plan

E

Elevate Outcomes

Follow-up, titration, devices, relapse prevention

Goal: convert one real-world HF admission into a repeatable decision pathway.

LEARNING OBJECTIVES

What the audience should leave with

1. **Spot danger early**

   Wet lungs + low BP + fast AF is not a routine HF admission.

2. **Treat the cause**

   LRTI and AF were the drivers; both needed active treatment.

3. **Start recovery before discharge**

   GDMT begins once perfusion, renal function and electrolytes allow.

Case snapshot: one sentence matters

K KNOW THE PATIENT

58-year-old man with fever, cough, severe dyspnoea and fast irregular pulse.

Infection

Temp 38.5°C
CRP high

Arrhythmia

AF with RVR
HR 140/min

Congestion

Orthopnoea
crackles + JVP

Key question: is this “stable AF + HF”, or is the arrhythmia part of the shock pathway?

KNOW THE PATIENT

The first five minutes: identify the phenotype

38.5°C	142/min	92/58	84%	30/min
Fever	Irregular HR	Blood pressure	Room air SpO_2	Respiratory rate

Examination

- Bilateral basal crackles
- Raised JVP + pedal oedema
- Cool peripheries, delayed capillary refill

Why this is dangerous

- Hypoxaemia + pulmonary oedema
- Fast AF reducing filling time
- Low BP limiting drug choices

Clinical profile: “wet” with threatened perfusion — not a routine rate-control case.

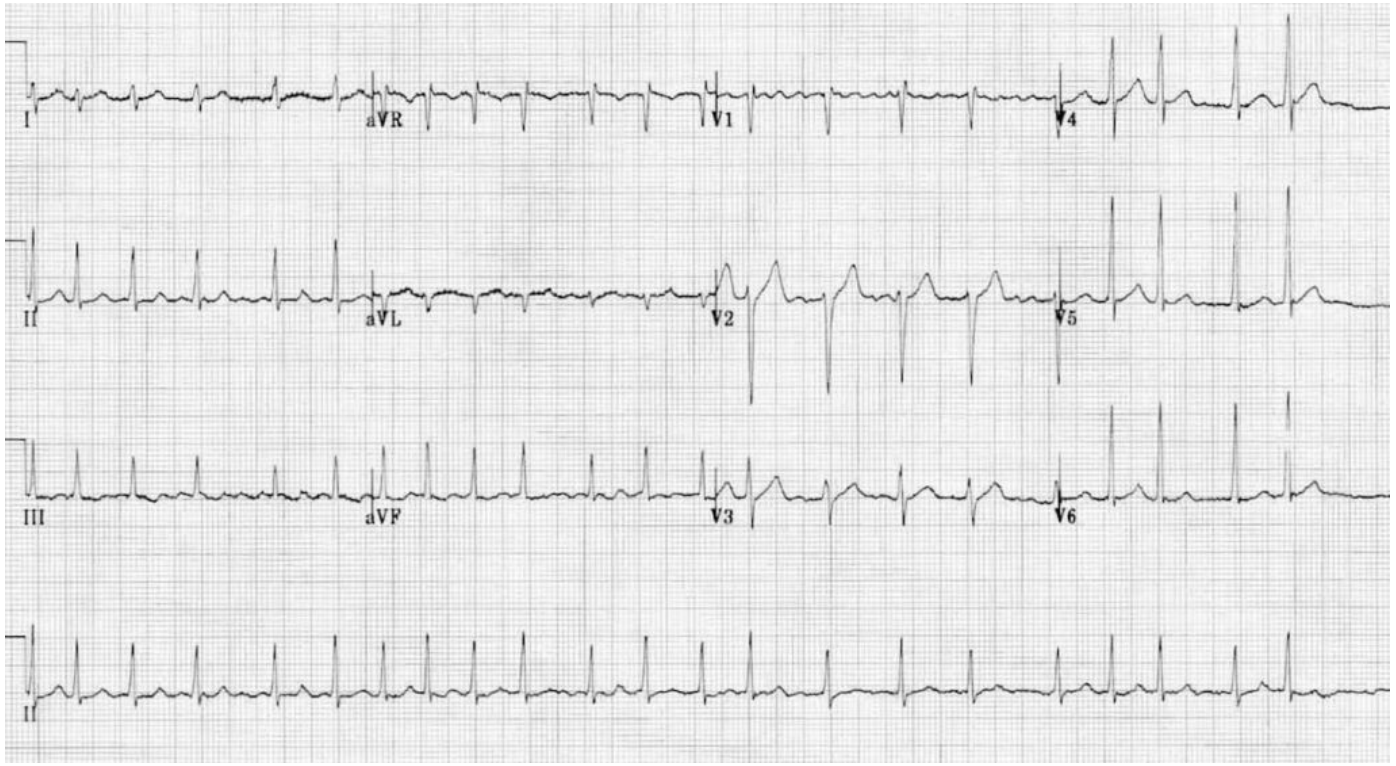
Initial investigations: read them as a pattern

I INTERPRET THE EVIDENCE

NT-proBNP **8,500 pg/mL**	WCC / CRP **14.2 ×10⁹/L / 96 mg/L**	Creatinine / eGFR **1.0 mg/dL / 82**
K^+ / Mg^{2+} **4.1 / 1.8 mmol/L**	Troponin **54 → 52 ng/L**	Lactate **2.3 mmol/L**

Meaning High filling pressures + infection signal + no clear ACS pattern + renal function still usable for treatment.

