Basra Health Directorate

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CLINICAL PHARMACY MONOGRAPH

Clinical Pharmacy Q & A Handbook

in Hereditary Blood Diseases

A Practical Bedside Reference in Pediatric Hematology Pharmacotherapy

COMPILED BY

Department of Pharmacy

Basra Center for Hereditary Blood Diseases

UNDER THE AUSPICES OF

Iraqi Association for Medical Research and Studies (IAMRS)

& Basra Health Directorate

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Disclaimer

The content of this handbook is intended for educational purposes and as a quick clinical reference for healthcare professionals practicing in the field of pediatric hematology. Every effort has been made to ensure accuracy of doses, indications, and recommendations at the time of publication. However, drug information and clinical guidelines may change. The reader is strongly advised to verify drug doses and indications against the most recent product Summary of Product Characteristics (SmPC), local protocols, and current evidence-based guidelines before clinical application. The authors and publishers accept no responsibility for any errors or omissions or for any consequences arising from the use of the information contained herein.

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Preface

The care of patients with hereditary blood diseases — particularly transfusion-dependent thalassemia, sickle cell disease, hemophilia, von Willebrand disease, and other congenital coagulation disorders — requires the close collaboration of multiple disciplines: hematology, pediatrics, pharmacy, nursing, and laboratory medicine. Within this multidisciplinary care, the clinical pharmacist plays a pivotal role in the safe, accurate, and evidence-based use of complex therapies that include iron chelators, clotting-factor concentrates, recombinant biologics, immunomodulators, and supportive care medications.

This handbook has been compiled from real clinical questions raised by the medical and pharmacy teams at the Basra Center for Hereditary Blood Diseases. The questions reflect day-to-day practical dilemmas in pediatric hematology — dosing in special populations, drug administration techniques, compatibility and interaction queries, and selection between therapeutic alternatives — and each answer has been verified against current international guidelines and authoritative drug information resources, which are cited throughout the text using the Vancouver referencing style.

The handbook is organized into two parts. Part I covers the original clinical questions and answers, spanning iron chelation, hemophilia therapy, immunosuppression, antibiotics, anticonvulsants, and supportive care. Part II contains a focused section on hydroxyurea — a cornerstone disease-modifying therapy in sickle cell disease — addressing its mechanism, dosing, monitoring, safety, patient counseling, and the factors that predict suboptimal response. A complete list of references is provided at the end of the booklet.

We hope this handbook will serve as a practical bedside companion for clinical pharmacists, hematologists, pediatricians, residents, and nursing staff working with children who live with hereditary blood diseases — and that it will contribute to the safer, more standardized, and more compassionate care of these patients.

The Editorial Team

Department of Pharmacy,

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Foreword

It is with considerable esteem that I present the inaugural edition of the Clinical Pharmacy Q&A Handbook in Hereditary Blood Diseases, a scholarly contribution conceived and compiled by the Department of Pharmacy at the Basra Center for Hereditary Blood Diseases, operating under the auspices of the Basra Health Directorate, Ministry of Health, Republic of Iraq, in formal collaboration with the Iraqi Association for Medical Research and Studies (IAMRS).

The conceptual foundation of this volume reflects a convergence of clinical acumen and academic rigour. Hereditary haematological disorders — encompassing transfusion-dependent thalassaemia, sickle cell disease, haemophilia, and congenital coagulopathies — constitute a therapeutic domain of exceptional complexity, wherein pharmacological precision and sustained clinical vigilance are indispensable. By systematically structuring this reference around authentic clinical inquiries posed by physicians, pharmacists, and nursing personnel within an active tertiary care environment, the editors have produced a work that transcends the conventional textbook paradigm, effectively bridging international evidence-based guidelines with the practical exigencies of frontline care.

It is clear The Editorial Team has executed this undertaking with exemplary integrity, thereby establishing a significant milestone in the advancement of clinical pharmacy practice within Iraq and the broader regional medical community.

It is anticipated that this handbook will serve as an authoritative bedside companion for clinical pharmacists, haematologists, paediatricians, medical residents, and nursing professionals, and that it will fulfil its principal aspiration: to contribute meaningfully to the safer, more standardised, and more compassionate management of every patient entrusted to their care.

Dr Durgham Alajwady

President, Iraqi Association for Medical Research and Studies

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PART I

Clinical Pharmacy Questions & Answers

This part collects the original clinical questions raised during the daily work of the pharmacy and medical teams at the Basra Center for Hereditary Blood Diseases. Each question is followed by a concise, evidence-based answer with references listed at the end of the booklet.

