Lateral ischemia abnormal ecg

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Is anterolateral ischemia dangerous. Abnormal t waves suggestive of lateral wall ischemia. What is inferior ischemia abnormal ecg. Consider lateral ischemia abnormal ecg. T wave abnormality consider lateral ischemia abnormal ecg. What is inferior ischemia ecg.

4 seconds ECG video by Stephen Smith on Vimeo. Here is your computer bed:Â ABNORMALITÁ MODERATA T-WAVE, CONSIDER ISCHEMIA LATERALEÂ ECG ABNORMALE You are working in triage where there are 30 patients who contend your attention and others who arrive every minute. Â The nursing assistant passes an ECG every few minutes, wishing to know if the patient needs emergency surgery. What is your immediate impression? The triage doctor saw the side inversions of the T wave and told the assistant that there was no STEMI and that the patient had to wait for the next available bed. Here is the EG again: There is a thin ST elevation in the cables III and aVF, with mutual ST depression in aVL This is a lower MI diagnosis There is a very low voltage in the cables of the limbs, so the ST segments are proportionally very abnormal. There are also quite well formed Q waves in II, III and aVF, so it could be subacute or even old (with persistent ST elevation -- aneurysm) Continued Case The patient was placed in ED on an unemergent basis. Here's the story: The patient has no previous heart anamnesi and presents to the DE with midstellar CP radiating to the left and started 14 hours before. Â It was intermittent, but it became constant 7 hours before. Â The patient also approves SOB. Â Nega N/V, fevers, shivers or cough. Â Smoke 5 cigarettes per day. He says he feels like something got stuck in his chest when he swallows something. The faculty doctor within the DE has seen better ECG and immediately recognized the lower acute (subacute) MI. He activated the cateterization lab. Another ECG was registered 39 minutes after the first, before leaving for the cateterization lab: There is a subtle evolution in the V3-V6 angiogram: 1. Trombotic occlusion of the marginal medium circumflex/second obtuse with weak side from left to left 2. 70% stenosis of the first diagonal 3. Diffused stenosis of 60-70% of mid-RCA, but with TIMI III flow over here is the post-cath ECG: The ST elevation in the lower cables is resolved. the reversal of the T wave is deeper, consistent with the reperfusion of Waves T Here is the profile of the troponine: It's a very big IM. The troponins have only a modest correlation with the size of the heart attack as it depends on reperfusion, but a Troponin The over 100 is very big. The tallest troponin I've ever seen was 500 ng/mL in a left main occlusion case. Â This was before the reperfusion,The highest my research colleague on the troponin, Fred Apple, has ever seen, is 1000! 4 months with cardiac arrest had an initial (and peak) troponin of 762 ng/mL! Here is the formal contrast echo the next day: Large inferolateral WMA left ventricular decrease performance-moderate. The left ventricular ejection fractionIt’s 35-40%. Anomaly of motion of the regional-anterolateral wall, acinetic. Regional wall movement anomaly-inferolateral, acinetic. Later, the triage doctor told me about the case. overwhelmed by patients in triage, pulled in many directions, and realizes he didn’t look at the whole ECG after seeing the inversions of the T-wave in the side ducts. The obvious anomalies have distracted him from the more subtle ones. Learning Points: 1. You don’t let the computer read fool you. You don’t even look until you’ve examined the ECG. 2. Do not let obvious anomalies prevent you from systematically examining the entire ECG. Large IMs often show only subtle (or not) ECG results Sorry, this site is only supported in a compatible HTML browser. The site may continue to work, but it may not be displayed properly. If you use Internet Explorer 6 or earlier, we recommend that you upgrade your browser to Intenet Explorer 8+ or try a compatible browser such as Firefox or Google Chrome. Research topics: General introduction to ST, T and U wave anomalies Basic concept: the specificity of ST-T and U wave anomalies is provided more by the clinical circumstances in which ECG changes occur than by the particular changes themselves. Therefore, the term, non-specific ST-T wave abnormalities, is often used when clinical data are not available to correlate with ECG results. This does not mean that ECG changes are irrelevant! It is the responsibility of the clinician treating the patient to determine the significance of the ECG results. Factors that influence the configuration of ST-T and U waves include: Intrinsic myocardial disease (e.g., myocarditis, ischemia, infarction, infiltrative or myopathic processes) Medications (e.g., digoxin, quinidine, tricyclics, and many others) Electrolytic abnormalities of potassium, magnesium Neurogenic factors (e.g., stroke, haemorrhage, trauma, tumour, etc.) Metabolic factors (e.g., hypoglycaemia, hyperventilation) Atrial repolarization (e.g., at rapid heart rates the atrial T-wave can pull down the beginning of