

Postictal ECG: aneurysm or heart attack?

Sir,

Hodson and Poullis (vol 62(4), 2001, p. 242) report the case of a 63-year-old male who is a known arteriopath with a past history of cerebrovascular disease (CVA) and a left ventricular aneurysm, which is most likely secondary to a silent myocardial infarction in the past. Left ventricular aneurysm is well known to cause persistent electrocardiographic (ECG) changes resembling fully evolved anterior or anterolateral myocardial infarction, i.e. pathological Q wave, upward coving ST segment elevation and T wave inversion (Lindsay et al, 1984; Topol, 1998; Wagner, 2001).

In this case, the initial ECG showed slightly more marked changes compared with the ECG done 6 hours later and it was the result of ischaemia secondary to sympathetic overactivity in the postictal phase on the background of a left ventricular aneurysm. A subsequent ECG taken 6 hours later revealed persistent changes mimicking anterior myocardial infarction. It also showed ST segment depression and T wave inversion in lateral leads (I, aVL, V6) which are chronic and non-specific and are well known to occur in many non-cardiac conditions including postictal state as mentioned in the article.

There were no evolving changes in the subsequent ECG which indicate acute myocardial infarction. If the ECG changes suggestive of acute anterior myocardial infarction were caused by postictal state then the subsequent ECGs should have been normal. In

fact, the ECG changes are the result of an underlying left ventricular aneurysm and ischaemic heart disease rather than postictal state and therefore it is justified to claim that the postictal ECG changes mimicked acute myocardial infarction in the reported case.

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Lindsay J Jr, Dewey RC, Talesnick BS et al (1984) Relation of ST segment elevation after healing of acute myocardial infarction to the presence of left ventricular aneurysm. *Am J Cardiol* **54**: 84-6

Topol EJ, ed (1998) *Textbook of Cardiovascular Medicine*. 1st edn. Lippincott Williams & Wilkins, Philadelphia: 442

Wagner GS, ed (2001) *Marriott's Practical Electrocardiography*. 10th edn. Lippincott Williams & Wilkins, Philadelphia: 197

Sir,

Our report was of a case of ECG changes that occurred in the postictal period and mimicked an acute myocardial infarction in a patient who was subsequently demonstrated to have a left ventricular aneurysm. While the ECG changes associated with left ventricular aneurysms are well described, in this case it was the development of pronounced lateral T wave inversion that suggested an acute event.

While an increased heart rate can induce changes to the terminal deflection of the QRS complex, this would not account for the new lateral T wave changes. In the discussion we stated that the ECG changes 'may have been the result of a combination of autonomic arousal secondary to the epileptic fit ... on a background of a left ventricular aneurysm'. I agree that the subsequent ECGs should have returned to normal. However, we did not have any pre-admission ECGs to allow us to determine whether the T wave changes that caused us to suspect an acute event were in fact the ones present in the first ECG trace before 'resolving' to the pattern in the second ECG.

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Non-steroidal hip arthropathy

Sir,

With reference to the case report on non-steroidal hip arthropathy (vol 62(5), 2001, p. 308), I would like to mention another possible explanation for accelerated degenerative arthropathy in patients taking non-steroidal anti-inflammatory drugs (NSAIDs), apart from its analgesic effect with consequent increased loading.

NSAIDs have been shown to accelerate cartilage damage in animal studies as a result of their inhibitory effect on cartilage proteoglycan biosynthesis (Rainsford et al, 1999). Some studies suggested that salicylate administration may accelerate articular cartilage damage in animal models of osteoarthritis by suppressing proteoglycan synthesis by the chondrocyte (Brandt, 1987). These NSAID-related effects on chondrocyte metabolism appear to be much more profound in osteoarthritic (degenerating) cartilage than in normal cartilage because of the enhanced uptake of NSAIDs by the osteoarthritic cartilage. Furthermore depletion of matrix proteoglycans plays a major role in the increased vulnerability of chondrocytes to the effects of NSAIDs in osteoarthritic cartilage.

Therefore, despite the symptomatic improvement, cartilage degeneration could be accelerated by NSAIDs. Both patients and clinicians should be aware of the risks of cartilage damage from long-term NSAIDs apart from their other serious side effects.

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Brandt KD (1987) Effects of nonsteroidal anti-inflammatory drugs on chondrocyte metabolism in vitro and in vivo. *Am J Med* **83(5A)**: 29-34

Rainsford KD, Skerry TM, Chindemi P, Delaney K (1999) Effects of the NSAIDs meloxicam and indomethacin on cartilage proteoglycan synthesis and joint responses to calcium pyrophosphate crystals in dogs. *Vet Res Commun* **23(2)**: 101-13

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