IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429
Volume 6, Issue 6, November 2026
Impact Factor: 8.2

Pharmacological Management of Bronchial Asthma: A Review of β2-Agonists, Inhaled Corticosteroids and Leukotriene Receptor Antagonists

Dr. Bhagvan M. Kale[1], Drx. Sameer Shaikh[2], Ajinath K. Patil[3], Shubham B. Tarte[4], Prashant B. Landge[5]

Principal, Shri Goraksha College of Pharmacy and Research Centre, Khamgaon, Maharashtra, India[1]
Assistant Professor, Shri Goraksha College of Pharmacy and Research Centre, Khamgaon, Maharashtra, India[2]
Students, Shri Goraksha College of Pharmacy and Research Centre, Khamgaon, Maharashtra, India[3-5]

Abstract : *Bronchial asthma is a chronic inflammatory disorder of the airways characterised by reversible airflow obstruction, bronchial hyperresponsiveness, and recurrent episodes of wheezing, breathlessness, chest tightness, and cough. Its management rests on two pharmacological pillars: rapid symptom relief and long-term control of the underlying inflammatory process. This review examines three major drug classes used in asthma therapy – β2-adrenergic agonists, inhaled corticosteroids, and leukotriene receptor antagonists – with specific reference to salbutamol, formoterol, budesonide, and montelukast. Their mechanisms of action, pharmacokinetic characteristics, therapeutic roles, and adverse effect profiles are discussed in the context of current stepwise asthma management, with the aim of consolidating core pharmacological concepts relevant to undergraduate pharmacy education.*

Keywords: Pharmacological Management of Bronchial Asthma: A Review of β2-Agonists, Inhaled Corticosteroids and Leukotriene Receptor Antagonists

I. INTRODUCTION

Asthma affects an estimated 260-300 million people worldwide and remains one of the most common chronic respiratory conditions encountered in clinical pharmacy practice. The disease is driven by a combination of airway inflammation, smooth muscle hyperreactivity, and mucus hypersecretion, all of which contribute to variable and often reversible airway narrowing. Because the pathology has both an acute bronchospastic component and a chronic inflammatory component, effective pharmacotherapy typically combines a "reliever" that acts quickly on smooth muscle tone with a "controller" that suppresses the inflammatory cascade over time. Understanding how these two therapeutic strategies complement one another is fundamental to rational prescribing and patient counselling in asthma care.

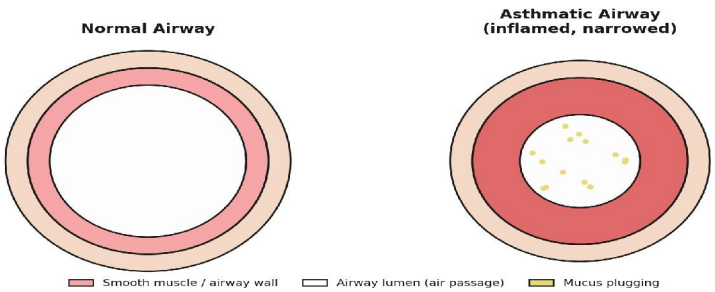
II. PATHOPHYSIOLOGICAL BASIS FOR DRUG THERAPY

Allergen or irritant exposure triggers mast cell degranulation and the recruitment of eosinophils, T-helper type 2 lymphocytes, and other inflammatory cells into the airway wall. Mediators released during this response – histamine, cysteinyl leukotrienes, prostaglandins, and numerous cytokines – provoke bronchial smooth muscle contraction, mucosal oedema, and increased mucus production. Repeated inflammatory insults can lead to airway remodelling, with thickening of the basement membrane and hypertrophy of smooth muscle, which underlies the progressive and sometimes irreversible component of the disease seen in longstanding asthma. Pharmacological agents are

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429
Volume 6, Issue 6, November 2026
Impact Factor: 8.2

directed at one or more of these steps: relaxing contracted smooth muscle, dampening the inflammatory cascade, or blocking specific mediator pathways such as the leukotrienes.

