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Second Asian Consensus Report on Functional Dyspepsia (2025): Updated Recommendations

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ABSTRACT

Background: Functional dyspepsia (FD) is a common gastrointestinal disorder that significantly impacts patients' quality of life. Over a decade ago, the Asian Neurogastroenterology and Motility Association (ANMA) and the Asian Pacific Association of Gastroenterology (APAGE) jointly developed the first Asian consensus report on FD. In this consensus report, members of ANMA and APAGE provide updated recommendations on the definition, diagnosis, epidemiology, pathophysiology, and management of FD, focusing on Asian populations.

Methods: The task force members conducted a systematic literature review and used a modified Delphi process to develop updated consensus statements. Based on members' feedback, statements that failed to reach at least 80% consensus in the first round of voting were revised. Revisions included rephrasing for clarity, incorporating additional evidence, and subgroup voting during a second round of discussion at a hybrid meeting.

Results: The task force developed 32 statements covering key aspects of FD. Major updates include new insights into the pathophysiology and emerging treatment options. The task force acknowledged that the limited scope and heterogeneity of available studies limit definitive conclusions about the utility of some emerging therapies such as probiotics and potassium-competitive acid blockers in FD management.

Conclusions: The second Asian Consensus Report on FD provides updated evidence-based recommendations to improve the diagnosis and management of FD in clinical practice, particularly in the Asian setting.

1 | Introduction

Dyspepsia remains one of the most common disorders encountered in clinical practice [1, 2]. Over the recent decades, its definition has evolved from any symptom felt to be attributable to the stomach and duodenum to one that includes only the cardinal symptoms of epigastric pain or burning, postprandial fullness, or early satiation [3].

Functional dyspepsia (FD) is diagnosed in the absence of structural, organic, systemic, or metabolic abnormalities that would explain these symptoms [2, 3]. FD is one of the most extensively

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studied disorders of gut–brain interaction (DGBI), previously known as functional gastrointestinal disorders (FGIDs), and is characterized by chronic or relapsing gastrointestinal symptoms without an identifiable organic cause [4, 5].

Many studies on FD pathophysiology have been conducted in Western populations [6]. FD pathophysiology is generally believed to be more prevalent in Western populations than in Eastern populations, with different subtypes dominating: Western patients more often exhibit ulcer-like and reflux-like symptoms, while Eastern patients predominantly experience dysmotility-type or postprandial distress syndrome (PDS) symptoms, including early satiety, bloating, and belching. Additional distinctions include variations in body mass index (BMI), socioeconomic associations, dietary habits, and a greater magnitude of symptom improvement after *Helicobacter pylori* eradication in Eastern populations [6]. Although differences in clinical features and epidemiology between Asian and Western FD patients have previously been reported, these differences have not been sufficiently addressed, highlighting the need for region-specific guidelines [6, 7]. Furthermore, healthcare systems differ significantly between Asia and the West. Hence, clinical management strategies developed elsewhere may not be appropriate in Asia [8].

To address the need for an Asian perspective to better understand the pathogenesis of FD, the Asian Neurogastroenterology and Motility Association (ANMA) and the Asian Pacific Association of Gastroenterology (APAGE) jointly generated the first Asian consensus report on FD in 2012 [2]. The joint consensus aimed to describe the experiences and views of Asian experts and to provide a relevant guide on the management of FD for primary care physicians working in Asia. It also provided critical insights by integrating local data to enhance the management of FD specific to the Asian population [2].

Substantial advances in the field, particularly in Asia, have occurred since the first Asian consensus report on FD was published over a decade ago. Therefore, an update is warranted to reflect a renewed understanding of FD tailored to the unique challenges faced by physicians in this region. The 2025 consensus report aims to update the 29 consensus statements from the 2012 report to reflect advances in the understanding of the pathophysiology of FD, describe updated diagnostic criteria, and identify emerging treatment options [2].

2 | Methodology

2.1 | Consensus Development Group

Both ANMA and APAGE agreed to update the 2012 Asian consensus report on FD. A total of 29 members, including gastroenterologists, epidemiologists, and practitioners of traditional, complementary, and integrative medicine (TCIM), were invited to form a joint task force. This ensured a multidisciplinary approach, with representatives from various countries in the Asia-Pacific region bringing a broad range of expertise in clinical practice. This diverse representation also underscores the commitment to ensure culturally relevant and regionally applicable recommendations.

Similar to the 2012 report, this task force was organized into four teams to discuss and propose consensus statements related to definition and diagnosis, epidemiology, pathophysiology, and management [2].

2.2 | Evidence Synthesis

Task force members performed a systematic literature review according to the general principles proposed in the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement [9].

A comprehensive literature search was conducted using databases such as Embase, Global Health, Ovid Emcare, Ovid MEDLINE, Ovid Nursing Database, and APA PsycINFO, covering publications of studies of human subjects published in English between January 2013 and March 2024. Publications outside this period were also considered if relevant studies were not found within the specified period.

The methodological quality of the identified studies was assessed according to the method of the Grades of Recommendation, Assessment, Development, and Evaluation (GRADE) Working Group, where applicable [10].

2.3 | Modified Delphi Consensus

Task force members used a modified, two-stage Delphi process to develop, refine, and achieve consensus [11].

The task force members generated 35 consensus statements through in-person and virtual meetings. During the first consensus meeting (December 9, 2023; Bangkok, Thailand), the task force conducted an in-depth discussion of each candidate statement and proposed changes. The statements were subjected to a first round of online voting. Members were invited to review the draft statements and provide their votes and comments. Each member was then asked to choose one of the following six levels of agreement for each statement (Table 1): (A) agree completely, (B) agree with minor reservation, (C) agree with major reservation, (D) disagree with major reservation, (E) disagree with minor reservation, and (F) disagree completely. Members were also asked to add comments, if any, for each statement. All votes were anonymous. Agreement with a statement by at least 80% of the task force (i.e., agree completely or agree with minor reservation) was defined a priori as consensus.

On March 16, 2024, the consensus task force convened for a second meeting in Kuala Lumpur, Malaysia. The outcomes from the first online vote were discussed. Twenty-four statements were accepted, three were rejected, and the remaining eight failed to reach the consensus level.

For the eight statements on which a consensus had yet to be reached, the task force members discussed and modified them based on input from the second meeting and then subjected them to a second round of voting. Consensus was eventually achieved for these eight remaining statements.

TABLE 1 | Grade of evidence and level of agreement.

Category	Description
Grade of evidence	
High	Further research is unlikely to change our confidence in the estimate of effect
Moderate	Further research is likely to have an important impact on our confidence in the estimate of effect and might change the estimate
Low	Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate
Very low	Any estimate of effect is very uncertain
Level of agreement[a]	
A	Agree completely
B	Agree with minor reservation
C	Agree with major reservation
D	Disagree with major reservation
E	Disagree with minor reservation
F	Disagree completely

[a]Each statement was accepted when 80% or more of participants voted A or B.

3 | Results

The systematic literature search identified 914 citations. After removing duplicates, 843 articles were reviewed. The task force's recommendations were divided into four key areas, comprising 32 statements, covering definition and diagnosis (Statements 1–3), epidemiology (Statements 4–10), pathophysiology (Statements 11–19), and management (Statements 20–32).

4 | Key Recommendations

4.1 | Definition and Diagnosis

Statement 1. Dyspepsia refers to a set of symptoms of primarily gastroduodenal origin, which includes epigastric pain, epigastric burning, postprandial fullness, early satiety, bloating, and nausea. FD is a disorder characterized by chronic dyspeptic symptoms in the absence of an organic explanation after clinical investigations.

Grade of evidence: Not applicable.

Level of agreement: A = 84%, B = 12%, C = 0%, D = 0%, E = 0%, F = 4%.

Statement 1 considers several observations and the latest definitions about dyspepsia and FD [6, 12].

