

test questions PharmaCE™

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POINT-OF-CARE MONITORING IN ANTICOAGULATION: 1

Goal

To provide information on the availability, mechanisms, limitations, and clinical application of POC devices used in the management of warfarin and parenteral direct thrombin inhibitors.

Objectives

After reviewing this article, the reader should be able to:

1. identify the advantages and disadvantages of POC monitoring of warfarin anticoagulation using the PT-INR;
2. identify tests performed, testing time, and approvals for use of various POC PT-INR monitors;
3. describe advantages of patient self-testing and patient self-monitoring compared with laboratory monitoring of warfarin anticoagulation using PT-INR POC devices;
4. given a list of POC tests, identify recommended tests for monitoring direct thrombin inhibitor anticoagulation during thrombosis treatment, PCI, and CPB.

Test Questions

1. The preferred monitoring test for warfarin anticoagulation is:
(a) ACT.
(b) INR.
(c) PT.

- (d) aPTT ratio.
(e) None of the above is correct.

2. POC testing devices for monitoring warfarin anticoagulation have been studied in which of the following settings?

- (a) patient self-monitoring
(b) anticoagulation clinics
(c) physicians' offices
(d) hospital wards
(e) All of the above are correct.

3. There are several available POC devices that have acceptable performance compared with laboratory monitoring.

- (a) true
(b) false

4. Which of the following POC devices measures aPTT and ACT in addition to PT-INR?

- (a) CoaguChek PST
(b) CoaguChekS
(c) RapidpointCoag
(d) Hemochron Jr. Signature/Plus
(e) INRatio

5. Which of the following POC devices is approved for patient self-testing?

- (a) RapidpointCoag
(b) Hemochron Jr. Signature/Plus
(c) ProTime
(d) Hemochron 401
(e) CoaguChek DM

6. PT-INR POC devices provide results in approximately how many minutes?

- (a) 2
(b) 10
(c) 20
(d) 30
(e) 60

7. Which of the following is an advantage of patient self-testing and self-monitoring compared with laboratory monitoring of PT-INR for warfarin anticoagulation?

- (a) lower mortality observed in clinical trials
(b) improved accuracy of PT-INR
(c) requires less patient training
(d) requires less qualified clinic personnel for management
(e) better anticoagulation control

8. Which of the following POC tests is used to monitor direct thrombin inhibitors during PCI?

- (a) aPTT
(b) ACT
(c) ECT
(d) ENOX test
(e) All of the above are correct.

9. Which POC test displays the best correlation with plasma concentrations of a direct thrombin inhibitor?

- (a) aPTT
(b) ACT
(c) ECT
(d) ENOX test
(e) heparin concentrations

10. Which of the following POC tests is recommended to monitor lepirudin anticoagulation during CPB?

- (a) aPTT
(b) ACT
(c) ECT
(d) ENOX test
(e) heparin concentrations

11. Clinical trials have demonstrated that higher ACT measurements are correlated with an increased rate of bleeding in patients undergoing PCI who are receiving bivalirudin.

- (a) true
(b) false

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IMPACT OF NSAIDs ON THE CARDIOPROTECTIVE EFFECTS OF ASPIRIN

Goal

To evaluate pharmacodynamic and clinical data regarding a potential deleterious effect of nonselective NSAIDs on the cardioprotective effects of aspirin.

Objectives

After reviewing this article, the reader should be able to:

1. describe the pharmacodynamic mechanism by which NSAIDs and aspirin may interact;
2. evaluate clinical pharmacodynamic studies regarding potential NSAID and aspirin interaction;
3. analyze the correlation between pharmacodynamic and patient-focused clinical outcomes studies;

- list limitations of clinical studies investigating this interaction.

Test Questions

1. Which of the following outcomes was *not* demonstrated in the indomethacin/aspirin pharmacodynamic study by Livio et al.¹³?

- Indomethacin produced concentration-dependent inhibition of platelet aggregation in vitro when used alone.
- Indomethacin did not inhibit the antiplatelet effects of aspirin in vitro.
- Indomethacin did not inhibit the antiplatelet effect of aspirin in vivo.
- Aspirin inhibits thromboxane B₂ throughout the 48-hour post-dosing period.
- Thromboxane B₂ concentrations returned to >60% of baseline in the aspirin and indomethacin group at 48 hours.