the ST segment) Ventricular conduction abnormalities and rhythms originate in the ventricles “Secondary” changes in the ST-T wave (these are normal changes in the ST-T wave due exclusively to changes in the ventricular activation sequence): ST-T changes observed in the branching blocks of the beam (generally the polarity of the ST-T is opposed to major or terminal QRS deflection) ST-T changes observed in ST-T fascicular blocks T changes observed in non-specific IVCD ST-T changes observed in WPW pre-excitation ST-T changes observed in PVC ST-T, ventricular arrhythmias and ventricular beat ST-T Wave anormalities (changes in the ST-T wave independent of changes in ventricular activation and which may be the result of global or segmented pathological processes that effect ventricular repolarization): Drug effects (e.g. digoxin, quinidine, etc.) Electrolytics (e.g., hypokaliemia) Ischemia, heart attack, inflammation, etc. Neurogen effects (e.g., subarracnoid hemorrhage causing along QT) Differential Diagnosis of the ST segment Normal elevation variant «Early «EarlyExample #1: “Early Repolarization”: noticeable ST-segment take-off in V4-6 ducts; ST-elevation in V2-3 is generally visible in most normal ECGs; ST-elevation in V2-6 is concave upwards, another feature of this normal variant. Ischemic heart disease (usually convex upwards, or straightened) Acute transmural lesion â as in this acute anterior MI ST-tract elevation after acute MI suggests ventricular aneurysm ST-tract elevation can also be seen as a manifestation of angular Prinzmetal (variant) (coronary artery spasm) during exercise test suggests coronary artery stenosis or very narrow coronary artery spasm (transmural ischemia) Acute concave pericarditis towards high ST elevation in most wires except VR None a reciprocal depression of the ST segment (except the VR) Unlike “early depolarization”, the T waves are usually of small amplitude and the heart rate is increased. You may see depression of the PR segment, a manifestation of atrial damage Other causes: Left ventricular hypertrophy (in the right precordial ducts with large S waves) Left branch block (in the right precordial ducts with large S waves) Advanced hyperthermia (promising J waves or Osborne waves) Differential dia ST-segment depression Normal variants or artifacts: ST-segment pseudo-depression (baseline errant due to poor skin-electrode contact) J-junctional physiological depression with sinus tachycardia (most likely due to atrial repolarization) Hyperventilation ST-segment depression induced by ischemic heart disease ischemia (induced by exercise or during angina attacks â as illustrated below) ST segment depression is often characterized as “horizontal”, “upsloping”, or “downsloping” MI Non-wave Reciprocal changes in acute acute MI Q-wave (e.g., ST depression in I & aVL cables with lower acute MI) Non-ischaemic causes of ST depression RVH (right precordial cables) or LVH (left precordial cables, I, aVL) Digoxin effect on ECG Hypokalaemia Mitral valve prolapse (some cases) CNS disease Secondary ST segment changes with IV conduction abnormalities (e.g., RBBB, LBB, WPW, WPW, etc.) This page covers ECG signs of myocardial ischemia seen with non-ST elevation acute coronary syndromes (NSTEACS). Models of ST elevation and Q myocardial infarction are treated elsewhere: CMA occlusion, anterior STEMI, lateral STEMI, lower STEMI, right ventricular infarction, posterior infarction and Wellens syndrome Myocardial ischemia History Non-ST elevation acute coronary syndrome (NST) CAC S) includes two main entities: Non-ST elevation myocardial infarction (NSTEMI) Unstable angina pectoris (UAP). The between these two conditions is usually retrospective, based on the presence/absence of increase of heart enzymes at 8-12 hours from the beginning of chest pain. Both produce the same spectrum of alterations and symptoms of the EG and are managed identically in the emergency department. Myocardial ModelsTwo main ECG models associated with NSTEACS: ST segment depression The T-wave flattening or reverse While there are numerous conditions that can simulate myocardial ischemia (e.g. left ventricular hypertrophy, dygoxin effect), dynamic ST segment and T-wave changes (i.e. different from the ECG baseline or change over time) are strongly suggestive of myocardial ischemy. Other ECG models of ischaemia Morphology of depression ST Depression ST can be either upsloping, downsloping, or horizontal (see diagram below). Horizontal or downhill ST pressure ≥ 0.5 mm to J point ≥ 2 contiguous cables indicate myocardial ischemia (according to Task Force 2007 Criteria). The ST ≥ 1 mm depression is more specific and transmits a worse prognosis. The ST ≥ 2 mm in ≥ 3 leads is associated with a high probability of NSTEMI and provides a significant mortality (35% mortality at 30 days). The increase in ST depression is not specific to myocardial ischemia. Examples of ST segment morphology in myocardial ischemia Deployment of the ST segment The ST depression due to myocardial ischemia can be present in a variable number of cables and with variable morphology: ST depression due