ST ELEVATION IN A PATIENT WITH SMALL BOWEL OCCLUSION AND GASTRIC DISTENSION – WHAT IS THE MOST LIKELY EXPLANATION? A HYPOTHESIS GENERATING CASE REPORT

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Abstract

We report a case of ST segment elevation and PR depression in inferolateral leads in a patient with small bowel occlusion and gastric distension, that disappeared immediately after gastric evacuation. Contrary to prior reports, we believe that these ECG changes do not represent an intrinsic cardiac electrical abnormality, but are likely artefactual. We hypothesise that the accumulated air between the heart and the lower limb and left precordial electrodes result in a significant departure from the simplified assumptions of standard 12 lead ECG analysis (that the electrical activity of the heart can be described by an electrical dipole at a fixed location in an electrical homogeneous sphere) in such a way that the ECG filtering process will not compensate for this bias and will artificially create the ECG pattern described in this report.

Case report

An 86 year old woman presented to the Emergency Department with a 36h history of upper abdominal pain, nausea, and generally unwell. She had been unable to eat or drink in the last 36h. She denied chest pain, diaphoresis, or shortness of breath. She has had a right hemicolectomy for colonic cancer 2 years before presentation. Subsequent follow-ups revealed no recurrence of the disease.

On arrival, she appeared unwell. Blood pressure was 90/60 mmHg, heart rate was 80 bpm, oxygen saturation was 92% in ambient air. There were signs of moderate dehydration. Lung fields were clear. Cardiovascular examination was unremarkable. The abdomen was severely distended but not tender, with no abdominal sounds on auscultation.

Sequential ECGs on admission showed ST elevation in leads II, III, aVF, V4-V6, and small R waves in all precordial leads (Figure 1). The ST elevation had a "hammock" appearance, and there was no clear isoelectric TP interval. In all other leads except lead I, a ST mid-segment deflection was also present, in time with the "hammock" present in infero-lateral leads. ECGs performed with 3 different machines showed similar appearances (recorded with electrodes places in standard position and kept attached to the skin in the same places). Blood tests revealed increased white cell count, and moderate renal impairment. Troponin level was marginally elevated. Echocardiogram showed no obvious wall motion abnormalities. The ECG findings were considered disproportionate to the clinical presentation, and a diagnosis of ST-elevation myocardial infarction was considered highly unlikely. A chest and abdominal plain x-ray revealed severely distended small bowel and stomach (Figure 2A,B). A nasogastric tube was inserted and 1L of fecaloid fluid was removed. Repeated ECG immediately after stomach evacuation revealed complete normalization of the ST segment in

all leads (Figure 3). A CT scan confirmed small bowel occlusion (Figure 2C). The patient underwent surgical intervention and had an uneventful recovery.

Discussion

ST elevation has been previously described in cases of gastric distention and bowel occlusion (1-3). The proposed explanations for the ST elevation included coronary vasospasm in response to pain and/or abdominal distension, stress cardiomyopathy, myocardial ischaemia due to mechanical compression of the circumflex artery caused by gastric distension (2), and epicardial stretch caused by diaphragmatic contractions synchronous with cardiac systole (3). However, these explanations have major drawbacks. For example, circumflex artery compression by gastric distension is physiologically implausible, as it would be difficult to explain how increased intraabdominal pressure would result in localized intramediastinal / intrapericardial increase in pressure that would only collapse the circumflex artery without global cardiac compression and tamponade. Indeed, ST elevation in massive hiatus hernia compressing the heart may result in ST elevation in anterior leads, but the echocardiogram in these cases reveals cardiac compression between the hiatus hernia and the sternum (4). Stress cardiomyopathy and coronary vasospasm are also unlikely explanations, as the ECG normalizes promptly after removal of gastric distention, there are no further evolving changes in the ST segment / T wave during subsequent ECGs, there is little cardiac biomarker release, there are no acute regional wall motion abnormalities compatible with large inferolateral transmural left ventricular ischaemia, and there is almost universal lack of anginal pain. Diaphragmatic contractions synchronous with cardiac systole is a rare condition described in the 1950's, but in these patients the diaphragmatic contraction is clinically manifest, which was not described in patients with ST elevation and gastric distension.

