

## Hiccups exemplify the overlap between myocardial infarction and pulmonary embolism symptomatology



In paraphrase, the authors of a recent case report suggest that there should be a high index of suspicion for acute myocardial infarction (AMI) when a patient presents with hiccups in the context of old age, atypical presentation [of AMI], and multiple risk factors for acute coronary syndrome (ACS) [1]. It is also the case, however, that both ACS [2] and pulmonary embolism (PE) [3], respectively, are age related disorders [2,3]. The diagnostic difficulties generated by this phenomenon are compounded by the overlap in the clinical manifestations of the two disorders. This overlap is exemplified by the occurrence of hiccups, not only in the presence of AMI [1], but also in the presence of PE [4,5].

In all three reports [1,4,5], apart from hiccups, the most striking atypical feature was total absence of chest pain or dyspnoea. The two disorders, AMI and PE, also share other atypical presenting features such as abdominal pain, documented in 11% of 94 consecutive patients with AMI [6], and in 12% of 90 PE patients [7]. Syncope/presyncope is another atypical symptom, documented in 19.1% of 1763 patients with atypical AMI (all without chest pain) [8] vs occurrence of PE in 97(17.3%) of 560 patients hospitalised for a first episode of syncope [9]. In the latter report 24(24.7%) of the 97 PE patients “had no clinical manifestations of the diagnosis [of PE], including tachypnea, tachycardia, hypotension, or clinical signs or symptoms of deep vein thrombosis” [9].

In the emergency department context, the electrocardiogram is one of the most frequently used diagnostic modalities for expediting the distinction between AMI and PE, both in the typical and in the atypical context of the clinical presentation of either of these two disorders. This modality, however, cannot reliably distinguish between the two disorders, given the fact that AMI-related ST segment elevation [1] can also be simulated by the PE-related ST segment elevation (in anterior leads and also in inferior leads) which occurs in the total absence of coronary artery occlusion [10]. In the latter review of 34 PE cases (mean age 56.5), troponin was elevated in 15(78.9%) of the 19 patients in whom it was evaluated [1].

Given the fact that both AMI and PE are age-related, and have overlapping clinical, electrocardiographic, and laboratory stigmata, there is considerable scope for underdiagnosis as well as overdiagnosis of these two disorders in elderly patients, especially when they present atypically.

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## Hypoglycemia work up when medication is not a risk factor



In the reported study, although as many as 97 patients did not have medication as a risk factor for hypoglycaemia, no mention was made of the endocrinological “work-up” of those subjects [1]. Patients who belong to that category should be managed along the lines recommended in a recent review of the approach to spontaneous hypoglycaemia [2]. In that review the recommendation was that those who are seemingly well should undergo a diagnostic work up in which blood samples are taken to evaluate parameters over and above plasma glucose before treatment with either intravenous glucose or glucagon. The rationale is that “the directions of glucose, insulin, C-peptide, and proinsulin during a hypoglycemic event facilitate diagnosis” [2]. That diagnostic advantage will be lost if pretreatment samples of blood are not obtained during the episode of spontaneous hypoglycaemia. Consequently, a subsequent, artificially-induced stress test will be necessary to reproduce hypoglycaemia [2]. Even in patients initially perceived to have medication as the sole risk factor for hypoglycemia there should be a heightened index of suspicion for coexisting undiagnosed hypoadrenalism when recurring episodes of hypoglycaemia occur despite a reduction in insulin dosage [3,4]. The reason is that type 1 diabetes may coexist with primary hypoadrenalism when the two disorders have an autoimmune basis [3,4]. Apart from being hypoglycemic such patients may otherwise be seemingly well, as was the case in a patient who presented at her routine diabetic clinic review only complaining of recurring hypoglycaemia [3]. In another patient with the association of type 1 diabetes and Addison’s disease the opportunity to make a timely diagnosis of hypoadrenalism was missed when blood levels of cortisol were not evaluated during a hospital admission for hypoglycaemic coma. The diagnosis was only made 6 months later, using the short synacthen test (intramuscular synthetic adrenocorticotrophic hormone) [4]. In conclusion, pretreatment blood samples should be taken for parameters which are indicative of insulinoma in all apparently well patients where medication-related hypoglycaemia has been ruled out. Even among insulin treated patients those with type 1 diabetes should have blood samples taken for cortisol levels if they experience recurring episodes of hypoglycaemia in spite of a reduction in insulin dose. That should be the case even though the patient is seemingly well, given the fact that clinical features of Addison’s disease can be subtle, and not all patients are pigmented [5]. Using those strategies physicians in ED can optimise