Q1. If the child has already eaten breakfast, can the Exjade (deferasirox) dose still be given afterward?

Answer: No. Deferasirox should be taken on an empty stomach, at least 30 minutes before food, preferably at the same time each day. If the morning dose is skipped, give it before the next meal instead. The reason is that food affects the absorption profile of deferasirox tablets for oral suspension, and consistent administration on an empty stomach ensures predictable bioavailability and chelation efficacy.

References: **[1, 2]**

Q2. Can deferoxamine be administered as a 2-hour infusion for the treatment of iron overload in patients with transfusion-dependent thalassemia (TDT)?

Answer: No. The standard protocol for chronic iron overload in TDT patients requires deferoxamine to be infused over 8–12 hours (typically via a subcutaneous pump overnight), 5–7 nights per week — not compressed into a 2-hour infusion. Compressing the infusion would significantly increase the infusion rate, raising the risk of hypotension and anaphylactoid reactions, while also reducing the chelation efficacy that depends on slow, sustained drug exposure due to deferoxamine's short plasma half-life (~20–30 min).

References: **[3, 4]**

Q3. What is the best iron chelation treatment for a child with transfusion-dependent thalassemia (TDT) who has iron overload and elevated liver enzymes?

Answer: Deferoxamine is generally the safest choice when liver enzymes are elevated, because it is not associated with hepatotoxicity to the same extent as oral chelators. Deferasirox carries a boxed warning for hepatic failure and is associated with transaminase elevations; deferiprone has been associated with hepatic enzyme elevations as well. Therefore, in a child with elevated liver enzymes, parenteral deferoxamine remains the preferred option, with close monitoring of LFTs.

References: **[3, 5]**

Q4. What is the recommended dose of NovoEight® (turoctocog alfa) for a child with severe Hemophilia A presenting with moderate oral cavity bleeding?

Answer: For a child with severe Hemophilia A and moderate oral cavity bleeding, the usual treatment goal is to raise the Factor VIII level to approximately 50–80% of normal. The recommended dose of NovoEight® (turoctocog alfa) is generally 25–40 IU/kg administered intravenously. The dose can be calculated using the following formula:

Dose (IU) = Body weight (kg) × Desired Factor VIII increase (%) × 0.5

Since each 1 IU/kg of Factor VIII increases the plasma Factor VIII level by approximately 2%, the exact dose should be individualized according to the patient's weight, bleeding severity, and clinical response.

References: **[6, 7]**

Q5. What is the recommended dose of methylprednisolone in alloimmune conditions?

Answer: The usual dose of IV methylprednisolone in severe alloimmune conditions (e.g., severe alloimmune haemolytic anaemia, alloimmune thrombocytopenia) is 10–20 mg/kg/day for 3 days, depending on the clinical indication and specialist guidance. Pulse high-dose steroids should be administered as a slow IV infusion with cardiac and blood-pressure monitoring.

References: **[8]**

Q6. What is the recommended dose of IV labetalol infusion in children?

Answer: The recommended IV infusion dose of labetalol in children is 0.25–3 mg/kg/hour, adjusted according to blood pressure response and continuous monitoring. Labetalol is preferred for hypertensive emergencies because it provides combined α- and β-blockade with a rapid onset and a relatively predictable dose-response.

References: **[8, 9]**

Q7. Can salbutamol (Ventolin) nebulizer be mixed with budesonide (Pulmicort) in the same nebulizer session?

Answer: Yes. Salbutamol and budesonide may be administered together in the same nebulizer session. This combination is commonly used in the management of acute exacerbations of asthma, and physical/chemical compatibility of the two solutions in the nebulizer chamber has been documented. Always use the nebulizer immediately after mixing and do not store the mixed solution.

References: **[10, 11]**

Q8. Can ondansetron syrup be used in infants younger than 6 months?

Answer: Use in infants younger than 6 months is not routinely licensed and should only be considered under specialist advice when the expected benefits outweigh the risks. Limited efficacy and safety data exist in this age group, and the risk of QT prolongation must be considered. When used, the lowest effective dose with ECG monitoring is recommended.

References: **[8, 12]**

Q9. Which antihypertensive drugs may be used in acute nephritis?