to subendocardial ischemia is generally widespread — typically present in I, II, V4-6 cables and a variable number of additional cables. A diffused ST depression model more ST elevation in aVR > 1 mm is suggestive of the main coronary left occlusion. The ST depression located in a particular territory (only lower or higher side conductors) is more likely to represent a reciprocal change due to STEMI. The corresponding ST elevation can be thin and difficult to see, but it must be sought. This concept of ST depression that fails to locate is further discussed on the blog Dr Smiths. Subendocardial ischemia diffused due to LMCA wave reverse Wave inversion T can be considered a test of myocardial ischemia if: At least 1 mm depthPresent in ≥ 2 continuous cables that have dominant R waves (R/S ratio > 1)Dynamic — not present on old ECG or change over time NB. T wave reverse is only significant if seen in leads with vertical QRS complexes (dominant R waves). Wave T reverse is a normal variant in the cables III, aVR and V1. Wide T wave reversal due to myocardial ischemia (more prominent in the side ducts) Wellens Syndrome Wellens syndrome is a model of inverted or biphasic T waves in V2-4 (in patients with ischemic chest pain) highly specific for the critical stenosis of the anterior left artery. Patients can be painless when the EG is taken and normally or minimally high heart enzymes; However, they are at extremely high risk for the front wallI'll be in the next 2-3 weeks. There are two models of T-wave anomaly in Wellens syndrome: Type A – Biphasic, with initial positivity and terminal negativity (25% of cases) Type B – Deeply and symmetrically reversed (75% of cases)T waves (Type A) Deeply inverted T waves (Type B) NB. There is confusion in the literature regarding the naming of T-wave models, with some authors using Type 1 (Type A) for two-phase T-waves and Type 2 (Type B) for inverted ones. It may be better to describe the T-wave pattern! The evolution of the Wellens T-wave can evolve over time from Type A to Type B (Smith et al). Evolution of T-wave reversal [A-D] after coronary reperfusion in STEMI reperfusion and in Wellens syndrome (NSTEMI). Modified by Smith et al. T-wave inversion evolution. The ECG in Acute MI, 2002 Nonspecific ST segment and T wave changes The following changes can occur with myocardial ischemia, but are relatively nonspecific: ST depression < 0.5 mmT wave inversion < 1 mmT wave flatteningUpsloping ST depression More Myocardial Ischaemia ECG Examples Examples Examples Examples Examples 1 Subendocardial Ischaemia: The most striking abnormality is diffuse ST depression, seen in conduits I, II, and V5-6. This is consistent with diffuse subendocardial ischemia. There is also some subtle ST elevation in V1-2 and aVR with small Q waves in V1-2, suggesting that the cause of diffuse ischemia is a proximal LAD occlusion. Example 2 Reciprocal variation: The most obvious abnormality is horizontal ST depression in III and aVF. This could be misinterpreted as inferior âischaemiaâ however, subendocardial âischemia does not localize. Regional ST depression should require checking the ECG for signs of reciprocal ST elevation. In this case there is thin ST elevation in aVL. This is a high side STEMI! Example 3 Wellens Syndrome: There are abnormal T waves in V1-4 â biphasic in V1-3 and inverted in V4. This pattern is known as Wellens Type A Syndrome and is highly specific for critical proximal LAD artery stenosis. Example 4a Dynamic ST depression in a patient with chest pain: diffuse ST depression (lead I, II, V5-6) indicates subendocardial ischaemia. The Q wave in Lead III with slightly elevated ST segment suggests the possibility of early lower STEMI. Example 4b ECG of the same patient after treatment with oxygen, nitrates, heparin and antiplatelets: ST changes are now resolved. Lower ST segments and Q waves are stable â this patient had a previous history of lower MI. Troponin was relieved, confirming that the initial ST depression was due to NSTEMI. Example 5 NSTEMI presenting with isolated U wave inversion: There are inverted U waves, most prominent in leads V5-6. This is a little known but very specific sign of myocardial ischemia â this patient had a 12-hour troponin of 4.0 ng/mL. Want to find out the full story behind this ECG?. References Advanced Reading Online Textbooks Mattu A, Tabas JA, Brady WJ. Electrocardiography in acute and critical care. 2e, 2019Brady WJ, Lipinski MJ et al. Electrocardiogram in clinical medicine. 1st, 2020Straus DG, DD Schocken. Marriott Practical Electrocardiography 13e, 2021 Hampton J. J. ECG Made Practical 7e, 2019Grauer K. ECG Pocket Brain (Expanded) 6e, 2014Brady WJ, Truwit JD. Decisioni critiche in Elettrocardiografia di Emergenza e Cura Acuta 1e, 2009Surawicz B, Knilans T. ChouÃ¢Â¦s Elettrocardiografia nella pratica clinica: Adulto e Pediatrico 6e, 2008Mattu A, Brady W. ECGÃ¢Â¡s for the Emergency Medical Part I 1e, 2003 e Parte II Chan TC. ECG in Medicina dâEmergenza e Cura Acuta 1e, 2004Smith SW. LâECG in acuto MI. 2002 [PDF] LITFL Ulteriori letture

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