Figure 1. Schematic Cross-Section: Normal vs Asthmatic Airway



III. CLASSIFICATION OF ANTI-ASTHMATIC AGENTS

Drugs used in asthma are broadly grouped as relievers (bronchodilators used for rapid symptom relief) and controllers (agents used for long-term suppression of airway inflammation). The three classes reviewed here illustrate this distinction well: short-acting β2-agonists function purely as relievers, inhaled corticosteroids are the cornerstone controller therapy, long-acting β2-agonists provide sustained bronchodilation but are used only alongside a controller, and leukotriene receptor antagonists serve as an oral controller option, particularly useful in specific asthma phenotypes.

IV. B2-ADRENERGIC AGONISTS

β2-agonists remain the most rapidly effective bronchodilators available. They act on β2-adrenergic receptors located on airway smooth muscle, activating adenylyl cyclase and raising intracellular cyclic AMP, which in turn lowers intracellular calcium and produces smooth muscle relaxation. Beyond bronchodilation, β2-receptor stimulation also stabilises mast cell membranes and enhances mucociliary clearance, contributing modestly to overall symptom control.

4.1 Salbutamol (Short-Acting β2-Agonist)

Salbutamol is the prototype short-acting β2-agonist (SABA) and remains the preferred agent for immediate relief of acute bronchospasm and for pre-treatment of exercise-induced bronchoconstriction. Administered by metered-dose inhaler, nebuliser, or dry-powder device, it produces bronchodilation within about five minutes, reaching peak effect at 30 minutes to one hour and lasting four to six hours. Its selectivity for β2- over β1-receptors is relatively high but not absolute, so tachycardia, palpitations, and fine skeletal muscle tremor may occur, particularly with frequent or nebulised dosing. Hypokalaemia can result from β2-mediated intracellular potassium shift and warrants monitoring in patients receiving high or repeated doses, especially alongside other hypokalaemic drugs. A key teaching point for pharmacy students is that increasing SABA use is a clinical warning sign of poor asthma control and should prompt a step-up in controller therapy rather than simple reassurance.

Pharmacokinetically, only a small fraction (roughly 10-20%) of an inhaled dose reaches the lower airways, with the remainder deposited in the oropharynx and swallowed; the swallowed portion undergoes first-pass hepatic metabolism, limiting systemic exposure from the oral route in comparison. Onset after inhalation is rapid because the drug acts locally on airway smooth muscle without needing to reach significant systemic concentrations. Elimination occurs mainly via renal excretion of the drug and its sulfate conjugate, with a plasma half-life of around four to six hours.

Typical adult dosing for acute symptom relief is one to two puffs (100 micrograms per puff) of a pressurised metered-dose inhaler as required, up to a maximum of eight puffs in 24 hours in most protocols, while nebulised solution (2.5-5 mg) is reserved for moderate-to-severe exacerbations or when inhaler technique is inadequate. In children, doses are adjusted by age and body weight, and a spacer device is strongly recommended to improve lung deposition and reduce oropharyngeal side effects. Clinically important drug interactions include an additive risk of tachycardia and arrhythmia

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 6, Issue 6, November 2026 Impact Factor: 8.2

with other sympathomimetics, blunting of bronchodilator response by non-selective β-blockers (which should generally be avoided in asthma), and an increased risk of hypokalaemia when salbutamol is combined with xanthine derivatives, corticosteroids, or non-potassium-sparing diuretics.

4.2 Formoterol (Long-Acting β2-Agonist)

Formoterol is a long-acting β2-agonist (LABA) distinguished from older LABAs by a notably fast onset of action, comparable to salbutamol, combined with a duration of bronchodilation extending to roughly twelve hours. This dual profile has allowed formoterol to be used both as part of scheduled maintenance therapy and, when combined with an inhaled corticosteroid, as an as-needed reliever within maintenance-and-reliever regimens recommended by current asthma guidelines. Formoterol must never be prescribed as monotherapy in asthma; used alone, LABAs have been associated with an increased risk of severe exacerbations and asthma-related death, a risk that is substantially mitigated when the LABA is combined with an adequate dose of inhaled corticosteroid. Adverse effects mirror those of salbutamol – tremor, tachycardia, and occasional hypokalaemia – though these tend to be less prominent at therapeutic inhaled doses.