Answer: In acute nephritis (especially acute post-streptococcal glomerulonephritis), hypertension is mainly caused by salt and water retention. Management options include:

- First-line measures: restriction of sodium and fluids; loop diuretics such as furosemide.
- Calcium-channel blockers: e.g., amlodipine, nifedipine.
- Beta-blockers: e.g., labetalol, atenolol.
- Vasodilators: e.g., hydralazine.

ACE inhibitors (e.g., enalapril, captopril) and ARBs (e.g., losartan) can be used but are NOT first-line during the acute phase of nephritis, particularly when there is acute kidney injury, reduced GFR, or hyperkalemia, because they may worsen renal function and raise potassium.

References: **[13, 14]**

Q10. What is the recommended pediatric dose of tigecycline?

Answer: Tigecycline pediatric dosing is age-dependent:

• Children 8–11 years: Loading dose 1.2 mg/kg IV (maximum 50 mg), then 0.6 mg/kg every 12 hours (maximum 25 mg per dose).

• Children 12–17 years: Loading dose 100 mg IV, then 50 mg every 12 hours.

Tigecycline should be reserved for serious infections caused by multidrug-resistant organisms when alternatives are unsuitable, because of a black-box warning for increased mortality risk in some indications.

References: **[8, 15]**

Q11. Can vitamin K cause epistaxis?

Answer: No. There is no direct association between vitamin K administration and epistaxis. Epistaxis is usually related to other underlying causes such as nasal mucosal trauma, dry mucosa, infection, hypertension, or an underlying coagulation disorder. Vitamin K actually supports normal coagulation by enabling carboxylation of factors II, VII, IX and X.

References: **[8]**

Q12. Is there an interaction between vancomycin and azithromycin?

Answer: There is no major pharmacokinetic interaction listed between vancomycin and azithromycin. However, azithromycin can prolong the QT interval, particularly in patients with underlying cardiac risk factors. Vancomycin itself is not a major contributor to QT prolongation. Use together cautiously and consider ECG monitoring in patients with QT-prolongation risk factors (electrolyte disturbances, congenital long-QT syndrome, concurrent QT-prolonging drugs).

References: **[16, 17]**

Q13. Why should pethidine be used cautiously in pediatric patients with sickle cell disease?

Answer: Pethidine (meperidine) is metabolized to norpethidine, which has a long half-life and can accumulate, especially in renal impairment or with repeated dosing. Norpethidine accumulation increases the risk of neurotoxicity, seizures, and respiratory depression. For these reasons, morphine is generally preferred for the management of moderate-to-severe vaso-occlusive pain crises in sickle cell disease.

References: **[18, 19]**

Q14. Is teicoplanin available at our center for a patient with MRSA infection?

Answer: No. At present, only vancomycin is available for MRSA coverage at our center. When using vancomycin, consider the following: vancomycin is more nephrotoxic than teicoplanin, so monitor renal function and trough levels; monitor for ototoxicity, especially with prolonged therapy or

concurrent ototoxic drugs; administer by slow IV infusion (over at least 60 minutes per gram) to reduce the risk of infusion-related reactions ("Red Man Syndrome").

References: **[20, 21]**

Q15. Can recombinant Factor VIIa be used for Glanzmann Thrombasthenia?

Answer: Yes. Recombinant activated Factor VII (rFVIIa, NovoSeven®) can be used in Glanzmann Thrombasthenia (GT), although it is not first-line — platelet transfusions are first-line, and rFVIIa is reserved for patients with platelet refractoriness, anti-HLA or anti-GPIIb/IIIa antibodies, or when platelets are unavailable. The commonly cited dose is 90 mcg/kg, repeated every 2 hours for at least 3 doses, until hemostasis is achieved.

References: **[22, 23]**

Q16. What is the scientific name and mechanism of action of Acupan® ampoule, and is it safe to use in pediatric patients and patients with sickle cell disease?

Answer: Acupan® contains nefopam, a centrally acting non-opioid analgesic that inhibits the reuptake of serotonin, norepinephrine, and dopamine. Nefopam is NOT routinely recommended in pediatric patients (efficacy and safety data are limited in children under 12 years), and it is NOT considered a standard treatment for pain crises in sickle cell disease. International sickle cell pain guidelines recommend a stepwise approach using paracetamol, NSAIDs, and opioids (morphine) for moderate-to-severe pain.

References: **[8, 18]**

Q17. Can dispersible deferasirox tablets be crushed and mixed with food?