The Rome IV criteria provide the most frequently used definition for DGBI. Currently, they classify FD into two types, that is, PDS or epigastric pain syndrome (EPS). PDS is associated with meal-induced dyspeptic symptoms such as postprandial fullness and early satiation, while EPS commonly presents as epigastric pain or burning sensation [3]. The supportive criteria for FD in Rome IV highlight that bloating, belching, and nausea may also be present in both subtypes [3, 4]. In particular, nausea is now recognized as a highly prevalent symptom in FD, with a higher prevalence in the EPS–PDS overlap group [13]. Most task force members agreed that meal-related symptoms (i.e., PDS) may occur in up to 70% of FD cases using the Rome IV criteria, while a smaller proportion of cases may not be meal-related (i.e., EPS). However, the EPS–PDS overlap subtype has also been recognized, because PDS and EPS symptoms may overlap in up to 20% of patients, based on the Rome IV criteria [14–17].

While the Rome IV criteria provide more specific diagnostic requirements for FD, some are considered potentially restrictive. For example, the duration of symptoms is 3 months, with PDS symptoms occurring three times a week and EPS symptoms occurring once per week, and the onset is at least 6 months before diagnosis. These criteria may limit the applicability of classifying FD as EPS or PDS subtypes in routine clinical practice and delay diagnosis and subsequent treatment [17–19].

Statement 2. Diagnosis of FD should be based on validated symptom criteria and appropriate investigations.

Grade of evidence: Low.

Level of agreement: A = 56%, B = 24%, C = 20%, D = 0%, E = 0%, F = 0%.

The task force members stressed the importance of relying on validated symptom criteria tailored to the specific patient population to ensure accurate diagnosis and appropriate management of FD [14].

The Rome IV Diagnostic Questionnaires were developed as a standardized tool for screening DGBI, which includes FD, and have been demonstrated to possess global validity [20, 21]. Although these questionnaires are invaluable for research and epidemiological studies, their complexity has hindered their widespread adoption in clinical practice.

For Asian countries where there are high incidences of gastric cancer, such as China, Japan, and Korea, and peptic ulcer, such as Bangladesh and parts of India, additional investigations are crucial to rule out organic causes [22–24]. Depending on local resources and epidemiology, appropriate initial investigations may include endoscopy, CT scan, complete blood count and blood biochemistry tests, *H. pylori* test, stool examination, and upper abdominal ultrasound before FD is diagnosed [25].

Statement 3. Endoscopy must be performed in the presence of alarm features and should be considered in populations with a high prevalence of upper gastrointestinal malignancy/organic gastrointestinal disease.

Grade of evidence: Moderate.

Level of agreement: A = 92%, B = 4%, C = 4%, D = 0%, E = 0%, F = 0%.

Although many patients are hesitant to undergo endoscopy, most task force members agreed it is a useful and critical diagnostic tool for determining an organic etiology for dyspepsia. Relying solely on alarm features and age is insufficient for diagnosing organic diseases in populations with a high prevalence of upper gastrointestinal malignancy/organic disease [26, 27]. It is imperative to have supporting evidence, such as results from appropriate investigations, to substantiate the diagnosis [28].

Nevertheless, the task force members suggest that healthcare providers should aim to diagnose FD in patients presenting with symptoms (e.g., postprandial fullness, early satiation, epigastric pain, and epigastric burning) in the clinic and treat it accordingly, with or without endoscopy findings, because FD is a positive diagnosis rather than a diagnosis based on exclusion. While endoscopy may help diagnose FD, its primary role is to rule out an organic disease [29].

4.2 | Epidemiology

Statement 4. FD is common in Asia.

Grade of evidence: Low.

Level of agreement: A = 68%, B = 20%, C = 8%, D = 0%, E = 0%, F = 4%.

The task force members agreed that it is important to increase awareness that FD is common and should be considered in patients presenting with suggestive symptoms.

A systematic review with meta-analysis of 67 studies encompassing 98 populations and 338 383 subjects investigated the global prevalence of uninvestigated dyspepsia, of which up to 80% are labeled as FD. Asian prevalence ranged dramatically, from 2.4% in Japan to 33% in Singapore, likely influenced by data collection methods, recruitment bias, and the differences in diagnostic criteria (Rome II, III, or IV). Nevertheless, the results confirmed the magnitude of FD within the global community and its concomitant implications for health services. This is particularly concerning in regions with limited access to healthcare, which may further highlight the unmet needs in FD management [30].

These findings reaffirmed the results of a large-scale multinational study involving 73 076 adults from 33 countries, which investigated the prevalence of FGIDs. Results showed that 40.3% of internet survey respondents and 20.7% of household survey respondents met at least one FGID criterion, with a higher prevalence among women [1].

It is important to understand that FD is common in Asia because patients suffering from this disease generally have an impaired quality of life [29]. Knowing that FD is common in the community will help raise awareness about the disease, improve management outcomes, and improve patients' quality of life.

Statement 5. FD commonly overlaps with other disorders of gut–brain interaction.

Grade of evidence: Low.

Level of agreement: A = 88%, B = 12%, C = 0%, D = 0%, E = 0%, F = 0%.

FD has many overlapping symptoms with other DGBI, for example, irritable bowel syndrome (IBS) and functional heartburn [31].

Overlapping of DGBI is known to impact patient outcomes and result in poorer quality of life, because this may result in more severe symptoms as well as an increased risk of experiencing anxiety, depression, or insomnia compared with patients without overlapping diseases [4, 5, 32].

Statement 6. FD commonly overlaps with gastroesophageal reflux disease.

Grade of evidence: Moderate.

Level of agreement: A = 68%, B = 20%, C = 8%, D = 0%, E = 0%, F = 4%.

Patients with FD show particularly frequent overlap with gastroesophageal reflux disease (GERD), with studies showing up to 30% overlap in those with GERD and those with dyspepsia/FD [4, 33].

When considering the symptomatic overlap between GERD and FD, a study of 439 patients in Vietnam found that the disease overlap was more common than either disorder alone, occurring in 51.3% of patients. The research also revealed that PDS was significantly more prevalent in patients with GERD–FD overlap versus those with FD only (72.9% vs. 44.2%, $p < 0.001$) [34].

Meanwhile, a prospective, multicenter cross-sectional study in Japan investigated factors associated with the overlap between FD and nonerosive reflux disease in *H. pylori*–uninfected individuals. The study identified several factors, including female sex, younger age, and specific dietary habits, associated with this overlap, further supporting the frequent co-occurrence of these conditions [35].

Statement 7. There is an association between psychological comorbidity and FD.

Grade of evidence: Moderate.

Level of agreement: A = 84%, B = 12%, C = 0%, D = 0%, E = 0%, F = 4%.

Significant associations of FD with depression, anxiety, and psychopathological factors have been reported [36, 37].

A systematic review of mostly case–control and cross-sectional studies primarily from Asia (i.e., China, Japan, South Korea, Iran, and Malaysia) included 13 studies involving 14056 subjects. Results showed that the prevalences of depression and anxiety symptoms in patients with refractory FD (63.3% and 61.5%, respectively) were higher than those with nonrefractory FD (20.9% and 23.3%, respectively) and healthy patients (10% and 10%, respectively) [36].

Statement 8. Demographic and dietary factors are potentially associated with FD.

Grade of evidence: Moderate.

Level of agreement: A = 80%, B = 16%, C = 4%, D = 0%, E = 0%, F = 0%.

Demographic factors such as socioeconomic status, smoking, abnormal BMI, and female sex have been reported as risk factors for FD. In particular, recent studies suggest that demographic and dietary factors are interlinked [38–40].

From the PRIBS study data, Arnaout AY et al. reported that in 15 low- and middle-income countries, FD prevalence was nearly four times higher than in high-income countries (37.9% vs. 10%), with associations to smoking, abnormal BMI, and diets high in fat, particularly among urban and wealthier populations [38]. In a Malaysian cross-sectional study, Beh KH et al. noted that being underweight (BMI < 18.5 kg/m^2) was independently associated with FD, suggesting a link to restrictive dietary behaviors and eating disorders, particularly in women [39]. Similarly, a prospective study in Australia reported that patients with FD consumed fewer full meals and reported symptoms such as fullness and bloating soon after eating; these were strongly linked to fat and total energy intake [40].

These observations highlight that both demographic factors (socioeconomic status, smoking, BMI, female sex) and dietary practices (fat-rich meals, restrictive eating) contribute to FD risk.

Statement 9. Postinfection FD is a recognized entity.

Grade of evidence: Moderate.