Formoterol's high lipophilicity allows it to partition into the smooth muscle cell membrane and re-enter the vicinity of the β2-receptor gradually, which is thought to explain its extended duration of action despite a conventional fast onset. Systemic bioavailability from the inhaled route is low, and metabolism proceeds mainly via direct glucuronidation and O-demethylation, with renal excretion of metabolites. Standard maintenance dosing is 6-12 micrograms twice daily by inhalation, usually delivered together with an inhaled corticosteroid in a single combination device (for example, budesonide-formoterol), which also underlies its use in "single-inhaler maintenance and reliever therapy" (SMART/MART) regimens, where the same inhaler is used both for scheduled dosing and for as-needed relief of breakthrough symptoms.

V. INHALED CORTICOSTEROIDS: BUDESONIDE

Inhaled corticosteroids (ICS) are regarded as the most effective long-term controller therapy in persistent asthma of any severity. Budesonide, a non-halogenated corticosteroid with high topical anti-inflammatory potency and relatively low systemic bioavailability due to extensive first-pass hepatic metabolism, is widely used both alone and in fixed-dose combination with formoterol.

Mechanistically, budesonide diffuses across the airway epithelial cell membrane and binds cytoplasmic glucocorticoid receptors. The activated receptor complex translocates to the nucleus, where it modulates gene transcription: pro-inflammatory genes encoding cytokines, chemokines, and adhesion molecules are down-regulated, while anti-inflammatory mediators are up-regulated. The net effect is reduced eosinophilic and lymphocytic infiltration of the airway mucosa, decreased mucosal oedema, reduced mucus hypersecretion, and a gradual reduction in bronchial hyperresponsiveness. Unlike bronchodilators, this anti-inflammatory action develops over days to weeks of regular use rather than providing immediate relief, and patients must be counselled accordingly to maintain adherence even when symptoms are absent.

Local adverse effects, including oropharyngeal candidiasis, hoarseness, and reflex cough, arise from deposition of drug in the oropharynx and can be substantially reduced by rinsing the mouth after each dose and by using a spacer device with pressurised inhalers. Systemic absorption at standard inhaled doses is low, so effects such as adrenal suppression, growth restriction in children, or reduction in bone mineral density are uncommon but become more relevant at high cumulative doses over prolonged periods, warranting periodic dose review and use of the lowest effective dose.

Budesonide has an oral bioavailability of only around 10-13% owing to extensive first-pass metabolism by hepatic CYP3A4, which limits systemic exposure from the swallowed fraction of an inhaled dose; the small proportion that does reach the circulation is highly protein bound and eliminated with a half-life of roughly two to three and a half hours. It is available as a pressurised metered-dose inhaler, a dry-powder inhaler, and a nebulised suspension (the latter also used in paediatric croup), typically dosed twice daily at 100-800 micrograms depending on disease severity, with

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 6, Issue 6, November 2026 Impact Factor: 8.2

once-daily dosing feasible in well-controlled mild disease. Because its metabolism depends on CYP3A4, concomitant use of potent CYP3A4 inhibitors such as ketoconazole, itraconazole, or ritonavir can substantially raise systemic budesonide exposure and increase the risk of corticosteroid-related adverse effects, so such combinations should be used cautiously and generally avoided where alternatives exist.