Answer: Dispersible deferasirox tablets should NOT be crushed. They must be dissolved in water, orange juice, or apple juice according to manufacturer recommendations, stirred until a fine suspension is obtained, and the suspension should be drunk immediately. Any residue should be re-suspended in a small amount of liquid and swallowed. (Note: this applies to the dispersible formulation. The newer film-coated tablet [Jadenu®] may be crushed and sprinkled on soft food.)

References: **[1, 2]**

Q18. Should azithromycin be taken with or without food?

Answer: Azithromycin tablets and oral suspension may be taken either with or without food. Taking the dose with food may help reduce gastrointestinal upset. For optimal absorption of the older immediate-release oral suspension, the patient may take it on an empty stomach, but the modern formulations are not significantly affected by food.

References: **[8, 24]**

Q19. What is the loading and maintenance dose of phenobarbital (Luminal) in children?

Answer: Phenobarbital pediatric dosing for status epilepticus / seizure control:

- Loading dose: 15–20 mg/kg IV, infused slowly (not exceeding 1 mg/kg/minute, maximum 60 mg/min).
- Maintenance dose: 3–5 mg/kg/day, given in 1–2 divided doses.

Monitor serum levels (therapeutic range 15–40 mg/L) and watch for sedation, respiratory depression, and hypotension during loading.

References: **[8, 25]**

Q20. What is the recommended dose of romiplostim (Nplate®)?

Answer: Romiplostim is usually started at 1 microgram/kg subcutaneously once weekly, and adjusted weekly based on platelet response, up to a maximum of 10 micrograms/kg per week. The goal is to maintain a platelet count of at least 50×10^9/L sufficient to avoid bleeding. The dose should be reduced or held if platelets rise above 200–400 $\times 10^9$/L to avoid thrombotic complications.

References: **[26, 27]**

Q21. What Wilate® product is available at our Hematology Center, and what are the differences between it and the available Haemate P®?

Answer: Wilate® (available at our center as 500 IU VWF / 500 IU FVIII per vial) has a physiological VWF:FVIII ratio of approximately 1:1, providing effective replacement of both factors.

Haemate P® (available at our center as 1200 IU VWF / 500 IU FVIII per vial) has a higher VWF:FVIII ratio of approximately 2.4:1, providing more VWF with relatively less FVIII exposure.

The choice between them depends on the indication: Haemate P® is preferred when a higher VWF load is desired with less FVIII accumulation (e.g., long-term prophylaxis in von Willebrand disease), while Wilate® is preferred when a more balanced replacement is needed.

References: **[28, 29]**

Q22. What is the drug of choice for cardiac iron overload (MRI T2* showing myocardial iron accumulation) in a TDT patient?

Answer: Deferiprone (alone or in combination with deferoxamine) is generally preferred for cardiac iron overload, because deferiprone penetrates cardiomyocytes more effectively and removes intracellular myocardial iron better than deferoxamine alone. The combination of deferiprone + deferoxamine has been shown to reduce cardiac iron and improve LV ejection fraction in TDT patients with severe cardiac siderosis, and is the standard for severe cardiac iron overload (T2* <10 ms).

References: **[3, 30]**

Q23. In a patient with Hemophilia A and inhibitors who is receiving Hemlibra® (emicizumab) and is scheduled for major surgery, should emicizumab be continued or discontinued? What is the perioperative Factor VIIa treatment protocol?

Answer: Emicizumab should be continued during the perioperative period — it is not discontinued for surgery. Additional bleeding-control therapy is required because emicizumab does not provide full surgical hemostasis on its own.

Recombinant Factor VIIa (NovoSeven® RT) is the first choice for bleeding control in patients with inhibitors on emicizumab. Recommended perioperative dosing:

- 90 mcg/kg immediately before surgery
- Repeat 90 mcg/kg every 2 hours for the duration of the procedure
- Postoperatively: 90 mcg/kg every 2–6 hours, gradually spacing as hemostasis is achieved
- Continue for approximately 7 days postoperatively, then taper.

Avoid concurrent activated prothrombin complex concentrate (aPCC) >100 U/kg in 24 h with emicizumab due to the risk of thrombotic microangiopathy.

References: **[31, 32]**

Q24. What is the recommended dose of Haemocomplettan® P (fibrinogen concentrate), and can it be used for prophylaxis in congenital fibrinogen deficiency?