Level of agreement: A = 68%, B = 32%, C = 0%, D = 0%, E = 0%, F = 0%.

A systematic review and meta-analysis of 19 studies estimated that the mean prevalence of FD in adults after acute gastroenteritis was 9.55% (909/9517). This translated to a 2.54-fold increased risk of FD in individuals more than 6 months after experiencing acute gastroenteritis versus patients in the control group (95% CI, 1.76 to 3.65). Several pathogens were associated with postinfectious FD symptoms, that is, *Salmonella* spp., *Escherichia coli*, *Campylobacter jejuni*, *Giardia lamblia*, and norovirus [41].

Meanwhile, the previously mentioned international cross-sectional study by Arnaout AY et al. reported that patients were more likely to have FD if they tested positive for COVID-19 within the last 12 months (41.1% vs. 35.3%, $p < 0.001$) [38].

Statement 10. FD has a significant negative impact on quality of life and healthcare utilization.

Grade of evidence: Moderate.

Level of agreement: A = 92%, B = 8%, C = 0%, D = 0%, E = 0%, F = 0%.

The negative effects of FD on patients' lives are evident from several clinical studies regarding poor quality-of-life measures and increased use of healthcare services.

In a large prospective study in China, patients with refractory and nonrefractory FD had a significantly worse quality of life than healthy volunteers based on the Short Form 36 Health Survey Questionnaire ($p < 0.05$) [42].

A retrospective study by Chuah KH et al. showed that among 1206 patients with luminal gastrointestinal diseases, 442 (36.7%) had DGBI, with FD being the most common (36.9%), followed by IBS (30.3%). Multivariate analysis showed that only those with FD (36.9%) were significantly associated with high healthcare burden (adjusted OR, 1.996; 95% CI, 1.117 to 3.567; $p = 0.020$) [43].

4.3 | Pathophysiology

Statement 11. Pathogenesis of FD is complex, and multiple mechanisms are involved.

Grade of evidence: Not applicable.

Level of agreement: A = 100%, B = 0%, C = 0%, D = 0%, E = 0%, F = 0%.

Multiple factors are associated with the pathophysiology of FD, including impaired gastric accommodation, delayed gastric emptying, hypersensitivity, social factors, previous gastrointestinal infection, gastric acid secretion, genetic factors, psychological factors, lifestyle (e.g., alcohol consumption and smoking), and stomach morphology [44, 45].

The complex interaction of these factors highlights the importance of a holistic approach to diagnosing and managing FD.

Some of these mechanisms are further explained in the succeeding statements.

The Rome III criteria historically distinguished between the FD subtypes PDS and EPS based on the hypothesis that these have different pathophysiologies.

Specific agents, such as the prokinetic drug acotiamide, may be effective in treating patients with predominantly PDS symptoms. For instance, a multicenter, randomized, placebo-controlled trial showed that in patients with FD, acotiamide 100 mg tid was significantly better than placebo in eliminating

PDS symptoms (i.e., meal-related symptoms of postprandial fullness, upper abdominal bloating, and early satiation) after 4 weeks (elimination rate of 15.3% vs. 9.0%, respectively; $p = 0.004$) [46].

However, for other drugs, there is currently limited evidence to support subtype-based therapy in FD, especially because the EPS and PDS subtypes overlap considerably, from 20% to 60% [16, 17, 47, 48]. Because FD is believed to be a heterogeneous disorder, symptom-based subtypes may be associated with multiple pathophysiologic abnormalities rather than distinct differences in mechanisms. The Rome IV criteria categorizing FD into EPS and PDS subtypes may also not accurately reflect underlying pathophysiology, as studies show similar gastric and duodenal abnormalities across both groups. This lack of distinction may explain why subtype-based treatments have not outperformed empirical therapy, with proton pump inhibitors (PPIs) showing efficacy in both subtypes due to their broader effects beyond acid suppression [45, 49].

Statement 12. Altered gastric accommodation and/or gastroduodenal motility play a role in FD.

Grade of evidence: Moderate.

Level of agreement: A = 72%, B = 24%, C = 4%, D = 0%, E = 0%, F = 0%.

Abnormal gastric motility is a major contributor to FD. It refers to disturbances in the stomach's ability to relax and accommodate a meal (impaired gastric accommodation) or to move food from the stomach into the small intestine at a normal rate (delayed gastric emptying), leading to symptoms such as early satiety and bloating [29, 50–53]. These are particularly evident in patients with PDS and may affect up to 40% of patients with FD. However, their presence and severity may not always correlate with symptoms [29, 50].

Impaired gastric accommodation appears to be a more prominent factor in PDS, affecting up to 40% of patients [51, 52]. However, this impairment may also be present across all FD subtypes [53].

While gastric motility assessments may help clarify FD pathogenesis, tests are not widely available. The panel believes that the role of gastrointestinal function tests is currently limited to classifying FD subtypes, while the usefulness of these tests in improving therapeutic outcomes remains unclear [29].

Statement 13. Visceral hypersensitivity is important in the pathogenesis of FD.

Grade of evidence: Not applicable.

Level of agreement: A = 80%, B = 20%, C = 0%, D = 0%, E = 0%, F = 0%.

Visceral hypersensitivity is another important pathophysiologic mechanism in FD. It is characterized by an increased sensitivity to gastric distention, acid, and fat infusion to the duodenum [54, 55]. This translates to a heightened perception of normal gut stimuli as pain or discomfort [44].

Potential causes of visceral hypersensitivity in FD include chronic inflammation, immune response, and changes in neurochemical signaling within the gut. These factors may contribute to increased neural activity and altered pain perception [56, 57].

Statement 14. Food, gut microbiota, and their interaction may be important in FD.

Grade of evidence: Moderate.

Level of agreement: A = 72%, B = 28%, C = 0%, D = 0%, E = 0%, F = 0%.

The precise mechanisms between food, gut microbiota, and FD remain elusive, but emerging evidence suggests a potential link.

The interaction between food and FD has been demonstrated in several studies [58]. For instance, a cross-sectional study by Adibi P et al. on 2987 adults showed that a diet low in fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAP) increased the risk of FD (OR, 1.85; 95% CI, 1.23 to 2.78; $p = 0.009$) [59]. In contrast, a prospective, single-blind study by Goyal et al. reported that a low-FODMAP diet was associated with significant symptomatic and quality-of-life improvements in those with PDS or bloating compared to those who received traditional dietary advice ($p = 0.04$) [60]. These findings underscore that the role of FODMAP in patients with FD needs to be clarified and treatments should be individualized.

Meanwhile, high carbohydrate, low fat and protein, and increased fruit intake were associated with a lower risk of FD [61, 62].

The role of other food types, as well as timing of food intake and their relationship with FD, has also been described in other studies, underscoring the role that food may have on FD development [63–65].

An association between FD symptoms and dietary factors is particularly important in Asian countries, where research suggests that specific food components may trigger dyspeptic symptoms. These food components include chili, spices, fats, fried foods, pastries, pickles, some vegetables (onions, cabbage), red meat, coffee, alcohol, milk, dairy products, wheat products (pasta, bread), and sweets [66].

For instance, compared to healthy controls, patients with FD have observed compositional differences in gastric and duodenal microbiota [67, 68]. Furthermore, duodenal microbial dysbiosis may contribute to FD by disrupting gut motility, enhancing visceral sensitivity, and compromising mucosal integrity. Changes in gut microbiota may also influence intestinal permeability and microbial diversity, potentially exacerbating FD symptoms [44].

The link between FD and gut microbiota is also supported by the emerging therapeutic potential of modulating gut microbiota. For example, a double-blinded, randomized, placebo-controlled trial demonstrated that 2 weeks of treatment with the gut-specific antibiotic rifaximin adequately relieved global dyspeptic symptoms (78% vs. 52%, $p = 0.02$) in patients with FD [69].

Statement 15. Altered central nervous system function, including psychological comorbidity and sleep disturbance, plays a role in FD.

Grade of evidence: Moderate.

Level of agreement: A = 84%, B = 16%, C = 0%, D = 0%, E = 0%, F = 0%.

In terms of altered central nervous system (CNS) function, several studies have identified key differences in brain activities as well as altered structural and functional connectivity among patients with FD versus healthy subjects, suggesting that pathogenic brain mechanisms are involved in FD development [70–79].