All the above hypotheses on the origin of ST elevation in cases of gastric / intestinal obstruction rely on the fact that the ST-elevation reflects an intrinsic cardiac electrical abnormality. No prior reports raised the hypothesis that the ST elevation seen in these cases of gastric distension / intestinal occlusion may be artefactual, resulting from the complex interplay of electrical signal recording, filtering, and analysis by the ECG machine. Indeed, several ECG features are suggestive of an artefactual origin in our case, such as:

1. The ST segment and T waves are near normal in lead I only. This has similarities to the ECG recorded when a limb electrode is defective and causes changes in electrical impedance; in these cases artefactual ST deviation may occur in all leads except the limb lead not using that electrode (5);
2. There is no clear TP interval in leads exposing ST elevation. The ECG machines forces the TP interval as the zero reference, so processes affecting TP interval definition may result in PR and ST segment shift. Indeed, the "PR" interval was deviated in opposite direction to the ST segment in all leads where the ST segment is shifted (PR elevation with ST depression in aVR; PR depression with ST elevation in inferolateral leads). In these leads, there was also baseline drift (most notably in lead II) and there was poor definition of the end of the T wave, resulting in artefactual QT prolongation (the true end of the T wave can be inferred from leads I, V1-V3).

Why would these ECG changes be artefactual?

It is important to remember that ECG signal recording, processing, filtering, analysis and interpretation rests upon several theoretical simplifications and assumptions (6). Since the beginning of ECG recording more than 100 years ago, at the core of ECG analysis rests the assumption that the electrical activity of the heart can be described by an electrical dipole of variable magnitude and orientation at a fixed location (at the centre of the theoretical

Eindhoven triangle) in an electrical homogeneous sphere (to which the equidistant Eindhoven triangle is inscribed) (7). For the vast majority of cases this assumption introduces negligible bias, even though it is clear that the heart is not in the centre of the theoretical sphere in which the Eindhoven triangle is inscribed, the electrical activity of the heart cannot be ascribed to a single point in space as it is in a dipole model, the anatomical location of the heart in relation to the recording electrodes is highly variable between individuals, and the human body is not electrically homogeneous.

In addition, the role of signal processing and filter settings in ECG recording is also critical. Low-frequency filters and baseline drift suppression are used to cancel the influence of respiration and other low-frequency signals on the final ECG trace. The ECG machines display the PQRST signals in relation to the TP interval which is forced to zero amplitude. When abnormal electrical signals interfere with the filtering process, phase shifts and artefactual ST deviation may occur (5,8).

Interestingly, the appearance of the ECG in our case is virtually identical with ECG changes that have been described after deployment of a Minnesota tube for variceal bleeding (2), as well as in large left-sided pneumothorax (without mediastinal shift) (9). The common denominator in these cases is the additional presence of electrically inert air in close proximity to the heart. We suspect that this is enough to result in a significant departure from the simplifications and assumptions that form the core of ECG analysis and interpretation, most notably from the "electrically homogeneous sphere". The acute interpolation of electrically inert air between the heart and the leg and left precordial electrodes may explain the main baseline, PR interval and ST segment shift in inferolateral leads, but the relative normal appearance of lead I (which uses the right and left arm electrodes).

Computerized simulation models of ECG analysis may prove useful to better understand the ECG changes associated with similar conditions to this case (10), and may provide a unifying explanation of ECG traces described in seemingly different clinical conditions. Meanwhile, ECG changes associated with gastric distension / bowel occlusion should be recognized as STEMI mimicry in order to avoid unnecessary delays in surgical treatment.

References.

1. Hibbs J, Orlandi Q, Olivari MT, Dickey W, Sharkey SW. Giant J Waves and ST-Segment Elevation Associated With Acute Gastric Distension. Circulation. 2016;133:1132-4. doi: 10.1161/CIRCULATIONAHA.115.020607.
2. Birse DR. Inferolateral ST-segment elevation associated with a gastric variceal bleed and the use of a Minnesota tube. BMJ Case Rep. 2014;2014. pii: bcr2013202795. doi: 10.1136/bcr-2013-202795.
3. Littmann L, Monroe MH. The "spiked helmet" sign: a new electrocardiographic marker of critical illness and high risk of death. Mayo Clin Proc. 2011;86:1245-6. doi: 10.4065/mcp.2011.0647.
4. Hokamaki J, Kawano H, Miyamoto S, Sugiyama S, Fukushima R, Sakamoto T, Yoshimura M, Ogawa H. Dynamic electrocardiographic changes due to cardiac compression by a giant hiatal hernia. Intern Med. 2005;44:136-40. Doi: 10.2169/internalmedicine.44.136
5. Mărgulescu AD. Inferior ST-Elevation on the ECG: What Is the Diagnosis? Circulation. 2017;135:1177-1180. doi: 10.1161/CIRCULATIONAHA.116.027118
6. Kligfield P, Gettes LS, Bailey JJ, Childers R, Deal BJ, Hancock EW, van Herpen G, Kors JA, Macfarlane P, Mirvis DM, Pahlm O, Rautaharju P, Wagner GS. Recommendations for the Standardization and Interpretation of the Electrocardiogram Part I: The Electrocardiogram and Its Technology A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Circulation. 2007;115:1306-1324. doi: 10.1161/CIRCULATIONAHA.106.180200

