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РОПНИЗ

OVERLAP SYNDROME BETWEEN **NSAID**-INDUCED GASTROENTEROPATHY AND **DGBI**

52

patients

49 y

mean age

44.2%

DGBI features

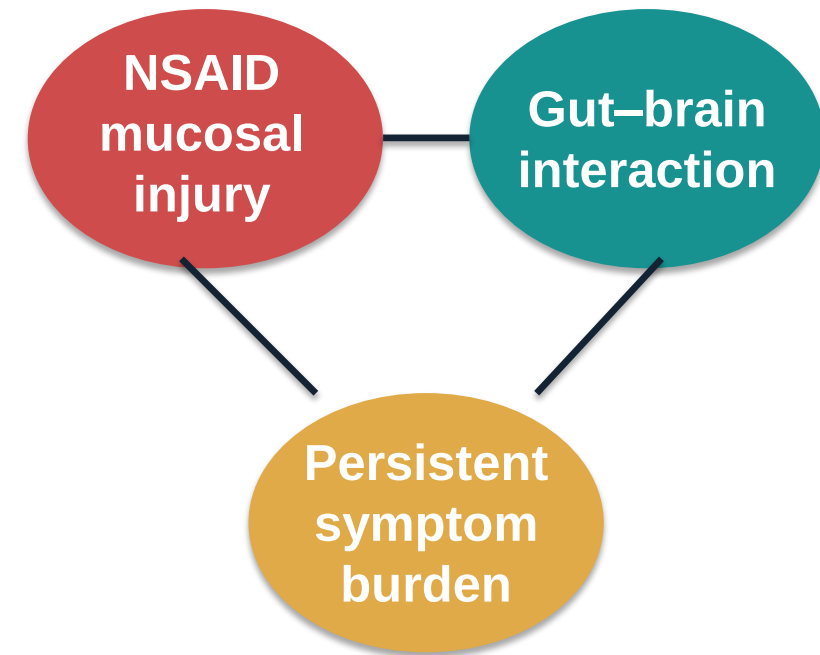
GUT ↔ BRAIN

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Two interacting sources of symptoms

- NSAIDs can injure the upper and lower GI tract, causing dyspepsia, ulceration, bleeding and enteropathy.
- Symptom intensity does not always parallel the extent of mucosal injury.
- DGBI may coexist with organic disease and amplify pain, nausea, bloating and bowel symptoms.
- Rome V (launched in 2026) emphasizes clinical context, bothersomeness, function and patient experience.

Conceptual model



Aim: quantify and characterize DGBI overlap in NSAID-induced gastroenteropathy.



Cohort profile

49 y

mean age

3 : 2

male : female

DGBI phenotypes focused on functional dyspepsia (FD), irritable bowel syndrome (IBS), and upper–lower GI symptom coexistence. Assessment considered symptom characteristics, frequency, bothersomeness and functional impact.

RESULTS

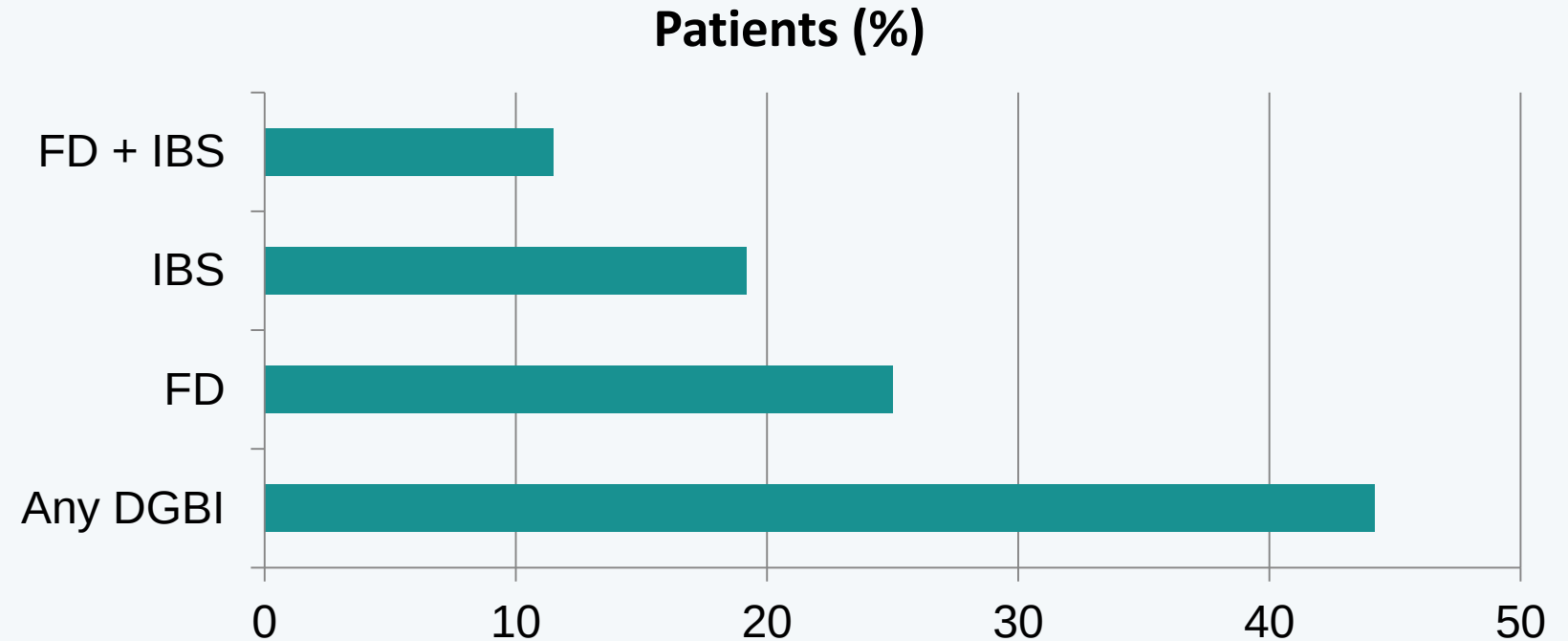
DGBI overlap was common

23 / 52

44.2%

**patients with
DGBI features**

Clinical implication:
symptoms may
reflect both mucosal
injury and altered
gut–brain interaction.



FD was identified in 13 patients (25.0%); IBS in 10 (19.2%). FD + IBS overlap occurred in 6 (11.5%). Categories are not mutually exclusive, so values should not be summed.

Percentages based on 52 patients • FD = functional dyspepsia • IBS = irritable bowel syndrome

Symptoms more prominent with DGBI overlap

Abdominal
pain /
discomfort

Postprandial
fullness

Early
satiation

Bloating

Nausea

Altered
bowel
habits

Bidirectional symptom model

NSAID
exposure

Mucosal
injury

Sympt
oms

Gut-brain
interaction

Visceral
sensitivity

When symptom burden exceeds what endoscopy alone would predict, DGBI provides a clinically useful second explanatory pathway.

Key conclusions

- DGBI features were present in 44.2% of patients with NSAID-induced gastroenteropathy.
- FD and IBS were the leading phenotypes; 11.5% had FD–IBS overlap.
- Persistent symptoms should not automatically be attributed to the visible severity of mucosal injury.
- Rome V supports patient-centered assessment of symptoms, bothersomeness and functional impact.
- Management should address both mucosal risk and gut–brain mechanisms, individualized to the patient.

Clinical pathway

- 1 Assess NSAID exposure and GI risk
- 2 Characterize persistent symptom pattern
- 3 Consider DGBI overlap when appropriate
- 4 Treat both pathways and reassess function

Take-home: 44.2% overlap