This gut–brain axis involvement in FD is further exemplified by the observed increased risks for anxiety, depression, and sleep disorders in these patients versus healthy subjects [80–82]. These interactions are further supported by studies demonstrating that modulation of the gut–brain axis is a valid therapeutic target for DGBI [80].

Meanwhile, a study in China evaluated sleep quality and mood symptoms in 115 adult patients with typical FD, compared with 61 healthy volunteers. Results showed significantly higher Pittsburgh Sleep Quality Index scores in patients with FD than in the control group (10.49 ± 3.38 vs. 6.77 ± 2.77, respectively; $p < 0.01$), indicating poorer overall sleep quality. Importantly, patients with FD had higher scores in subjective sleep quality, sleep latency, sleep duration (i.e., fewer hours of sleep), sleep disturbance, use of sleeping medication, and daytime dysfunction than those in the control group [37].

Similarly, a study involving 8923 university students from Japan investigated the association between sleep disturbance and FD. Results showed a 1.9% prevalence of FD, with all types of sleep disturbances being independently positively associated with FD (total sleep disturbance: OR, 4.11; 95% CI, 2.89 to 5.78) [83].

These findings support the potential role of CNS and psychological disturbances in FD pathogenesis.

Statement 16. Acid exposure may play a role in FD.

Grade of evidence: Moderate.

Level of agreement: A = 48%, B = 32%, C = 16%, D = 0%, E = 0%, F = 4%.

Gastric acid stimulation appears to trigger dyspeptic symptoms in both healthy individuals and FD patients, with a more pronounced effect, that is, gastric and duodenal hypersensitivity, in FD patients [29]. Antisecretory medications may also benefit some patients, regardless of EPS or PDS subtype, suggesting a potential role for acid sensitivity in symptom generation [50].

Duodenal acidification may also indirectly contribute to FD symptoms. Evidence suggests that it can promote proximal gastric relaxation, heighten sensitivity to distension, and hinder gastric accommodation during and immediately after a meal [56, 84].

Statement 17. Pathogenic gastrointestinal infection and COVID-19 may cause postinfection FD.

Grade of evidence: Moderate.

Level of agreement: A = 40%, B = 44%, C = 12%, D = 4%, E = 0%, F = 0%.

A postinfectious origin has been previously suggested for other functional bowel disorders, for example, IBS after an acute intestinal infection and gastroparesis syndrome following a viral infection. Similarly, a study by Tack J et al. involving 400 consecutive dyspeptic patients showed that a subset of these patients has a history suggestive of postinfectious dyspepsia, with a high prevalence of impaired accommodation that may be due to a dysfunction at the level of gastric nitrergic neurons [85].

Previously, the development of clinical symptoms in patients with postinfectious FD has also been associated with the migration of inflammatory cells in the duodenal mucosa [86].

Because the current consensus report was prepared during the post–COVID-19 period, several reports on the association of COVID-19 with DGBI have been published, specifically FD and IBS, reporting deterioration in gastrointestinal symptoms [87]. Emerging evidence also suggests a possible link between COVID-19 and the development of FD [88–90]. Further research is needed to confirm this association.

Statement 18. Familial and genetic predisposition may play a role in FD.

Grade of evidence: Moderate.

Level of agreement: A = 48%, B = 32%, C = 16%, D = 4%, E = 0%, F = 0%.

Family history and genetic factors are thought to contribute to the development of FD. Candidate genes include G-protein beta3 subunit (GNB3), cyclooxygenase-1 (COX-1), transient receptor potential vanilloid 1 (TRPV1), serotonin transporter (SLC6A4), cholecystokinin-1 receptor (CCK-1R), interleukin family members, and the ghrelin gene [91–94].

The serotonin transporter gene (5-HTTLPR) polymorphism may be associated with EPS but not PDS [95]. Meanwhile, single nucleotide polymorphisms in transforming growth factor beta 1 (TGFB1) showed significant associations in patients with PDS as the predominant subtype [96].

Further research is needed to definitively establish the role of specific genetic factors in FD development and progression. This includes investigating the functional significance of identified polymorphisms and potential interactions with environmental factors.

Statement 19. Duodenal low-grade inflammation may play a role in FD.

Grade of evidence: Moderate.

Level of agreement: A = 28%, B = 60%, C = 8%, D = 4%, E = 0%, F = 0%.

Emerging evidence suggests the potential role of mild or low-grade duodenal inflammation in patients with FD. While the exact mechanisms remain under investigation, both infectious and noninfectious inflammatory processes may be involved. The idea that duodenal low-grade inflammation may play a role in FD also aligns with other FD treatment guidelines [29, 44].

The neuropeptide glial cell line–derived neurotrophic factor (GDNF) may provide a link between duodenal inflammation and FD symptoms [97]. Increased expression of GDNF in the duodenum has been observed in FD patients, potentially influencing disease processes and symptom perception.

Psychological factors such as anxiety and depression may exacerbate FD through inflammatory pathways [81]. These conditions have been associated with increased mast cell activity in the gut, which can contribute to inflammation.

4.4 | Management

Statement 20. A holistic multidimensional approach with good doctor–patient communication is recommended for patients with FD.

Grade of evidence: Not applicable.

Level of agreement: A = 64%, B = 28%, C = 8%, D = 0%, E = 0%, F = 0%.

Because of the complex and multifaceted nature of FD, the task force members agree that a holistic approach that addresses various contributing factors is recommended [98]. This approach acknowledges the interplay of physiological, psychological, and lifestyle factors, allowing tailored treatment plans that address individual needs. This will also help address both physiological and psychological components of the disease, tailored to individual patient profiles.

While the evidence is still developing, nonpharmacological approaches such as stress management and dietary modifications may also offer benefits with minimal side effects [99].

Strong doctor–patient communication is crucial as it fosters trust, empowers patients in decision-making, and promotes adherence to treatment strategies. This ultimately leads to better symptom management and improved quality of life [32].

Statement 21. Recognizing food triggers is important in patients with FD, as dietary modifications in these patients may provide symptom relief.

Grade of evidence: High.

Level of agreement: A = 68%, B = 28%, C = 4%, D = 0%, E = 0%, F = 0%.

Fatty foods are consistently associated with symptom exacerbation in FD patients. Compared to high-carbohydrate meals, high-fat meals induce more symptoms, such as nausea, pain, and discomfort [58, 100]. Patients with FD have also shown a negative preference for fatty foods, independent of symptom severity [101].

Carbohydrates and proteins have also been implicated in FD symptom induction. Higher protein intake was associated with a higher prevalence of epigastric pain. In comparison, higher carbohydrate and lower fat or protein intake were associated with a lower likelihood of uninvestigated dyspepsia [61].

Capsaicin, the active component in chili peppers, has complex effects on FD. Acute ingestion can exacerbate symptoms, but chronic consumption may improve overall symptom scores, epigastric pain, fullness, and nausea in FD patients [58].

A cross-sectional study by Tabibian SR et al. involving 3362 participants reported that higher fruit intake is associated with a 32% lower risk of FD and lower risks of early satiation and postprandial fullness than the lowest intake. Meanwhile, a higher intake of vegetables was associated with a lower risk of FD, but only in men [62].

Exclusion diets and dietary modifications may be considered in FD management. Studies have shown that reducing the consumption of trigger foods, abandoning unhealthy cooking methods such as roasting, reducing smoking, and limiting alcohol and fast food intake might be beneficial for relieving symptoms [102]. Higher fruit intake has been associated with lower odds of early satiation and postprandial fullness [62]. Dietary advice for FD should be individualized according to patient-specific factors [58, 103, 104].

Statement 22. Patients with dyspepsia with proven *H. pylori* infection should undergo eradication therapy. However, symptom benefit varies between different populations.

Grade of evidence: Moderate.

Level of agreement: A = 80%, B = 16%, C = 4%, D = 0%, E = 0%, F = 0%.

Eradication therapy is a well-established treatment for patients with dyspepsia and confirmed *H. pylori* infection, and it has been demonstrated to result in long-term improvement in symptoms [17, 105, 106].

However, the degree of symptom benefit varies. While some studies have demonstrated different degrees of symptom improvement following eradication, others observed no significant difference compared to placebo [106, 107].

