

Consensus Statement on the Diagnosis of Angioedema Mediated by Bradykinin.

Part I. Classification, Epidemiology, Pathophysiology, Genetics, Clinical Symptoms, and Diagnosis

Instructions to obtain 0.5 Continuing Medical Education Credits

These credits can be earned by reading the text and taking this CME examination online through the SEAIC web site at www.seaic.es.

The questions should be answered within 6 weeks from the publication of the examination.



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CME Items

- 1) In hereditary angioedema type III
 - a. Acute edema episodes can be precipitated by drugs containing estrogens
 - b. A mutation in the *F11* gene has been detected in a subgroup of patients
 - c. Males are predominantly affected
 - d. All the answers are true
 - e. Answers A and B are true
- 2) The main mediator in edema episodes induced by angiotensin-converting enzyme inhibitors is
 - a. C2-kinin
 - b. C1 inhibitor
 - c. Histamine
 - d. Leukotrienes
 - e. None of the answers is true
- 3) Kallikrein is a kininogenase that transforms high-molecular-weight kininogen into
 - a. C2-kinin
 - b. Plasmin
 - c. Bradykinin
 - d. Factor XII
 - e. None of the answers is true
- 4) The diagnosis of bradykinin-induced angioedema should be suspected in the following cases:
 - a. Every patient with recurrent peripheral edema plus urticaria
 - b. A patient with colicky abdominal pain, mild leukocytosis and ascites, and thickening of the intestinal wall in the abdominal ultrasonography.
 - c. A patient taking angiotensin-converting enzyme inhibitors with angioedema that does not respond to conventional treatment
 - d. All the answers are true
 - e. Answers B and C are true
- 5) Which of the following situations are known precipitating factors for acute edema attacks in patients with hereditary angioedema due to C1-inhibitor deficiency?
 - a. Respiratory infections
 - b. *Helicobacter pylori* infection
 - c. Trauma
 - d. Drugs containing estrogens
 - e. All the answers are true
- 6) Which is the main mediator of edema episodes in hereditary angioedema type III?
 - a. Bradykinin
 - b. Kallikrein
 - c. C2 kinin
 - d. C1 esterase inhibitor
 - e. None of the answers are true
- 7) Which of the following enzymes catabolize bradykinin and contribute to decreasing bradykinin levels?
 - a. Carboxypeptidase
 - b. Metallopeptidase
 - c. Aminopeptidase
 - d. Angiotensin-converting enzyme
 - e. All the answers are true
- 8) C1 esterase inhibitor is a regulator of contact system through inhibition of
 - a. C1r, C1s
 - b. Mannose-binding lectin-associated serine proteases
 - c. Factor XIIa, kallikrein
 - d. Factor XIa
 - e. Plasmin, tPA
- 9) Regarding hereditary angioedema without C1-INH deficiency (subtype HAE-FXII), which of the following is true?
 - a. Diagnosis is confirmed by an oral controlled challenge with estrogens
 - b. The presence of a family history confirms the diagnosis
 - c. Detection of factor XII mutation is the only test that confirms the diagnosis
 - d. A decrease in aminopeptidase activity confirms the diagnosis
 - e. Aminopeptidase P activity is increased in most cases
- 10) With regard to the clinical features of hereditary angioedema due to C1 inhibitor deficiency, which of the following sentences is true?
 - a. Clinical symptoms never occur in infancy
 - b. The illness should be ruled out if an abdominal attack develops without edema at other sites (peripheral or upper airway)
 - c. Abdominal attacks can progress to hypovolemic shock secondary to loss of fluid
 - d. Erythema marginatum is present in 80% of edema attacks in patients with hereditary angioedema and C1 inhibitor deficiency
 - e. None, all the answers are false