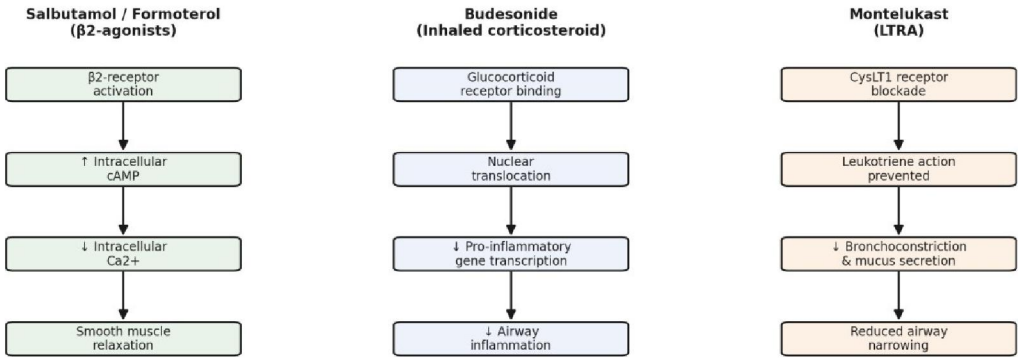
VI. LEUKOTRIENE RECEPTOR ANTAGONISTS: MONTELUKAST

Montelukast represents an oral, non-steroidal alternative or adjunct in asthma control, acting through selective and competitive antagonism of the cysteinyl leukotriene type-1 (CysLT1) receptor on airway smooth muscle and inflammatory cells. Cysteinyl leukotrienes (LTC4, LTD4, LTE4) are potent mediators of bronchoconstriction, increased vascular permeability, and mucus secretion, generated predominantly via the 5-lipoxygenase pathway of arachidonic acid metabolism in mast cells and eosinophils. By blocking their receptor, montelukast reduces bronchoconstriction and airway inflammation, though its overall anti-inflammatory effect is generally considered milder than that of inhaled corticosteroids.

Administered orally once daily, usually in the evening, montelukast offers the practical advantage of avoiding inhaler technique altogether, which makes it particularly useful in young children, in patients with poor inhaler adherence or technique, and in those with concomitant allergic rhinitis, since leukotriene pathways contribute to both conditions. It is also of specific value in aspirin-exacerbated respiratory disease and in exercise-induced bronchoconstriction. Montelukast is generally well tolerated; headache and gastrointestinal disturbances are the most frequently reported effects. Regulatory agencies have highlighted rare but important neuropsychiatric effects, including sleep disturbance, agitation, and mood changes, which pharmacists should be alert to when counselling patients or caregivers.

Following oral administration, montelukast is rapidly absorbed, with an oral bioavailability of about 64% for the film-coated tablet; it is more than 99% bound to plasma proteins and undergoes extensive hepatic metabolism, primarily via CYP3A4 and CYP2C9, with excretion almost entirely through the bile. Standard adult dosing is 10 mg once daily, while paediatric formulations (4 mg or 5 mg chewable tablets, or oral granules) are dosed according to age. Because montelukast is metabolised by CYP3A4/2C9, potent inducers of these enzymes, such as phenobarbital or rifampicin, can reduce its plasma concentration and potentially its clinical efficacy, and this should be considered when asthma control appears to worsen unexpectedly in a patient established on montelukast.

Figure 2. Simplified Mechanism of Action of the Three Drug Classes



VII. DRUG INTERACTIONS: A COMPARATIVE SUMMARY

While each drug's individual interaction profile has been discussed above, several cross-cutting points are worth emphasising for safe co-prescribing. Non-selective β-blockers (including many ophthalmic preparations used for glaucoma) can antagonise the bronchodilator effect of both salbutamol and formoterol and are best avoided in patients with asthma. Concomitant use of β2-agonists with xanthine derivatives (such as theophylline), corticosteroids, or diuretics may have an additive hypokalaemic effect, which becomes clinically relevant in patients also taking digoxin,

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 6, Issue 6, November 2026 Impact Factor: 8.2

where hypokalaemia increases the risk of digoxin toxicity. Budesonide's dependence on hepatic CYP3A4 metabolism makes it susceptible to clinically significant interactions with potent CYP3A4 inhibitors (ketoconazole, itraconazole, clarithromycin, ritonavir), which can raise systemic corticosteroid exposure. Montelukast's metabolism via CYP3A4/2C9 means that enzyme inducers can reduce its efficacy, while it has a comparatively low potential to interact with other commonly co-prescribed drugs, contributing to its favourable safety profile in polypharmacy. Pharmacists should always review a patient's full medication list, including over-the-counter and herbal products, when initiating or adjusting any of these agents.