Nevertheless, regardless of symptoms or complications, *H. pylori* is an important human pathogen that causes chronic and progressive gastric mucosal damage. Unless there are compelling reasons to say otherwise, the current consensus generally recommends offering eradication treatment to all patients with *H. pylori* infection to minimize transmission and to mitigate possible complications and associated treatment costs [108, 109].

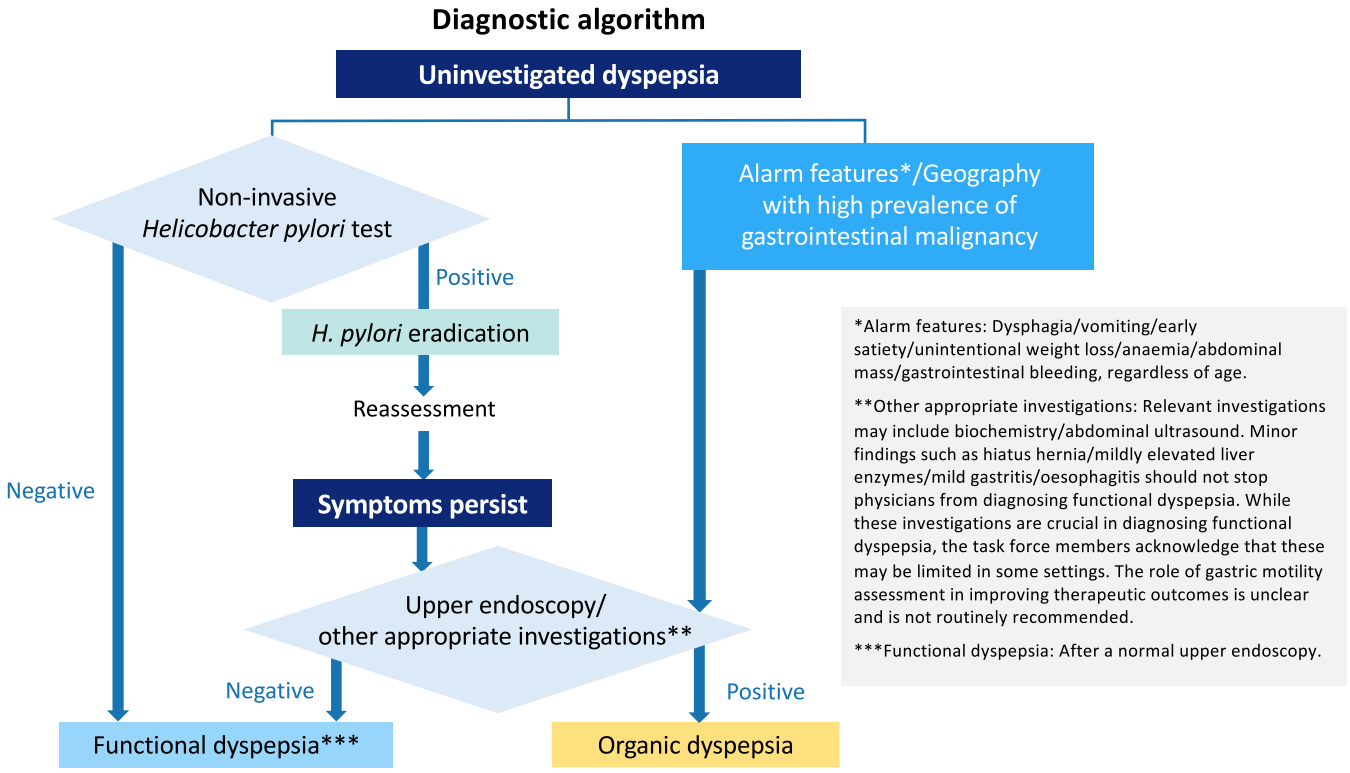

FIGURE 1 | Diagnostic algorithm for functional dyspepsia in Asian primary care settings.

Eradication strategies may benefit patients with FD who are *H. pylori*–positive through several mechanisms. For instance, *H. pylori* eradication has been demonstrated to reduce eosinophil counts in the stomach, potentially contributing to symptom relief [110, 111]. *H. pylori* eradication may also indirectly improve FD symptoms by reducing competition for space with beneficial gut bacteria [112].

Treatment duration of eradication therapies should be considered before efficacy is assessed. In a systematic review of 25 randomized controlled trials with a total of 5555 patients with FD, *H. pylori* eradication therapy demonstrated symptom improvement during long-term follow-up after 1 year or more (RR, 1.24; 95% CI, 1.12 to 1.37; $p < 0.0001$) but not during short-term follow-up at < 1 year (RR, 1.26; 95% CI, 0.83 to 1.92; $p = 0.27$) [106].

Clinicians should be mindful of patient variability in treatment outcomes, particularly across different regions and population subtypes [113].

Based on these observations, the task force members reached a consensus and developed a diagnostic algorithm for FD in the Asian setting (see Figure 1).

Statement 23. Proton pump inhibitors may be considered for the management of FD.

Grade of evidence: Moderate.

Level of agreement: A = 68%, B = 24%, C = 8%, D = 0%, E = 0%, F = 0%.

In general, current guidelines recognize the role of PPIs in FD management and may be considered as a first-line pharmacologic agent in appropriate patients [17, 27, 29, 114, 115]. While a consensus was reached regarding Statement 23, a third of the task force members (32%) had at least some reservations on the role of PPIs in FD because the evidence is less robust when contextualized to the Asian population.

PPIs are more effective than placebo for patients with FD, but overall symptom improvement may be minimal [114]. In particular, evidence in the Asian context is less robust. For instance, a Cochrane subgroup meta-analysis of four studies from Eastern countries that included 959 participants failed to achieve significant symptom benefits among those who received PPIs versus placebo [116]. A subsequent network meta-analysis of 71 randomized trials, including 19 243 adult participants, examined the efficacy of various drugs for FD. Results showed that in a subgroup analysis including only trials from the East, only H2-receptor antagonists (H2-RAs), and not PPIs, were associated with the resolution of dyspepsia symptoms [48].

Similarly, a single-blinded randomized controlled trial investigated the efficacy and safety of treating 180 patients according to FD subtypes versus empirical PPI therapy. After 8 weeks, similar percentages of patients in both groups achieved the primary efficacy outcome of global symptom improvement (74.4% and 72.2%, respectively; $p = 0.74$). These results show that initial treatment of FD with empirical PPI alone was not more effective than treating according to FD subtype for up to 8 weeks [49].

Randomized controlled trials have shown that chronic PPI use increases the risk of enteric infections [117, 118]. Prolonged use of PPIs has also been postulated to increase the risk of other adverse events, such as chronic hypochlorhydria-associated complications, including anemia, hypergastrinemia, and osteoporotic fractures due to associated impaired calcium absorption [119]. However, some of these risks and other postulated adverse events in the literature, including but not limited to dementia, renal and cardiac diseases, have not yet been supported by high-quality studies and are susceptible to confounding bias [119–121].

Based on the above, the task force suggests that PPIs may be continued in patients with FD who have symptom benefit but should be discontinued if there is a lack of response after 12 weeks.

Statement 24. H2-receptor antagonists may be considered for the management of FD.

Grade of evidence: High.

Level of agreement: A = 56%, B = 32%, C = 12%, D = 0%, E = 0%, F = 0%.

In an aforementioned network meta-analysis that examined the efficacy of various drugs for FD, results suggest that H2-RAs were among the most effective drug classes versus placebo for symptom improvement and resolution in patients with FD [48]. In particular, H2-RAs may be more effective in Asian populations than Western populations [48, 122].

The pathophysiology of FD symptom development has previously been described as multifactorial and may not be primarily gastric acid–related. Because patients from Asian countries may have lower acid secretion than those from Europe and the United States, acid inhibition by H2-RAs may be sufficient to improve symptoms in patients with FD [122, 123]. H2-RAs have been described as having anti-inflammatory properties; however, unlike PPIs, H2-RAs are not associated with an increased risk of enteral dysbiosis [124].

Statement 25. There is insufficient evidence to recommend potassium-competitive acid blocker therapy for the treatment of FD.

Grade of evidence: Low.

