

## **Debate**

**PVT – Cause or Consequence of Decompensation?**

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PVT in cirrhosis: more often a consequence than a cause of decompensation

## Results from a prospective cohort (2011-2021)

- ❖ 367 rigorously defined cACLD patients followed up to 10 years.
  - ❖ **102 developed decompensation**
  - ❖ **22 developed non-malignant PVT**
- ❖ Among the 15 patients who developed both events:
  - ❖ **Decompensation first: 8/15**
  - ❖ **PVT first: 5/15**
  - ❖ **Simultaneous: 2/15**

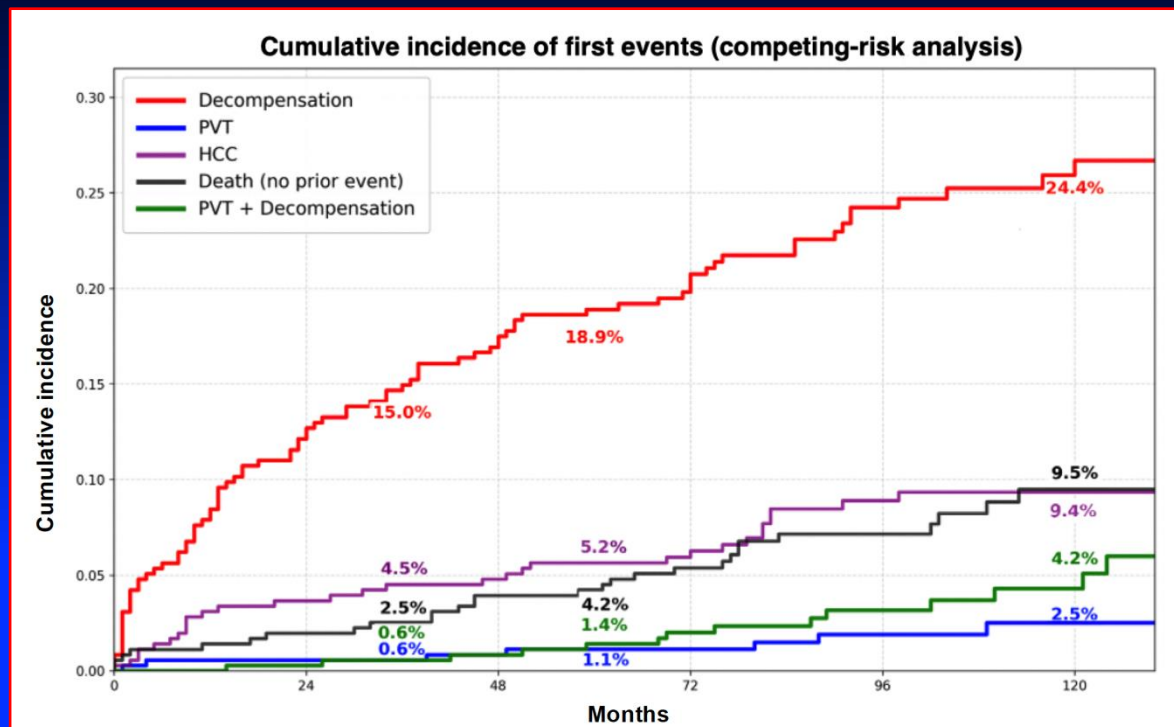
# The chronology supports consequence, not cause

## First argument: timing

Competing-risk analysis shows that decompensation is the dominant first event.

At 10 years:

- Decompensation: 24.4%
- Isolated PVT: 2.5%
- PVT + decompensation: 4.2%



The visual separation of the curves strongly supports decompensation first, PVT later.