Answer: Treatment (acute bleeding): 70 mg/kg (range 50–100 mg/kg) IV, or calculated as:

Dose (mg/kg) = [target fibrinogen − measured fibrinogen (g/L)] / 0.014

Prophylaxis: Yes — Haemocomplettan® P can be used for long-term prophylaxis in congenital fibrinogen deficiency at a typical dose of 20–40 mg/kg every 7–14 days, adjusted to maintain trough fibrinogen ≥1 g/L.

Reconstitution: with Water for Injection (WFI) only. Normal saline is NOT recommended by the manufacturer.

References: **[33, 34]**

Q25. What is Xromi®?

Answer: Xromi® is the liquid (oral solution) dosage form of hydroxyurea (hydroxycarbamide), strength 100 mg/mL, strawberry-flavored. It is indicated for the prevention of vaso-occlusive complications of sickle cell disease in patients ≥9 months of age. The liquid formulation is intended to enable use in young children and patients with difficulty swallowing capsules, and to allow accurate weight-based dosing.

References: **[35, 36]**

Q26. What is Octaplex®?

Answer: Octaplex® is a human plasma-derived four-factor prothrombin complex concentrate (4F-PCC). It contains the vitamin K-dependent coagulation factors II, VII, IX, and X, together with proteins C and S. It is used primarily for the urgent reversal of bleeding caused by vitamin K antagonists (e.g., warfarin) and for the management of acquired or congenital factor II, VII, IX, or X deficiencies when purified single-factor concentrates are unavailable.

References: **[37, 38]**

Q27. What is the recommended dose of phenytoin in children?

Answer: Phenytoin pediatric dosing:

• Loading dose (status epilepticus): 20 mg/kg IV as a single dose, infused slowly at a rate not exceeding 1–3 mg/kg/min (maximum 50 mg/min in adults; lower in neonates).

• Maintenance dose: 2.5–5 mg/kg twice daily IV/PO (for children up to 12 years), starting approximately 12 hours after the loading dose.

Monitor serum levels (target 10–20 mg/L total, or free phenytoin 1–2 mg/L), and watch for hypotension and arrhythmia during IV loading.

References: **[8, 25]**

Q28. Is there an interaction between phenobarbital and phenytoin?

Answer: Yes. Phenobarbital and phenytoin are both potent hepatic cytochrome-P450 enzyme inducers, and they may alter each other's serum concentrations in an unpredictable bidirectional way. Co-administration requires therapeutic drug monitoring of both agents. The combination may also potentiate sedation and cognitive side effects.

References: **[8, 39]**

Q29. What is the pediatric dose of levetiracetam?

Answer: Initial dose: 10 mg/kg twice daily orally or IV, increased gradually (in increments of 10 mg/kg every 2 weeks) up to 25–30 mg/kg twice daily according to response and tolerability. Maximum maintenance dose is 60 mg/kg/day (not exceeding 3000 mg/day). Levetiracetam has a favorable pharmacokinetic profile with minimal drug interactions, making it a useful first-line option in many pediatric seizure disorders.

References: **[8, 40]**

Q30. What is the recommended dose of ceftriaxone in acute chest syndrome?

Answer: Ceftriaxone 50 mg/kg/day IV once daily (maximum 2 g/day) is the standard β-lactam component. However, ceftriaxone alone does not cover atypical organisms (Mycoplasma pneumoniae, Chlamydia pneumoniae), which are common precipitants of acute chest syndrome in children with sickle cell disease. Therefore, ceftriaxone is almost always paired with a macrolide — azithromycin 10 mg/kg (max 500 mg) IV/PO on day 1, then 5 mg/kg (max 250 mg) IV/PO on days 2–5.

References: **[41, 42]**

Q31. Can diclofenac and ketorolac be used together?

Answer: No. Diclofenac and ketorolac are both NSAIDs, and combining two NSAIDs does NOT provide additional analgesic benefit; instead, the combination compounds the risks of gastrointestinal ulceration and bleeding, acute kidney injury, and antiplatelet effects (bleeding). When stronger analgesia is needed, an opioid should be added rather than a second NSAID.

References: **[8, 43]**

Q32. What is the difference between Myfortic® and CellCept®?

Answer: CellCept® is mycophenolate mofetil (MMF) — a prodrug that is rapidly hydrolyzed in the gut and liver to the active moiety, mycophenolic acid (MPA).

Myfortic® is enteric-coated mycophenolate sodium (EC-MPS), designed to delay release of MPA further down the GI tract in order to reduce upper-GI side effects.