ECG: the rhythm is part of the haemodynamic problem



Interpretation

- Irregular narrow-complex tachycardia
- No consistent P waves
- Ventricular rate ≈140/min
- No STEMI pattern

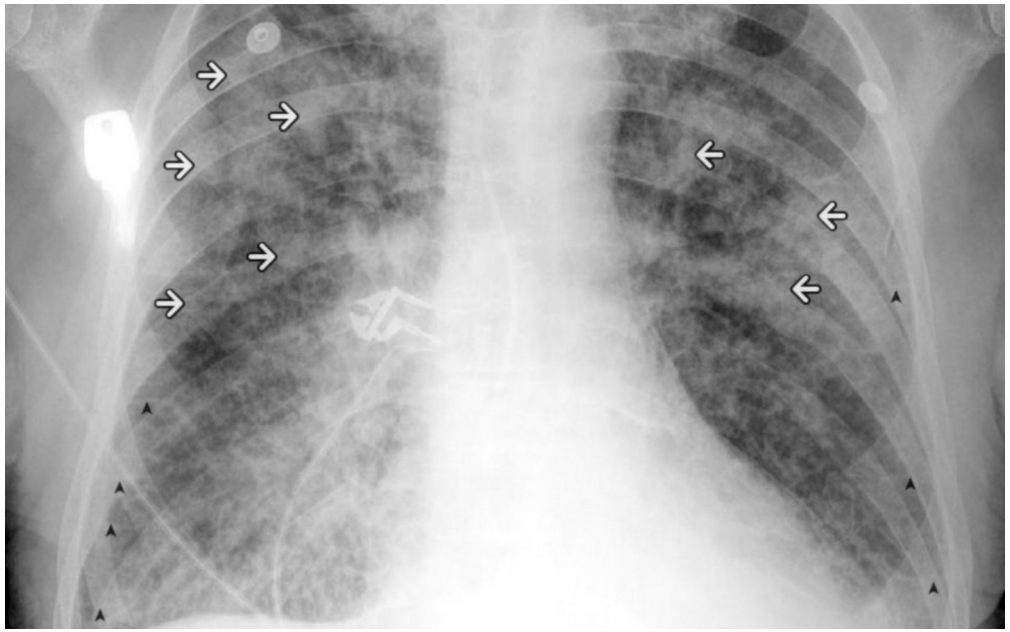
Fast AF

Decision point: rate control, amiodarone, or immediate synchronized cardioversion?

INTERPRET THE EVIDENCE

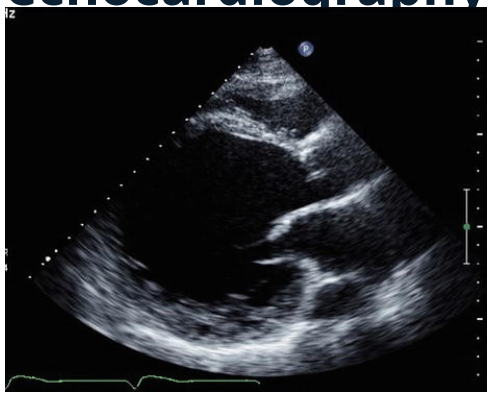
Imaging confirms congested HFrEF physiology

Chest X-ray



Bilateral congestion; no pneumothorax.

Focused echocardiography

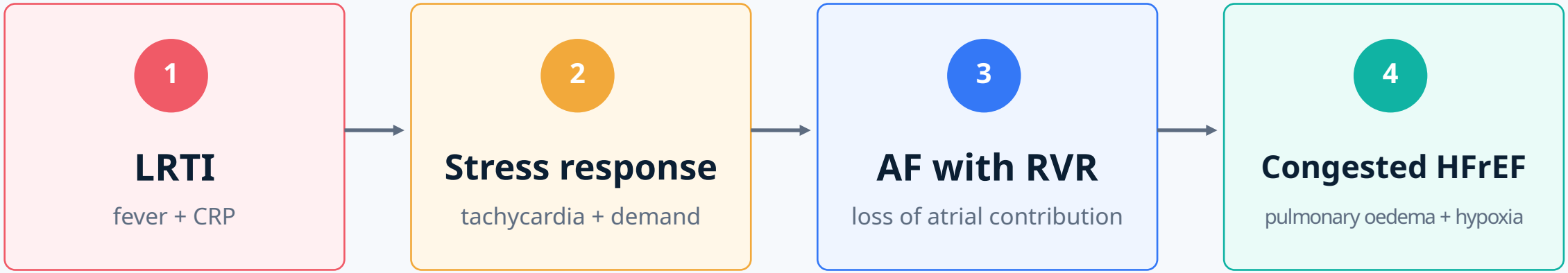


LVEF	30%
LV	Dilated
MR	Mild
RV	Preserved

Systolic dysfunction + congestion + infection + AF = acute decompensated HFrEF.