VIII. USE IN SPECIAL POPULATIONS

8.1 Pregnancy and Lactation

Uncontrolled asthma during pregnancy poses greater risk to both mother and fetus – including pre-eclampsia, preterm birth, and low birth weight – than the risks associated with standard asthma medications, so treatment should generally be continued rather than withheld. Salbutamol and budesonide both have a long history of use in pregnancy and are considered the preferred SABA and ICS respectively, with extensive clinical experience supporting their safety. Data on formoterol and montelukast in pregnancy are more limited, and formoterol is typically continued only when already required for adequate control, in combination with an ICS. Small amounts of inhaled corticosteroids and β2-agonists pass into breast milk, but the quantities are generally considered too low to pose a risk to the breastfed infant.

8.2 Paediatric Use

Children require age-appropriate device selection: pressurised inhalers with a spacer (and a face mask in infants and toddlers) are preferred over dry-powder devices, which demand an inspiratory flow that young children often cannot generate reliably. Growth velocity should be monitored in children on long-term inhaled corticosteroids, as a small, usually transient, reduction in growth rate has been described with regular ICS use, reinforcing the principle of using the lowest effective dose. Montelukast's chewable tablet and granule formulations make it a convenient oral option in this age group, though caregivers should be counselled specifically on the possibility of behavioural or mood-related side effects.

8.3 Elderly Patients and Organ Impairment

Older adults may have coexisting cardiovascular disease that increases sensitivity to the tachycardic and arrhythmogenic potential of β2-agonists, warranting cautious dose titration and monitoring. Hepatic impairment can reduce first-pass metabolism of both budesonide and montelukast, potentially increasing systemic exposure, so dose adjustment or closer monitoring may be warranted in patients with significant liver disease. Renal impairment has comparatively little effect on the disposition of these four drugs, as none rely predominantly on renal elimination of active drug.

IX. PATIENT COUNSELLING AND THE ROLE OF THE PHARMACIST

Correct inhaler technique is one of the most common yet correctable causes of poor asthma control, and pharmacists are often best placed to identify and correct technique errors at the point of dispensing. Key counselling points include: demonstrating slow, deep inhalation coordinated with actuation for pressurised inhalers (or a fast, forceful inhalation for dry-powder devices); recommending a spacer device with pressurised inhalers to improve lung deposition and reduce oropharyngeal deposition; instructing patients to rinse the mouth and spit (not swallow) after each dose of an inhaled corticosteroid to reduce the risk of oral candidiasis and hoarseness; and reinforcing that controller therapy (ICS, ICS-LABA, or montelukast) must be taken regularly even in the absence of symptoms, since its benefit is preventive rather than immediate.

Pharmacists should also routinely ask about the frequency of SABA use, since reliance on salbutamol more than twice a week (excluding pre-exercise use) is a recognised marker of inadequate asthma control and should prompt referral for step-up therapy or medical review. Patients and caregivers started on montelukast should be counselled to report any new mood changes, sleep disturbance, or behavioural changes promptly. Finally, an individualised written asthma

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429
Volume 6, Issue 6, November 2026
Impact Factor: 8.2

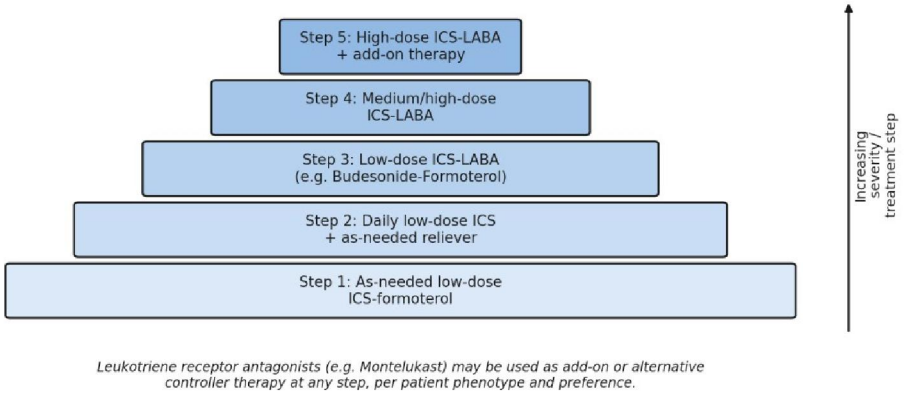
action plan, developed with the prescriber, helps patients recognise worsening control early and know precisely which medications to adjust and when to seek urgent care.