The two products are NOT milligram-for-milligram interchangeable: 1 g of CellCept® is roughly equivalent to 720 mg of Myfortic®. Switching between formulations should be done with careful dose conversion and clinical monitoring.

References: **[44, 45]**

Q33. What is the dose of lactulose syrup for a 15-year-old patient?

Answer: The usual dose of lactulose syrup for a 15-year-old (adolescent/adult dosing) is 15–30 mL daily, given in single or divided doses, adjusted according to clinical response (target 2–3 soft stools per day). Onset of action is typically 24–48 hours.

References: **[8, 46]**

Q34. What is the recommended dose of antithymocyte globulin (ATG) in aplastic anemia?

Answer: Two formulations are used; horse ATG is generally preferred over rabbit ATG for first-line treatment of severe aplastic anemia, based on superior response rates in randomized trials:

- Horse ATG (ATGAM®): 40 mg/kg/day IV for 4 consecutive days (total 160 mg/kg).
- Rabbit ATG (Thymoglobulin®): 3–3.5 mg/kg/day IV for 5 days.

ATG is always given with premedication (corticosteroids, antihistamines, antipyretics) to reduce infusion reactions, and is combined with cyclosporine for the immunosuppressive regimen.

References: **[47, 48]**

Q35. Is there an interaction between diclofenac and ondansetron?

Answer: No major pharmacokinetic interaction is reported between diclofenac and ondansetron. However, the two drugs should NOT be mixed in the same syringe or IV infusion line unless physicochemical compatibility has been confirmed, as diclofenac (alkaline) and ondansetron (acidic) may precipitate when mixed.

References: **[49, 50]**

Q36. What is the correct infusion time for IV vancomycin to avoid Red Man Syndrome?

Answer: IV vancomycin must be infused at a rate of no faster than 10 mg/minute and over at least 60 minutes per gram (whichever is longer). Practical guidance:

- Doses up to 1 g → infuse over ≥ 60 minutes.
- 1.5 g → infuse over ~90 minutes.
- 2 g → infuse over ~120 minutes.

If Red Man Syndrome (flushing, pruritus, hypotension) occurs, stop the infusion, give antihistamine (diphenhydramine), and resume at a slower rate once symptoms resolve.

References: **[20, 51]**

Q37. What is the pediatric dose of piperacillin/tazobactam for a child weighing 25 kg with pneumonia?

Answer: For children weighing < 40 kg, the standard dose of piperacillin/tazobactam is 112.5 mg/kg (100 mg piperacillin + 12.5 mg tazobactam) per kg per dose, IV every 6 hours.

For a 25 kg child: 25 × 112.5 = 2812.5 mg piperacillin-tazobactam IV every 6 hours (i.e., approximately 2.5 g piperacillin component every 6 hours), administered as a 30-minute IV infusion.

References: **[8, 52]**

Q38. How should factor VIII (Xyntha®) be administered?

Answer: Factor VIII (Xyntha®, moroctocog alfa) is reconstituted with the supplied diluent (sterile water for injection 4 mL per vial) and administered by slow IV injection over approximately 2–5 minutes. The rate should be adjusted to patient comfort — slower infusion if the patient experiences a sensation of pressure or transient pulse changes.

References: **[53]**

Q39. What is the usual maintenance dose of warfarin in children?

Answer: The typical pediatric maintenance dose of warfarin is 0.09–0.33 mg/kg/day, individually titrated to maintain the INR within the target range for the underlying indication (commonly INR 2.0–3.0 for most indications, 2.5–3.5 for mechanical mitral valves). Younger children (especially infants) often require higher per-kg doses than adolescents because of faster hepatic metabolism. Monitoring should be frequent at initiation and during dose changes.

References: **[8, 54]**

Q40. How should tranexamic acid ampoules be administered IV, and what is the recommended volume for dilution?

Answer: Tranexamic acid for IV administration is typically given as a 1% solution (10 mg/mL) after dilution in 0.9% sodium chloride or 5% dextrose. The maximum recommended infusion rate is 100 mg/minute (and ≤ 5 mL/min of the 1% solution). In clinical practice, doses are typically infused over 10–15 minutes to minimize the risk of hypotension and dizziness. Rapid IV bolus must be avoided.

References: **[8, 55]**

Q41. Is there a drug interaction between morphine and ondansetron?