Questions 2 and 3 refer to the following case:

A 65-year-old male patient with a medical history of hypertension developed low back pain after lifting a heavy television set. He currently takes aspirin and hydrochlorothiazide every morning. He has tried acetaminophen with no relief.

2. What is the expected pharmacodynamic consequence of concomitant aspirin and ibuprofen therapy?

- Platelet aggregation is not affected by either therapy.
- Platelet aggregation, but not thromboxane B₂ inhibition, is reversed when aspirin and ibuprofen are used together.
- Thromboxane B₂ inhibition is reversed when using aspirin before ibuprofen.
- There is no effect on thromboxane B₂ inhibition when aspirin is taken before ibuprofen.
- Thromboxane B₂ inhibition is not affected regardless of administration schedules.

3. Which of the following is a limitation of clinical studies evaluating the concomitant use of aspirin and NSAIDs on cardiovascular events?

- The importance of the results is dependent on the similarity of patients and treatment to those commonly used in practice.
- Clinical outcomes regarding cardiovascular morbidity and mortality are evaluated.
- Small sample size limits extrapolation to practice.
- The extrapolation of results is independent of the study population.
- The randomized design lacks pharmacodynamic interpretation.

Questions 4 and 5 refer to the following case:

An obese 58-year-old woman with a history of osteoporosis and cardiovascular risk factors, including a 20 pack-year

smoking history and a father who died of an MI at age 47, takes alendronate and aspirin daily. The patient's chief complaint is swelling of her left lower extremity and trouble ambulating. Clinical examination and X-ray reveal osteoarthritis in her left knee.

4. Which of the following medication management approaches is appropriate?

- Stop taking daily aspirin and initiate an NSAID.
- Initiate NSAID therapy and use aspirin as needed.
- Initiate NSAID therapy and recommend to take aspirin 2 hours before NSAID.
- NSAID therapy is contraindicated; treat with an opioid.
- Prescribe an NSAID as needed and continue on aspirin.

5. The patient has heard that there may be an interaction between aspirin and ibuprofen, specifically, and asks for your advice. Which of the following statements is most appropriate?

- Studies have not determined whether this interaction is a cause for concern.
- Taking aspirin and ibuprofen together will increase the risk of death.
- Taking both agents is more beneficial than taking aspirin alone to prevent cardiovascular death.
- There is an interaction between aspirin and other pain medications, but not ibuprofen.
- The interaction is only a concern if you are a diabetic.

Questions 6 and 7 refer to the following case:

A 45-year-old man with a medical history of hyperlipidemia and cluster headaches routinely takes atorvastatin and aspirin. The patient's cluster headaches are relieved by ibuprofen use as needed.

6. Which of the following statements regarding the Physician's Health Study¹⁷ is *true* and pertinent to managing the patient's medications?

- Results demonstrated that intermittent NSAID use had no effect on the risk of first MI in patients on aspirin therapy.
- Results demonstrated that intermittent NSAID use significantly increased the risk of first MI in patients on aspirin therapy.
- Female patients were excluded from this analysis.
- Results demonstrated that frequent NSAID use did not affect the risk of first MI in patients on aspirin therapy.
- Results contradict early pharmacodynamic studies.

7. Medication counseling for this patient should include which of the following statements?

- Do not use aspirin on days when you experience cluster headaches.

- Contact your physician if headaches are frequent and you are using ibuprofen often/daily.
- Regardless of when the headache occurs, only use ibuprofen if you have not taken aspirin yet.
- Regardless of when the headache occurs, only use ibuprofen 2–3 hours after you take aspirin.
- Do not take ibuprofen; ask your physician to change your medication.

Questions 8 and 9 refer to the following case:

A 65-year-old male patient with a history of hypertension, diabetes, and MI takes enalapril, hydrochlorothiazide, metformin, atenolol, aspirin, and simvastatin. The patient complains of chronic low back pain and is not able to play golf. He has tried acetaminophen without relief.

8. Which of the following statements is *true* regarding concomitant aspirin and NSAID therapy and the risk of death post-MI?