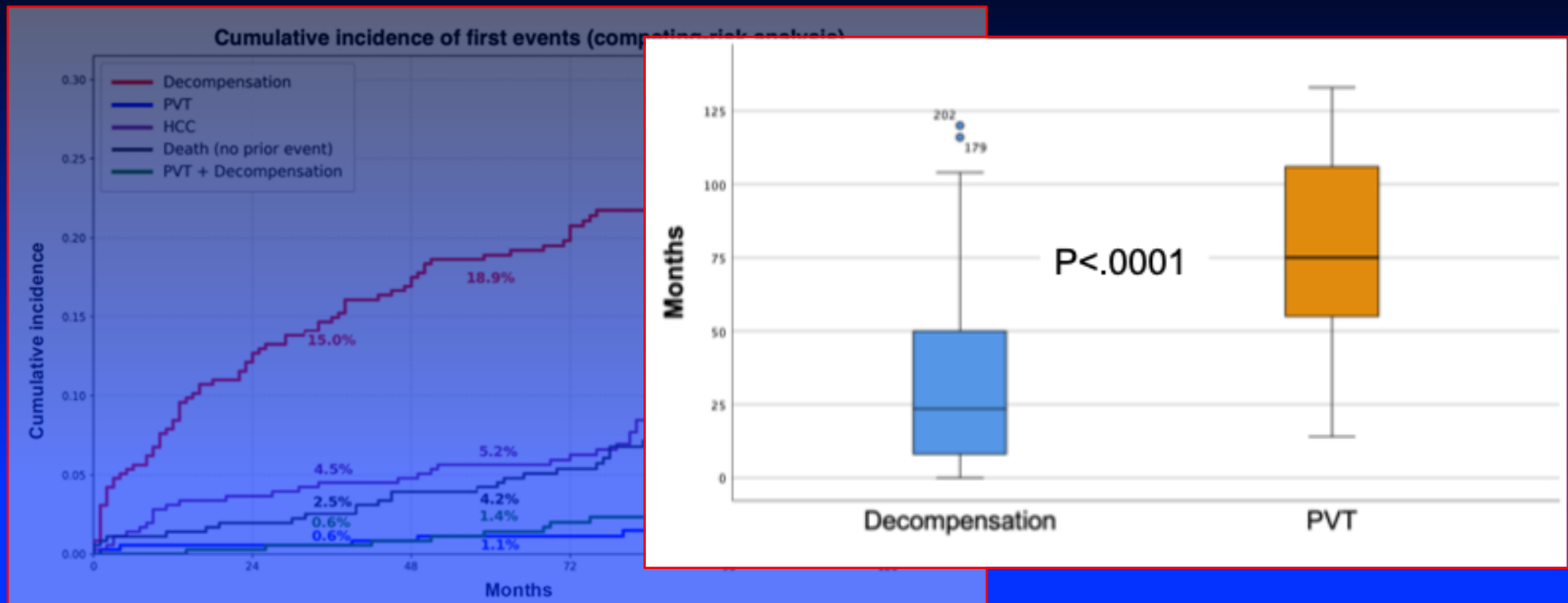
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Decompensation occurred significantly earlier and exhibited greater temporal variability, whereas PVT presented later and within a narrower time window.

# The biology also supports consequence

## Second argument: biology

- Decompensation showed **broad upstream multisystem dysregulation** already at baseline: angiogenesis, endothelial dysfunction, fibrogenesis, inflammation, hypoxia/metabolic stress, gut barrier failure
- Isolated PVT showed a **restricted signature**: mainly **Tie2** and **mesothelin**.

# The biology also supports consequence

## Second argument: biology

**PVT  
Restricted  
signature:  
mainly Tie2 and  
mesothelin.**

|                | 10-yr isolated PVT | 10-yr Decomp + PVT | 3-yr Isolated<br>Decompensation | 10-yr Isolated<br>Decompensation |
|----------------|--------------------|--------------------|---------------------------------|----------------------------------|
| Angiopoietin-1 | 0,944              | 0,001              | 0,001                           | 0,001                            |
| Angiogenin     | 0,820              | 0,513              | 0,001                           | 0,001                            |
| Angiopoietin-2 | 0,579              | 0,653              | 0,001                           | 0,001                            |
| IL-6           | 0,114              | 0,747              | 0,001                           | 0,001                            |
| CA9            | 0,207              | 0,007              | 0,001                           | 0,001                            |
| VEGFD          | 0,119              | 0,976              | 0,001                           | 0,001                            |
| HA             | 0,206              | 0,029              | 0,001                           | 0,001                            |
| TPO            | 0,464              | 0,009              | 0,002                           | 0,001                            |
| TGFβ1          | 0,505              | 0,183              | 0,001                           | 0,001                            |
| Ecad           | 0,674              | 0,241              | 0,003                           | 0,001                            |
| I_FABP         | 0,309              | 0,924              | 0,001                           | 0,003                            |
| NECTIN-2       | 0,647              | 0,001              | 0,202                           | 0,006                            |
| COMP           | 0,373              | 0,620              | 0,009                           | 0,006                            |
| IL-8           | 0,707              | 0,846              | 0,723                           | 0,007                            |
| MMP-7          | 0,259              | 0,340              | 0,316                           | 0,013                            |
| TNF-α          | 0,069              | 0,791              | 0,013                           | 0,016                            |
| Endoglin       | 0,218              | 0,179              | 0,763                           | 0,031                            |
| CRP            | 0,932              | 0,281              | 0,461                           | 0,032                            |
| VEGFA          | 0,676              | 0,872              | 0,001                           | 0,034                            |
| IL-1           | 0,128              | 0,873              | 0,584                           | 0,052                            |
| vWF            | 0,205              | 0,580              | 0,060                           | 0,066                            |
| ANGPTL4        | 0,083              | 0,001              | 0,740                           | 0,081                            |
| ADMA           | 0,070              | 0,139              | 0,053                           | 0,095                            |
| IL-10          | 0,757              | 0,889              | 0,110                           | 0,133                            |
| MMP-9          | 0,685              | 0,296              | 0,012                           | 0,172                            |
| OPN            | 0,450              | 0,378              | 0,434                           | 0,196                            |
| SPINK1         | 0,507              | 0,178              | 0,418                           | 0,058                            |