Answer: Yes — a mild pharmacodynamic interaction is reported. Ondansetron, a 5-HT_3 receptor antagonist, may slightly reduce the analgesic effect of morphine (since serotonergic signaling contributes to opioid-induced analgesia). Both drugs carry a small risk of QT prolongation. Despite this, the combination is widely and safely co-administered in clinical practice (e.g., for opioid-induced nausea and vomiting), with monitoring as appropriate.

References: **[43, 56]**

PART II

Hydroxyurea in Sickle Cell Disease

Hydroxyurea (hydroxycarbamide) is the first disease-modifying therapy approved for sickle cell disease and remains the cornerstone of treatment for children and adults with HbSS or HbSβ^0-thalassemia. The following eight questions and answers address the most common clinical, pharmacologic, monitoring, counseling, and response-related issues that arise when initiating and maintaining hydroxyurea therapy in pediatric patients.

HU-Q1. What is the mechanism of action of hydroxyurea in sickle cell disease?

Answer: Hydroxyurea is a ribonucleotide reductase inhibitor. In sickle cell disease, its principal therapeutic action is induction of fetal hemoglobin (HbF), which interferes with the polymerization of deoxygenated HbS and reduces erythrocyte sickling. Additional mechanisms contribute to its clinical benefit: reduction in total leukocyte and neutrophil counts (decreasing endothelial adhesion), reduction in reticulocyte count, generation of nitric oxide (improving vascular tone and reducing endothelial activation), and reduction in expression of adhesion molecules on red cells and leukocytes.

References: **[57, 58]**

HU-Q2. What is the recommended starting dose and maximum tolerated dose of hydroxyurea in pediatric sickle cell disease?

Answer: The recommended starting dose of hydroxyurea in children with sickle cell anemia (HbSS or HbSβ^0-thalassemia) is 20 mg/kg/day orally, given as a single daily dose. The dose is then escalated by 5 mg/kg/day every 8 weeks based on hematologic response and tolerability, up to a maximum tolerated dose (MTD) of approximately 35 mg/kg/day (not exceeding 35 mg/kg/day in most protocols). The 2014 NHLBI guidelines recommend offering hydroxyurea to all children with sickle cell anemia beginning at 9 months of age, regardless of clinical severity.

References: **[36, 59, 60]**

HU-Q3. How should patients on hydroxyurea be monitored?

Answer: Patients receiving hydroxyurea require regular laboratory monitoring:

- Complete Blood Count (CBC) with differential, reticulocyte count and platelets: every 2–4 weeks during dose escalation, then every 2–3 months once a stable dose is achieved.
- Mean corpuscular volume (MCV) — a rising MCV is an early marker of adherence and pharmacologic effect.
- Fetal hemoglobin (HbF) — should be measured at baseline and every 3–6 months; a rising HbF (target generally ≥ 20%) indicates therapeutic response.
- Renal and hepatic function: at baseline and every 6 months.
- Pregnancy testing in adolescents at risk, given the teratogenic potential.

References: **[60, 61]**

HU-Q4. What are the main contraindications and precautions for hydroxyurea use?

Answer: Main contraindications/precautions for hydroxyurea:

• Pregnancy: hydroxyurea is teratogenic in animal studies and is contraindicated during pregnancy; effective contraception should be used by both male and female patients of reproductive age.

• Lactation: hydroxyurea is excreted in human milk and is generally contraindicated during breastfeeding (although recent pharmacokinetic data suggest very low infant exposure — specialist advice required).

• Severe myelosuppression: absolute neutrophil count (ANC) < 1.5 × 10^9/L, platelets < 80 × 10^9/L, hemoglobin < 4.5 g/dL or reticulocytes < 80 × 10^9/L when hemoglobin < 9 g/dL.

• Severe renal or hepatic impairment: dose reduction required (in renal impairment, start at 50% of the usual dose if CrCl < 60 mL/min).

• Concurrent live vaccines should be avoided during therapy.

References: **[36, 62, 63]**

HU-Q5. What are the most common and serious adverse effects of hydroxyurea?

Answer: Common adverse effects:

• Hematologic — dose-related myelosuppression (neutropenia, thrombocytopenia, reticulocytopenia, macrocytic anemia).

• Dermatologic — hyperpigmentation of nails and skin, dry skin, melanonychia.

• Gastrointestinal — mild nausea, mucositis.

Serious adverse effects (uncommon):

• Cutaneous vasculitis and leg ulcers (more in adults with myeloproliferative disease than in sickle cell patients).

• Secondary malignancies — a long-term, controversial concern; long-term sickle cell cohort data have not demonstrated a clear increased risk in children.