- Studies have not been conducted in patients post-MI.
- Data show an increased risk of death in post-MI patients taking aspirin and ibuprofen, but not aspirin and other NSAIDs.
- Data show an increased risk of death in post-MI patients taking aspirin and other NSAIDs, but not aspirin and ibuprofen.
- Data show an increased risk of death in patients with cardiovascular disease, including MI, who are taking aspirin and ibuprofen.
- Data show a significantly higher risk of death in patients taking aspirin and ibuprofen or other NSAIDs.

9. Medication management for this patient should include which of the following?

- Increase the dose of aspirin to 650 mg po qid.
- Add naproxen 500 mg po bid for 2 weeks and take naproxen 1–2 hours after aspirin therapy.
- Continue acetaminophen; NSAIDs are contraindicated.
- Add ibuprofen 800 mg tid and stop aspirin for 2 weeks.
- Use only over-the-counter NSAIDs as needed.

10. Which of the following statements is *true* regarding pharmacodynamic and clinical outcomes studies evaluating concomitant use of aspirin and NSAIDs?

- Pharmacodynamic studies are more powerful than clinical outcomes studies.
- Clinical outcomes studies consistently contradict pharmacodynamic studies.
- Single-dose pharmacodynamic studies and clinical studies evaluating intermittent NSAID use do not demonstrate a deleterious interaction.

- (d) Limitations exist to the pharmacodynamic data, but not the clinical outcomes data.
- (e) Neither pharmacodynamic nor clinical outcomes studies utilized surrogate outcome markers.

Questions 11 and 12 refer to the following case:

A 35-year-old female smoker with a history of gestational diabetes and heterozygous familial hypercholesterolemia takes rosuvastatin, metformin, and aspirin. The patient complains of shoulder pain that she attributes to picking up her twin 3-year-old daughters.

11. Which of the following is a factor in determining appropriate pain therapy for this patient?

- (a) Pharmacodynamic study results with aspirin and NSAIDs have clearly translated into deleterious clinical outcomes.
- (b) Randomized, controlled clinical trials have demonstrated a deleterious effect of NSAID therapy on the cardiovascular protective effects of aspirin.
- (c) No relationship has been shown between pharmacodynamic findings and clinical outcomes studies.
- (d) Intermittent use of NSAIDs has not been demonstrated to reverse the cardiovascular effects of aspirin in pharmacodynamic and clinical outcomes studies.

- (e) Chronic use of NSAIDs has not been demonstrated to reverse the cardiovascular effects of NSAIDs in pharmacodynamic and clinical outcomes studies.

12. Which of the following is *not* a limitation to applying the literature to determine appropriate pain therapy for this patient?

- (a) The clinical outcomes studies evaluating a potential NSAID and aspirin interaction are typically retrospective and/or observational in nature.
- (b) The clinical outcomes studies evaluating a potential NSAID and aspirin interaction have primarily included healthy male patients or patients with documented MI.
- (c) Studies involving surrogate markers have shown an interaction between aspirin and NSAIDs, but this effect has not been confirmed in terms of clinical endpoints.
- (d) Differences have been demonstrated with intermittent or chronic NSAID use and the potential to interact with aspirin.
- (e) Data have consistently demonstrated deleterious effects of NSAIDs on aspirin in both clinical and pharmacodynamic endpoints.

13. Inhibition of COX-1 by aspirin therapy alone is:

- (a) reversible and sustained for approximately 24 hours.

- (b) irreversible and sustained for approximately 24 hours.
- (c) reversible and sustained for approximately 10 days.
- (d) irreversible and sustained for approximately 10 days.
- (e) reversible and sustained for approximately 28 days.

14. A common marker of COX-1 activity includes:

- (a) thromboxane B₂.
- (b) platelet antiaggregation.
- (c) C-reactive protein.
- (d) platelet-rich plasma.
- (e) sodium arachidonate.

15. Which of the following is *not* a reason why investigating the potential interaction between NSAIDs and aspirin is important?

- (a) The American Heart Association recommends aspirin for primary prevention of MI in patients at risk of developing cardiovascular disease.
- (b) Arthritis and musculoskeletal diseases are common indications requiring chronic NSAID therapy.
- (c) Aspirin and NSAIDs bind to different active sites on COX.
- (d) NSAIDs may prevent covalent binding of aspirin to COX-1.
- (e) Differences in aspirin interaction potential may be seen with various NSAIDs due to differences in the magnitude and duration of COX inhibition.