Decompensation  
angiogenesis,  
endothelial  
dysfunction,  
fibrogenesis,  
inflammation,  
hypoxia/metabolic  
stress, gut barrier  
failure

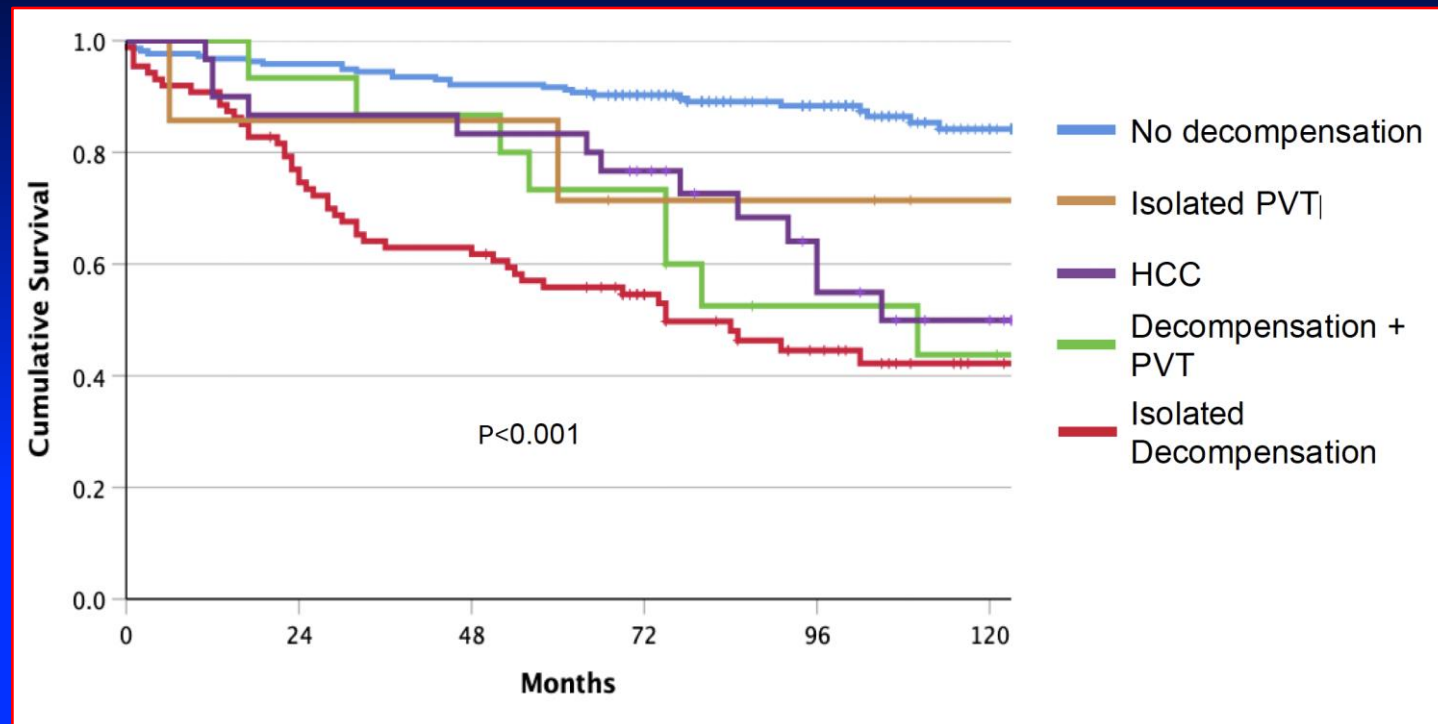
Decompensation is the **biologically active transition**; PVT is more consistent with a **late flow-related complication**.

|            |       |       |       |       |
|------------|-------|-------|-------|-------|
| SERPINA1   | 0,393 | 0,491 | 0,259 | 0,555 |
| Mesothelin | 0,027 | 0,038 | 0,470 | 0,848 |
| VEGFR-2    | 0,327 | 0,007 | 0,660 | 0,870 |
| Tie2       | 0,016 | 0,101 | 0,433 | 0,934 |

# Prognosis clearly favors decompensation as the key event

## Third argument: prognosis

- Decompensation was the **strongest determinant of mortality**.
- Early mortality risk with decompensation: **HR 11.86**
- HCC also mattered: **HR 4.40**
- PVT showed **no independent short- or long-term effect on survival**.
- Patients with isolated PVT had outcomes closer to stably compensated patients than to decompensated patients.



# PVT in cirrhosis: more often a consequence than a cause of decompensation

- ❖ In cACLD, decompensation is the earlier and dominant clinical transition.
- ❖ PVT occurs usually later, less frequent, and biologically more restricted.
- ❖ Therefore, PVT is better viewed primarily as a downstream manifestation of disease progression

## Take-home message:

- ❖ In cACLD, PVT is usually a consequence of progressive portal-hypertensive and systemic deterioration, not the primary cause of decompensation.

Thanks