• Reproductive effects: reversible reduction in spermatogenesis; potential teratogenicity.

The FDA label carries boxed warnings for myelosuppression and malignancies.

References: **[36, 60, 64]**

HU-Q6. What is Xromi® and how is it different from conventional hydroxyurea capsules?

Answer: Xromi® is the first authorized liquid (oral solution) formulation of hydroxyurea (hydroxycarbamide), strength 100 mg/mL, strawberry-flavored. It was approved by the EMA in July 2019 and by the FDA in 2024 for the prevention of vaso-occlusive complications of sickle cell disease in patients ≥ 9 months of age.

Differences from conventional capsules:

• Liquid form — suitable for infants, young children, and patients who cannot swallow capsules.

• Allows precise weight-based dosing (especially important during dose escalation to MTD).

• Easier adherence in pediatric patients.

• The capsule formulations (e.g., Hydrea®, Siklos®) remain valid alternatives for older children and adolescents.

References: **[35, 36]**

HU-Q7. What practical counseling points should be discussed with families starting a child on hydroxyurea?

Answer: Key counseling points:

1. Hydroxyurea is a long-term, daily medication — adherence is essential for benefit; clinical effect appears gradually over 3–6 months.

2. Take at the same time each day (typically once daily). If a dose is missed, take it as soon as remembered the same day; do NOT double up.

3. Caregivers handling the capsules/liquid should wash hands after handling; avoid crushing capsules (use the liquid Xromi® instead for young children).

4. Regular blood tests are required — explain the schedule of CBC monitoring.

5. Contraception advice for adolescents — both males and females must use effective contraception, with continued contraception for 3–6 months after stopping treatment.

6. Vaccines: routine inactivated vaccines are safe; avoid live vaccines while on therapy unless specialist advice is given.

7. Report fever, mouth sores, bleeding, severe fatigue, or rashes promptly.

8. Store the oral solution at room temperature, protected from light, and discard 3 months after first opening (per Xromi® SmPC).

References: **[36, 60, 65]**

HU-Q8. What factors predict a poor response to hydroxyurea?

Answer: Approximately 10–25% of patients with sickle cell disease show a suboptimal response to hydroxyurea even after appropriate dose escalation. Identifying the factors that predict poor response is essential to optimize therapy, distinguish true non-response from non-adherence, and guide consideration of alternative or adjunctive therapies. The factors are conventionally grouped into three domains: clinical, adherence/dosing, and pharmacogenetic factors.

Factor	Description (Sickle Cell Disease)
Clinical Factors	
Age	Non-adherence more common in adolescents; older age less clearly linked to poor response than in thalassemia.
Baseline HbF	Low baseline HbF associated with lower magnitude of response.
SCD genotype	HbSC subtype shows less HbF induction than HbSS; response in HbSS is still highly variable.
Adherence and Dosing	
Non-adherence	Most common cause of apparent non-response; rising MCV is the key objective marker confirming adherence.
Subtherapeutic dose (not at MTD)	Fixed low-dose regimens underperform; weight-based escalation to maximum tolerated dose (MTD) is essential before declaring failure.
Pharmacogenetic Factors	
XmnI polymorphism (−158 C>T)	Absence of the favorable T allele (CC genotype) is associated with lower HbF response.
BCL11A variants	Unfavorable BCL11A variants reduce γ-globin induction and limit HbF rise.
HBS1L-MYB (MYB locus)	Unfavorable MYB alleles are associated with reduced HbF induction in poor responders.

NOS1 / ARG2 variants	Affect the nitric oxide pathway; variants may reduce hydroxyurea efficacy beyond HbF induction.
CYP2C9 / CYP2E1 variants	Alter hydroxyurea metabolism, leading to variable drug exposure and unpredictable response.
KLF10 variants	Studied in SCD pharmacogenetic analyses; may modulate HbF regulation.

Practical clinical approach: Practical approach to a poor responder: (1) confirm adherence using MCV trend and pill counts; (2) ensure escalation to maximum tolerated dose (MTD ≈ 30–35 mg/kg/day); (3) reassess after 6 months at MTD; (4) consider pharmacogenetic testing where available; and (5) discuss alternative or additional disease-modifying therapies (chronic transfusion, l-glutamine, crizanlizumab, voxelotor, or hematopoietic stem cell transplantation) in confirmed non-responders.

References: **[60, 66, 67, 68]**

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