X. INTEGRATING THE THREE CLASSES IN CLINICAL PRACTICE

Contemporary asthma management, as reflected in international guideline frameworks such as the Global Initiative for Asthma (GINA), favours a stepwise approach in which low-dose ICS-formoterol is now recommended as the preferred reliever even at the mildest stages of disease, replacing SABA-only treatment wherever possible. This shift recognises that SABA-only use fails to address the underlying inflammation and may mask worsening disease. As severity increases, the dose of inhaled corticosteroid is increased and/or a long-acting β2-agonist is added, always in fixed combination with an ICS rather than alone. Montelukast may be used as an add-on controller, as an alternative to ICS in selected patients unable or unwilling to use inhaled therapy, or as monotherapy in mild disease with a strong allergic-rhinitis component, though it is generally regarded as less effective than ICS for typical eosinophilic asthma. The comparative features of the four drugs discussed are summarised below.

Drug	Class	Mechanism	Route / Onset	Key Adverse Effects
Salbutamol (Albuterol)	Short-acting β2-agonist (SABA)	Selective β2-receptor agonism → ↑cAMP → bronchial smooth muscle relaxation	Inhalation; onset 5 min, duration 4-6 h	Tremor, tachycardia, hypokalaemia, palpitations
Formoterol	Long-acting β2-agonist (LABA)	Highly lipophilic β2-agonist with prolonged receptor engagement	Inhalation; onset 1-3 min, duration ~12 h	Tremor, tachycardia; monotherapy risk without ICS
Budesonide	Inhaled corticosteroid (ICS)	Glucocorticoid receptor activation → ↓cytokine transcription, ↓airway inflammation	Inhalation; effect builds over days-weeks	Oral candidiasis, dysphonia, cough; systemic effects rare at inhaled doses
Montelukast	Leukotriene receptor antagonist (LTRA)	CysLT1 receptor blockade → prevents leukotriene-mediated bronchoconstriction	Oral; once daily	Headache, GI upset; rare neuropsychiatric effects

Figure 3. Stepwise Pharmacological Approach to Asthma Control (GINA-based concept)



IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429
Volume 6, Issue 6, November 2026
Impact Factor: 8.2

XI. CONCLUSION

Rational pharmacotherapy of bronchial asthma depends on recognising the complementary roles of bronchodilators and anti-inflammatory agents. Salbutamol provides rapid relief of acute bronchospasm but does not treat the underlying inflammation; formoterol extends bronchodilator cover but must always be paired with an inhaled corticosteroid; budesonide addresses the chronic inflammatory basis of the disease and remains the preferred long-term controller; and montelukast offers a useful oral alternative or adjunct, particularly in specific patient subgroups. Careful attention to pharmacokinetics, drug interactions, and the needs of special populations further refines therapy on an individual basis. A sound understanding of the mechanisms, pharmacokinetics, and safety profiles of these agents equips the pharmacy professional to counsel patients effectively, to reinforce correct inhaler technique and adherence, and to recognise early warning signs of poor asthma control.

REFERENCES

1. Global Initiative for Asthma (GINA). Global Strategy for Asthma Management and Prevention. GINA Report, latest update.
2. Tripathi KD. Essentials of Medical Pharmacology. Jaypee Brothers Medical Publishers.
3. Brunton LL, Knollmann BC (eds). Goodman & Gilman's The Pharmacological Basis of Therapeutics. McGraw-Hill.
4. Rang HP, Ritter JM, Flower RJ, Henderson G. Rang and Dale's Pharmacology. Elsevier.
5. British National Formulary (BNF). Respiratory system: Asthma chapter.
6. Katzung BG, Vanderah TW (eds). Basic and Clinical Pharmacology. McGraw-Hill.
7. Sweetman SC (ed). Martindale: The Complete Drug Reference. Pharmaceutical Press.
8. U.S. Food and Drug Administration. Prescribing information and safety communications for montelukast.
9. National Institute for Health and Care Excellence (NICE). Asthma: diagnosis, monitoring and chronic asthma management.
10. DiPiro JT, et al. (eds). Pharmacotherapy: A Pathophysiologic Approach. McGraw-Hill.