Level of agreement: A = 68%, B = 28%, C = 4%, D = 0%, E = 0%, F = 0%.

Potassium-competitive acid blockers (PCABs) are a novel class of acid-suppressive drugs that have shown promise in FD treatment. Recent studies have demonstrated that PCABs, such as vonoprazan and tegoprazan, may effectively improve symptoms in patients with FD, providing satisfactory symptom relief rates comparable to or better than traditional treatments [125–127].

Available studies report varying results, with some suggesting potential benefits and others finding no significant difference compared to other treatments [125–127]. More robust studies are required to assess the long-term efficacy and safety of PCABs in FD patients, particularly in comparison to established treatment options.

While some studies have shown improvement in FD symptoms following PCAB therapy (e.g., vonoprazan), the quality of evidence remains low [125]. The task force members acknowledge that the evidence is currently insufficient to recommend its definitive use for FD treatment.

Statement 26. Prokinetics are effective in some FD patients.

Grade of evidence: High.

Level of agreement: A = 68%, B = 20%, C = 12%, D = 0%, E = 0%, F = 0%.

Current evidence suggests that prokinetics may effectively reduce overall symptoms of FD regardless of its subtype [128–131].

A systematic review and meta-analysis including 38 studies showed that prokinetics were associated with a statistically significant improvement in the global symptoms of patients with FD, with a number needed to treat of 7 [128]. In particular, patients from Eastern countries appeared to respond better to prokinetics, with a lower number needed to treat than Western populations (5 vs. 10 in Western populations), likely reflecting differences in genetics, diet, culture, or physiology.

The meta-analysis also noted that in two studies, prokinetics benefited patients with FD who had the EPS subtype. At the same time, 14 studies on patients with mixed PDS/EPS subtypes also demonstrated prokinetic efficacy. However, there were no statistically significant subgroup differences [128].

A systematic review and meta-analysis of 43 studies similarly found no significant differences in prokinetic treatment outcomes across FD subtypes (PDS, EPS, or mixed) [129].

Both meta-analyses noted substantial heterogeneity and low-quality evidence in the studies included and noted that high-quality randomized controlled trials are needed to fully elucidate the efficacy of prokinetics in FD [128, 129].

Although prokinetics may improve symptoms in patients with FD, current data suggest that complete symptom resolution is often not achieved [48].

A meta-analysis of 28 randomized clinical trials involving 5790 participants aimed to explore the efficacy and safety of prokinetics for FD. Results showed that different prokinetic medications have varying efficacy profiles compared to placebo. Results also showed that metoclopramide and cinitapride may be more effective than other options, but data quality limitations require further investigation [132].

Meanwhile, itopride shows promise in patients with FD [48, 133]. For instance, a phase 2b trial showed significantly improved symptoms in patients with FD treated with itopride at 50, 100, or 200 mg tid versus placebo ($p < 0.05$ for all

comparisons between itopride and placebo) [134]. However, this benefit versus placebo was not demonstrated in succeeding phase 3 trials [135].

Domperidone, tegaserod, and mosapride demonstrated nonsignificant benefits in other clinical studies [48, 136].

Reported drug-related adverse events between acotiamide, cinitapride, domperidone, and placebo were similar [132]. Previously, safety concerns associated with an earlier prokinetic agent, cisapride, have led to its withdrawal from the market [137]. Notably, metoclopramide has been associated with extrapyramidal symptoms, gynecomastia, galactorrhoea, and menstrual irregularities, limiting its use [138].

Statement 27. Fundic relaxant drugs (e.g., acotiamide) may be effective in improving FD, especially PDS.

Grade of evidence: Moderate.

Level of agreement: A = 52%, B = 36%, C = 12%, D = 0%, E = 0%, F = 0%.

Fundic relaxants aim to improve relaxation of the upper stomach in FD patients, particularly those experiencing postprandial fullness [46, 139].

A systematic review and meta-analysis of six publications that included seven randomized controlled trials (one publication reported two trials) investigated the efficacy of acotiamide in the treatment of patients with FD. Results showed that acotiamide (50–300 mg tid) was significantly more effective in achieving overall FD symptom improvement versus placebo (RR, 1.29; 95% CI 1.19 to 1.40, $p < 0.00001$) [140]. Similar results were observed in favor of acotiamide versus placebo (RR, 0.89; 95% CI, 0.79 to 0.99) in a previously mentioned systematic review and network meta-analysis on the efficacy of different drugs for FD [48].

Aside from symptom relief, acotiamide has been associated with improved quality of life, work productivity, and long-term safety in patients with FD [141–143].

Among the serotonin 1A (5-HT1A) receptor agonists, tandospirone demonstrated better results than buspirone in providing symptom relief for patients with FD [48, 144, 145].

It should be noted that studies on fundic relaxants have mainly focused on patients with predominantly PDS or mixed PDS features of FD, while also highlighting substantial overlap between the two subtypes [48]. Further research is needed to explore the role of other fundic relaxants in FD treatment across different patient populations.

Statement 28. Neuromodulators may be considered in the management of FD not responding to initial treatment.

Grade of evidence: Moderate.

Level of agreement: A = 56%, B = 40%, C = 4%, D = 0%, E = 0%, F = 0%.

Neuromodulators, including antidepressants and antipsychotics, may be an option for FD patients who experience limited improvement with first-line treatments like PPIs and prokinetics [16]. However, patients should be informed of the possible side effects, including dizziness, drowsiness, constipation, and dry mouth [17, 146].

Neuromodulators are still classified as off-label treatments for FD in some countries. Patients must be counseled appropriately before they are prescribed these drugs.

Among the neuromodulators, evidence suggests that tricyclic antidepressants (TCAs), antipsychotics (sulpiride or levosulpiride), and tandospirone may be beneficial for patients with FD [48, 144, 147]. On the other hand, current evidence does not consistently support the efficacy of selective serotonin reuptake inhibitors or serotonin-norepinephrine reuptake inhibitors for FD, because results are mixed and limited by small or heterogeneous trials [17, 48].

A systematic review and network meta-analysis of 10 trials involving 970 participants evaluated the efficacy of 10 different types of neuromodulators versus placebo. Results showed that flupentixol + melitracen (OR, 10.00; 95% CI, 1.59 to 62.73), tandospirone (3.24; 1.38 to 7.60), imipramine (2.21; 1.02 to 4.79), and amitriptyline (1.71; 1.06 to 3.09) were significantly superior to placebo in terms of improving global FD symptom scores [144].

Meanwhile, the TCA nortriptyline has shown conflicting results in FD management based on Asian populations. For instance, a prospective study in Thailand noted that patients with FD who previously failed to respond to PPI and prokinetic treatment reported that nortriptyline 10 mg qd was not superior to placebo [148]. This contrasts with a randomized clinical study in South Korea, which noted that the prokinetic drug, mosapride (controlled-release formulation, 15 mg qd), and nortriptyline 10 mg qd had similar efficacy in patients with FD. In this study, the rate of overall dyspepsia improvement was comparable between groups (53.7% vs. 54.0%, respectively; $p = 0.976$). Results were similar in a subgroup analysis by FD subtypes [149].

Pregabalin may be effective in patients with FD, although further research is required to establish a definitive role in its treatment. Similarly, additional research is needed for mirtazapine, but it is considered a possible treatment for patients with FD and early satiety and weight loss [17].

While the exact mechanism of antidepressants in improving FD symptoms remains unclear, possible explanations may involve stress reduction, improved gut–brain communication, and pain perception modulation [150].

Table 2 lists the different classes of pharmacological agents available for FD management.

Statement 29. Psychotherapy should be considered to improve the symptoms of FD in patients not responsive to initial treatment.

Grade of evidence: High.

TABLE 2 | Different classes of pharmacotherapy agents for the management of FD.