INTERPRET THE EVIDENCE

Diagnostic synthesis: one pathway, not four diagnoses



Learning point: do not treat AF, pneumonia and HF as separate silos — each worsens the other.

Decision point: should AF be cardioverted now?

T TRANSLATE INTO PRACTICE

The patient is hypoxic, hypotensive and in pulmonary oedema.

Unstable AF

Do not wait for prolonged rate-control attempts.

Synchronized DC cardioversion

Prepare airway, sedation, pads and anticoagulation plan.

Audience vote: What would you do in the next 10 minutes?

TRANSLATE INTO PRACTICE

Emergency management: stabilise first, then refine

A/B

Oxygenation

High-flow O_2; NIV if needed
Target SpO_2 92–96%

C

Circulation

Continuous monitor
Two IV lines; BP q5 min

⚡

Rhythm rescue

Synchronized DC shock
Sedation + airway support

Rx

Treat trigger

IV antibiotics
IV loop diuretic

Avoid reflex IV beta-blocker or diltiazem in a hypotensive patient with acute HFrEF and pulmonary oedema.

TRANSLATE INTO PRACTICE

After rhythm rescue: decongest, protect organs, treat infection

Decongestion strategy

- IV furosemide 80 mg, then response-guided dosing
- Urine output target >100 mL/hour early
- Daily weight + strict fluid balance
- Creatinine/K^{+}/Mg^{2+} every 12–24 h

Infection strategy

- Blood/sputum cultures first if feasible
- Empiric antibiotics within the first hour
- Review at 48–72 h and de-escalate
- Avoid excess IV fluid unless shock persists

TRANSLATE INTO PRACTICE

Before discharge: convert stabilisation into prognosis

Once BP, renal function and potassium are stable, start the four foundations.

ARNI

Sacubitril/valsartan

24/26 mg BD

β-blocker

Bisoprolol

1.25 mg OD

MRA

Spironolactone

25 mg OD

SGLT2i

Dapagliflozin

10 mg OD

AF anticoagulation

CHA_2DS_2-VASc = 3 → oral anticoagulation unless contraindicated.

TRANSLATE INTO PRACTICE

The discharge prescription is only half the plan

1

Self-care

Daily weight
Salt awareness
Fluid plan

2

Safety labs

Creatinine
K^+ / Mg^{2+}
BP diary

3

Follow-up

2 weeks
Titrate therapy
Check rhythm

4

Red flags

Weight gain
Orthopnoea
Syncope

Discharge is a handover: patient, family, physician and cardiologist must know the next step.

Follow-up: measure recovery, do not assume it

E

ELEVATE OUTCOMES

Day 0

85 kg
NT-proBNP 8,500
LVEF 30%

→

2 weeks

79 kg
NT-proBNP 2,800
NYHA II

→

8 weeks

78.8 kg
NT-proBNP 650
LVEF 35–38%

Biomarkers and echo are not trophies — they guide titration, rhythm strategy and device timing.

ELEVATE OUTCOMES

What if LVEF remains ≤35% after optimisation?

Reassess after at least 3 months of optimised therapy.

ICD

Primary prevention if EF remains low and survival/function justify it.

CRT

If QRS is wide, especially LBBB morphology.

AF pathway

If AF recurs: anticoagulation, rhythm strategy, ablation discussion.

Do not decide device therapy before the recovery window unless there is another urgent indication.

Why this case needs two disciplines

Cardiology / EP lens

- Haemodynamic impact of AF
- Cardioversion and anticoagulation
- Decongestion and GDMT sequencing
- ICD/CRT and rhythm-control pathway

Internal medicine lens

- Infection source and severity
- Antimicrobial stewardship
- Renal, electrolyte and metabolic monitoring
- Safe transition to community care

ELEVATE OUTCOMES

Five learning points to retain

1. **Recognise the phenotype** — Congestion + hypoxaemia + low BP = high-risk acute HF.
2. **AF may be the emergency** — Unstable AF in pulmonary oedema needs urgent synchronized cardioversion.
3. **Treat the trigger** — Infection, arrhythmia and HF must be managed together.
4. **Start the four foundations** — ARNI/ACEi, β-blocker, MRA and SGLT2i before discharge when safe.
5. **Follow-up is treatment** — Early review, labs, titration and repeat EF determine long-term outcome.

KNOWLEDGE CHECK

Three questions before we close

1 **In unstable AF with pulmonary oedema, should cardioversion wait for 48 h anticoagulation?**

No — instability takes priority; anticoagulate as soon as feasible.

2 **When should beta-blocker be started in this admission?**

After haemodynamic stabilisation and adequate decongestion.

3 **Should SGLT2 inhibitor stop once NT-proBNP improves?**

No — continue unless contraindication or intolerance develops.

Thank you

Questions • Discussion • Practice change