



# Acute hepatitis after paracetamol poisoning: Analysis of potentially influencing factors

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#### **Background and Importance**



Paracetamol poisoning (PP) can lead to acute hepatitis: acute inflammation and necrosis of the liver parenchyma.



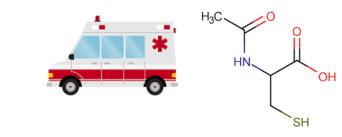
Inflammatory parameters usually begin to rise within 48-72h. An unfavourable course of acute hepatitis predisposes to acute liver



#### failure.

### Aim and Objectives

- Analysis of the prevalence of acute hepatitis after PP
- To identify which epidemiological and intoxication-associated variables are related to the development of AH



## **Materials and Methods**

**Observational**, descriptive and retrospective study

**January 2019 – August 2024** 

**Patients treated with N-acetylcysteine for PP in Emergency Department** 

Acute hepatitis was
defined as

- Non-specific gastrointestinal symptoms / asthenia /
- abdominal pain
- Elevated aspartate-aminotransferase enzyme, bilirrubin >2.5mg/dL
  - INR > 1.5

Analytical test conducted at the conclusion of the NAC perfusion and 48 hours post-poisoning was reviewed



Variables under examination



✓ Age, sex

- ✓ Potential risk factors for paracetamol hepatotoxicity
- ✓ Massive paracetamol intake, time between PP and starting of NAC treatment
- ✓ Variables related to a correct management of NAC (dosage, preparation, administration, and infusion duration)



**Onset of acute hepatitis** 

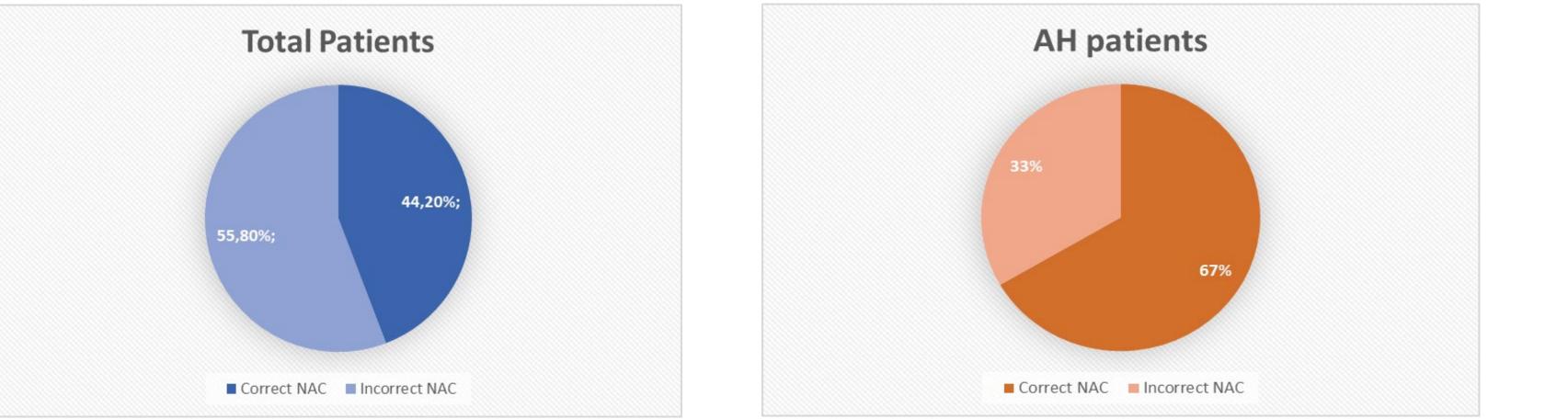




The prevalence of patients with acute hepatitis was 6.9% (3/43) A total of 20.9% patients presented with PRFPH, none of them developed acute hepatitis

Analysis of variables related to use of NAC

NAC was inappropriately used in 55.8% of total patients and in 66.6% of AH patients (p>0.05)





>30g of paracetamol and the start of NAC was 8 hours after intoxication (p=0.003)

#### **Conclusion and Relevance**

>Acute hepatitis was predominantly observed in young female patients.  $\succ$  The percentage of NAC errors was high, particularly among the acute hepatitis patients. > There was a statistically significant association between AH and both massive intakes and delayed initiations of NAC. >Further study of more patients may help to clarify this correlation.

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