Drug	Mechanism of action	Adverse reactions
Acid suppression therapy		
Pantoprazole [116]	Proton pump inhibitor	**RCT proven**: Enteric infections **Other reported adverse consequences:** Bone fractures, acute interstitial nephritis, chronic kidney disease, deficiencies of vitamins and minerals (vitamin B_{12}, magnesium, potassium, sodium, calcium), fundic gland polyps, heart attacks, strokes and dementia **Side effects**: Headache, abdominal pain, nausea, vomiting, diarrhea, constipation, and flatulence
Omeprazole [116, 151]		
Lansoprazole [116, 152]		
Esomeprazole [116]		
Rabeprazole [116, 119]		
Ranitidine [153–155]	H2-receptor antagonist	Headache, drowsiness, fatigue, abdominal pain, constipation, or diarrhea CNS effects including agitation, confusion and delirium Tachyphylaxis may occur within 7–14 days of continued treatment and limit effective therapy. Prolonged use may result in sequelae of androgen receptor antagonism (e.g., gynecomastia)
Cimetidine [155]		
Famotidine [156, 157]		
Prokinetics		
Domperidone [138]	D2 receptor antagonist	Gynaecomastia, galactorrhoea, arrhythmia
Itopride [138]	D2 receptor antagonist, acetylcholinesterase inhibition	Rash, giddiness, diarrhea
Mosapride [138, 149]	5-HT_4 receptor agonist	Headache, dizziness, sleepiness, gastrointestinal dysfunction, pruritus, sweating
Fundic relaxants		
Acotiamide [138]	M1/M2 muscarinic antagonist	Headache, diarrhea
Tandospirone [17, 48, 144]	5-HT1 receptor agonist (also is an anxiolytic)	CNS effects, drug-induced movement disorder, serotonin syndrome
Buspirone [17, 48, 145]	5-HT1 receptor agonist (also is an anxiolytic)	
Neuromodulators		
Levosulpiride [17, 48, 138, 147]	D2 receptor antagonist, 5-HT4 agonist, weak 5-HT3 antagonist	Menstrual abnormalities, extrapyramidal side effects, drug-induced parkinsonism
Sulpiride [17, 48, 147]	D2/D3/5-HT1A antagonist	Extrapyramidal side effects, anticholinergic effects
Mirtazapine [17, 115, 158, 159]	Tetracyclic antidepressant	Dizziness, fatigue, drowsiness/sleepiness, pruritus
Amitriptyline [17, 48, 144, 147]	Tricyclic acid	Anticholinergic effects, arrhythmia, CNS depression, hyponatremia, glaucoma, serotonin syndrome
Imipramine [48, 144, 147]	Tricyclic acid	
Nortriptyline [48, 144, 147]	Tricyclic acid	

Abbreviations: 5-HT, 5-hydroxytryptamine or serotonin; CNS, central nervous system; D2/D3, dopamine 1/2 receptor; FD, functional dyspepsia; RCT, randomized controlled trial.

Level of agreement: A = 40%, B = 44%, C = 16%, D = 0%, E = 0%, F = 0%.

People with FD frequently report higher anxiety, depression, and stress compared to the general population [29]. In a systematic review and meta-analysis of 14 randomized clinical trials with a total of 1434 patients with FD, subgroup analysis showed that psychotherapy was associated with symptom improvement, relief of gastrointestinal symptoms, and decreased depression levels compared to the control [160].

Similar results have also been demonstrated in other studies, showing that psychotherapy may be an option for patients with FD who do not respond well to initial treatments [160, 161].

Different psychotherapeutic approaches, including cognitive behavioral therapy, stress management, mindfulness, and hypnotherapy, are being explored for managing FD [162–165].

While the efficacy of psychotherapy in FD may be encouraging, its applicability, especially in the Asian setting, faces challenges

because of cultural differences, limited resources, and a preference for traditional interventions in some regions [161, 164, 166].

Statement 30. Emerging evidence shows probiotics may have a role in FD; however, more research is required.

Grade of evidence: Low.

Level of agreement: A = 68%, B = 24%, C = 8%, D = 0%, E = 0%, F = 0%.

Previous meta-analyses on probiotics and FD reported inconclusive results [167, 168]. However, recent studies suggest that certain probiotic strains, particularly *Bifidobacterium animalis* subsp. *lactis* BL-99, *Bacillus coagulans* MY01, and *Bacillus subtilis* MY02, may effectively improve FD symptoms [169, 170].

The effectiveness of probiotics in patients with FD may also be strain-dependent, highlighting the need for research on specific formulations [169, 170].

Probiotics are hypothesized to have several functions in the gut. They may play a role in FD treatment by regulating intestinal flora and producing short-chain fatty acids [171]. However, because preliminary findings are inconsistent, more large-scale studies are needed to definitively establish their efficacy and safety in FD management.

Statement 31. Pharmacological traditional, complementary, and integrative medicine may be used for the treatment of FD. These should be selected based on evidence, availability, and health system context. Consider multidisciplinary collaboration with a traditional, complementary, and integrative medicine practitioner. Further research in this area is encouraged.

Grade of evidence: Moderate.

Level of agreement: A = 56%, B = 32%, C = 8%, D = 0%, E = 4%, F = 0%.

TCIM pharmacotherapies are increasingly recognized for their efficacy in treating FD, and select formulations are currently incorporated into conventional medicine [172, 173]. Iberogast (STW5), an herbal formulation associated with FD symptom improvement, has been recognized for its fundic relaxant properties in patients with FD [19, 174].

In Asia, TCIM has been incorporated into the management of FD in China, Japan, and Korea. In Japan, the herbal medicine Rikkunshito has been incorporated as a first-line treatment for patients with FD [29, 175]. Meanwhile, Zhizhu Kuanzhong (ZZKZ), a traditional Chinese medicine, and Motilitone, a prokinetic agent derived from the plant extracts of Pharbitidis semen and Corydalis tuber, have demonstrated efficacy in clinical trials and have been included in FD treatment guidelines in China and Korea, respectively [138, 176].

A meta-analysis of 23 randomized clinical trials, including 2496 patients, investigated the efficacy of ZZKZ capsules for FD. Results showed ZZKZ alone or combined with Western medicine was associated with a significantly better clinical effect rate than Western medicine alone in FD treatment (OR, 3.32; 95% CI, 2.66 to 4.15; $p < 0.00001$) [177]. A more recent randomized controlled study by Wang QQ et al. also showed that ZZKZ was superior to the TCA doxepin in alleviating stigma and medication nonadherence ($p < 0.001$), with comparable efficacy in improving dyspeptic symptoms and psychological conditions in refractory FD patients [178].

Similarly, a network meta-analysis of 28 randomized controlled trials conducted by HO L et al. identified 26 unique Chinese herbal medicines and evaluated their efficacy for FD versus prokinetics. Results showed that a modified Zhi Zhu decoction had a moderate beneficial effect on alleviating global symptoms after 4 weeks of treatment (risk difference, 0.28; 95% CI, −0.03 to 0.75) and may be considered as an alternative for patients with FD unresponsive to prokinetics [179].

Meanwhile, an ongoing randomized multicenter trial aims to provide further evidence on the clinical efficacy and safety of ZZKZ versus placebo in treating patients with FD–PDS [180].

A double-blind, randomized, noninferiority, multicenter trial including 389 patients with FD demonstrated that Motilitone was comparable with pantoprazole in improving global and individual symptoms, as well as dyspepsia-specific quality of life, in patients with FD, with no additive benefits when combined with pantoprazole [181].

With the emergence of other TCIMs, careful selection based on available research and regulatory status within specific healthcare systems will be crucial [182]. Collaboration between conventional and TCIM practitioners may help optimize treatment selection and outcomes for patients with FD [183].

Statement 32. Nonpharmacological traditional, complementary, and integrative medicine may be an effective therapy for FD. These should be selected based on evidence, availability, and health system context.

Grade of evidence: Moderate.

Level of agreement: A = 72%, B = 28%, C = 0%, D = 0%, E = 0%, F = 0%.

Nonpharmacological TCIM, such as acupuncture, moxibustion, and potentially transcutaneous auricular vagus nerve stimulation (taVNS), may also offer alternative management strategies for patients with FD.

Evidence suggests that acupuncture, specifically manual, electroacupuncture, and moxibustion, may benefit patients with FD. Several studies have demonstrated greater improvement than sham acupuncture or prokinetic medication [184–187]. In particular, a meta-analysis of seven randomized clinical trials by Mai X et al. that included 853 patients showed that electroacupuncture may be more effective than sham acupuncture in reducing symptom scores [188].

