Clinical Setting: 68 y F who presents with agitation, with a reported intentional benzodiazepine overdose.

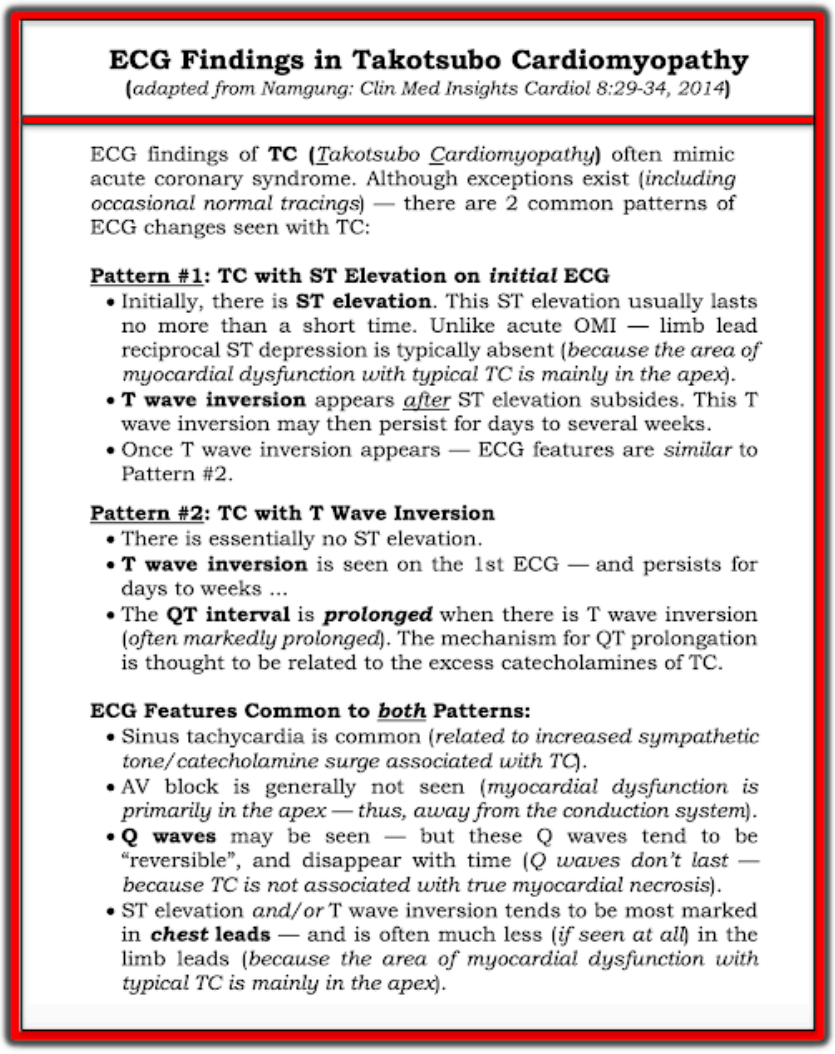
HR - 70
BP - 175/110
RR - 20
SpO2 - 97%
Temp - 99.2 F

This is a case of **Takotsubo Cardiomyopathy.**

What stands out here - the absolute massive T wave inversions. Looking through the whole tracing, there is a sinus rhythm, normal rate. Normal axis, normal appearing QRS complexes. The T waves are inverted throughout, and MASSIVELY huge. This causes significantly prolonged QTc as well.

With these giant T inverted T waves, there are only a couple options. Cardiomyopathy, typically stress cardiomyopathy, can cause this. CNS catastrophe (large subarachnoid hemorrhage) is the other main item on the differential. See the link below for Dr. Smith's take on this case.

Takotsubo can present in 2 main ways - like this EKG, and also like a STEMI - and these can be indistinguishable via EKG, and sometimes on ECHO (a wraparound LAD lesion can cause the typical "apical ballooning" seen in Takotsubo).



ECG Findings in Takotsubo Cardiomyopathy

(*adapted from Namgung: Clin Med Insights Cardiol 8:29-34, 2014*)

ECG findings of **TC** (*Takotsubo Cardiomyopathy*) often mimic acute coronary syndrome. Although exceptions exist (*including occasional normal tracings*) — there are 2 common patterns of ECG changes seen with TC:

Pattern #1: TC with ST Elevation on *initial* ECG

- Initially, there is **ST elevation**. This ST elevation usually lasts no more than a short time. Unlike acute OMI — limb lead reciprocal ST depression is typically absent (*because the area of myocardial dysfunction with typical TC is mainly in the apex*).
- **T wave inversion** appears *after* ST elevation subsides. This T wave inversion may then persist for days to several weeks.
- Once T wave inversion appears — ECG features are *similar* to Pattern #2.

Pattern #2: TC with T Wave Inversion

- There is essentially no ST elevation.
- **T wave inversion** is seen on the 1st ECG — and persists for days to weeks ...
- The **QT interval** is ***prolonged*** when there is T wave inversion (*often markedly prolonged*). The mechanism for QT prolongation is thought to be related to the excess catecholamines of TC.

ECG Features Common to *both* Patterns:

- Sinus tachycardia is common (*related to increased sympathetic tone/catecholamine surge associated with TC*).
- AV block is generally not seen (*myocardial dysfunction is primarily in the apex — thus, away from the conduction system*).
- **Q waves** may be seen — but these Q waves tend to be "reversible", and disappear with time (*Q waves don't last — because TC is not associated with true myocardial necrosis*).
- ST elevation *and/or* T wave inversion tends to be most marked in ***chest* leads** — and is often much less (*if seen at all*) in the limb leads (*because the area of myocardial dysfunction with typical TC is mainly in the apex*).

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(case source - Link to Dr Smith's Blog for this case)