A systematic review of 14 randomized controlled trials by Dai N et al. that included 1128 patients with FD assessed the potential effect of therapeutic massage/Tuina on FD symptoms. Results

showed that Tuina plus conventional therapy also significantly improved overall symptoms (RR, 1.14; 95% CI, 1.06 to 1.23), quality of life (mean difference scores, 10.44; 95% CI, 7.65 to 13.23), and epigastric pain (mean difference scores, −0.76; 95% CI, −1.11 to −0.41). No safety or cost-effectiveness issues were reported [189].

Initial research showed that taVNS improved clinical response rates compared to placebo in patients with FD, suggesting that it may be a promising option for this disorder [190–192].

While yoga may address various aspects of health that may be relevant to FD, more research is needed to determine its effectiveness in this population [193, 194].

4.5 | Approach to the Management of FD in the Asian Setting

A holistic, multidimensional strategy should be sought for managing FD. FD management aims to mitigate symptoms and improve quality of life. Reassurance that underlying pathologies have been excluded relieves patients' anxiety over sinister causes and further empowers patients to overcome their symptoms [17, 27, 32].

FD subtyping based on the Rome criteria appears to have limited value in guiding therapeutic strategy [45]. Similar to other current guidelines on dyspepsia, the task force members do not recommend using a symptom- or subtype-based approach in FD management decisions, because overlaps exist and multiple mechanisms and pathological processes are likely to be involved [17, 27].

Initial management for all patients should include lifestyle, dietary modification, and *H. pylori* eradication. These may sufficiently relieve symptoms in some patients to avoid prolonged pharmacotherapy [49].

The management algorithm in Figure 2 structures initial pharmacotherapy as an equally weighted three-part wheel, where the initial agent selection depends on local evidence and availability. Unlike the current guideline, the previous one focused on FD subtyping to guide initial therapy. Based on recent evidence, the task force members have recommended treating FD regardless of subtypes [17, 27, 49]. Treatments include PPIs/H2-RAs, prokinetics, and fundic relaxants.

Switching to another class of drugs within these three can be considered if symptoms persist despite 8–12 weeks of therapy. In patients who failed to respond to initial therapy, subsequent treatment—including neuromodulators and/or psychotherapy—should be considered. Although there are no high-quality studies specifically addressing the timing of psychiatric referral, the task force members agree that simultaneous earlier referral to psychiatrists should be considered if the initial consult identifies significant psychological symptoms. This would allow concomitant treatment of the underlying psychiatric conditions rather than following a step-wise escalation of care and referring only when multiple lines of FD therapy have failed.

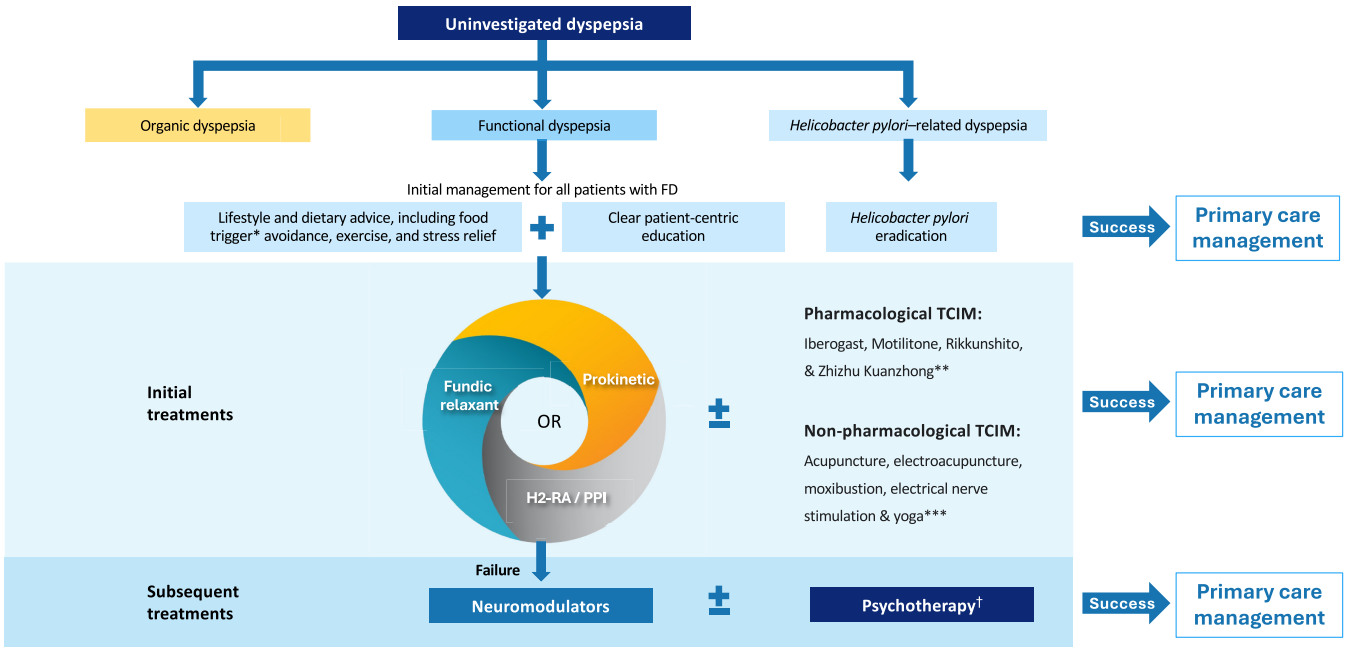

FIGURE 2 | Management of FD in the Asian setting. *Foods that may generate or exacerbate dyspeptic symptoms include spice/chili, fat/oil intake, dairy products, especially in the Asian context, and coffee and alcohol. **These pharmacological TCIM options are supported by evidence. ***These nonpharmacological TCIM options have shown benefit in patients with FD, although more research is required. †Gut–brain behavioral interventions that have received focus in FD include psychodynamic therapy, cognitive behavioral therapy, stress management and mindfulness, and hypnotherapy. FD, functional dyspepsia; H2-RA, histamine receptor antagonist; PPI, proton pump inhibitor; TCIM, traditional, complementary, and integrative medicine.

TCIM may be used alone or as an adjunct to conventional treatment at any point in the therapeutic journey. Its use should be based on the health system's context, preferably in collaboration with TCIM practitioners if integrative medicine services are available. The following management algorithm consolidates the above recommendations and consensus statements (see Figure 2).

5 | Conclusions

This updated report builds upon the 2012 consensus, incorporating significant advancements in FD research and management. It incorporates the latest evidence-based approaches, including the growing body of research on nonpharmacological therapies and the evolving role of specific medications. Recognizing the dynamic nature of scientific understanding of FD, this report serves as a foundation for continuous improvement and future revisions as new knowledge emerges. We encourage ongoing research efforts focused on Asian populations to further refine diagnosis and treatment strategies and ultimately improve patient outcomes.

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Conflicts of Interest

Sanjiv Mahadeva has received support for meeting attendance from Dong-A ST, Korea. Kewin Siah is the secretary general for ANMA, co-chair of APAGE ELC DGBI's Special Interest Group, and the president of IBS Support Group Singapore. Uday Goshal has been an advisor for Bioquest Solutions and Readystock and received a grant from Zydus Pharmaceuticals; he holds a scientific patent for a product that is yet to be marketed. Hidekazu Suzuki has received grants or contracts from Tosoh and Biofermin Pharmaceutical and has received honoraria from Takeda Pharmaceutical, Astellas Pharma, Tsumura, Otsuka Pharmaceutical, and Lonch. Kee Huat Chuah has received grants and honoraria from Abbott Laboratories. Ratha-korn Vilaichone has received honoraria from Takeda Pharmaceutical, Meiji, Farmaline, and Kespa. Kentaro Sugano has received funding from Abbott Laboratories for medical writing support of the present manuscript and has received consulting fees from FUJIFILM Corporation, Zeria Pharmaceutical, and Biofermin Pharmaceutical, as well as honoraria from Takeda Pharmaceutical. Sanjiv Mahadeva and Tiing Leong Ang are Editorial Board members of JGH and co-authors of this article. To minimize bias, they were excluded from all editorial decision-making related to the acceptance of this article for publication. The other authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this consensus report are available in the public domain, as cited in the References section.

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