7. Horan LG. Manifest orientation: the theoretical link between the anatomy of the heart and the clinical electrocardiogram. J Am Coll Cardiol. 1987;9:1049-56.
8. Tayler D, Hitt A, Jolley B, Sanders G, Chamberlain D. Phase shift in transmitted electrocardiograms: a cautionary tale of distorted signals. Resuscitation. 2009;80:859-62. doi: 10.1016/j.resuscitation.2009.04.026.
9. Price JW. Novel electrocardiographic changes associated with iatrogenic pneumothorax. Am J Crit Care 2006;15 415-419.
10. Ferrer A, Sebastián R, Sánchez-Quintana D, Rodríguez JF, Godoy EJ, Martínez L, Saiz J. Detailed Anatomical and Electrophysiological Models of Human Atria and Torso for the Simulation of Atrial Activation. PLoS One. 2015;10:e0141573. doi: 10.1371/journal.pone.0141573.

Figure legends.

Figure 1. ECG on admission displaying ST elevation in leads II, III, aVF, V4-V6, and small R waves in all precordial leads. The ST elevation had a "hammock" appearance, and there was no clear isoelectric TP interval. In all other leads except lead I, a ST mid-segment deflection was also present, in time with the "hammock" present in infero-lateral leads. The PR segment is deviated in opposite direction to the ST segment in all leads where the ST segment is shifted (PR elevation with ST depression in aVR; PR depression with ST elevation in infero-lateral leads). In these leads, there is baseline drift (most notably in lead II) and there is poor definition of the end of the T wave, resulting in artefactual QT prolongation only in those leads (the true end of the T wave can be inferred from leads I, V1-V3).

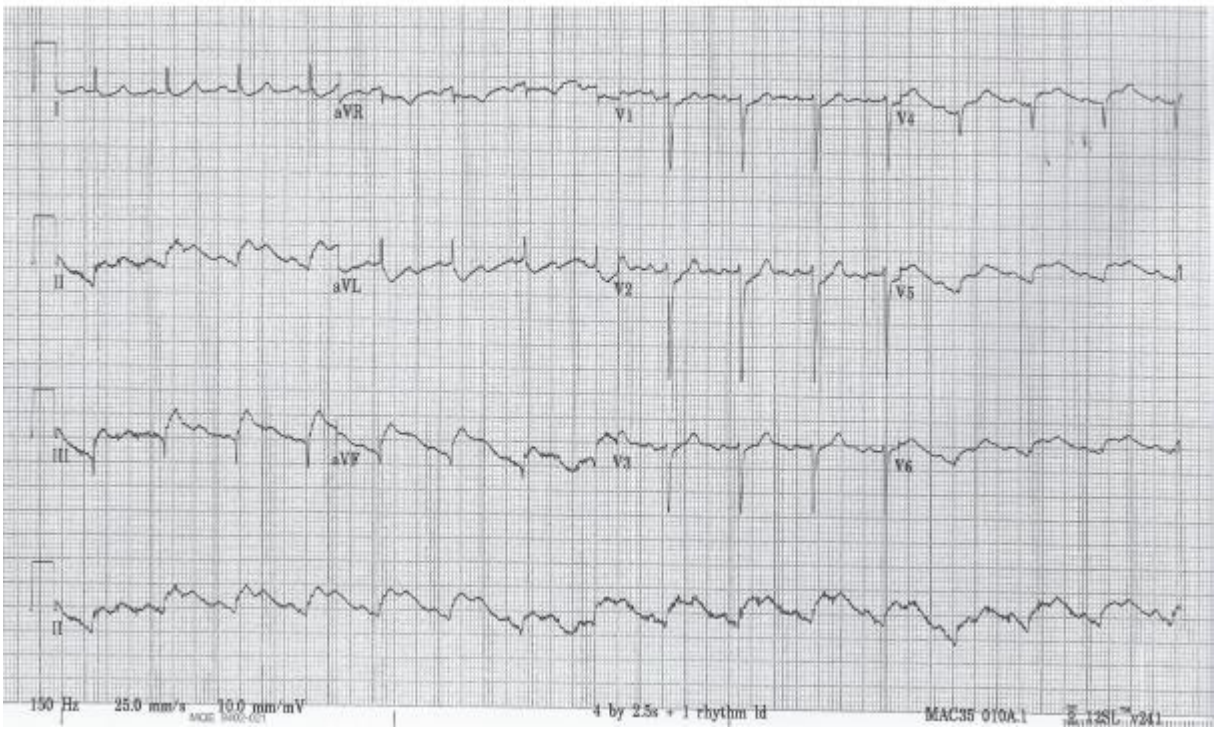

Figure 2. A. Chest X-ray on admission, showing distended stomach overlaying the cardiac silhouette. B. Abdominal plain X-ray (composed image based on 2 separate X-rays) showing severely distended stomach and small bowel loops. C. Abdominal CT scan confirming small bowel occlusion.

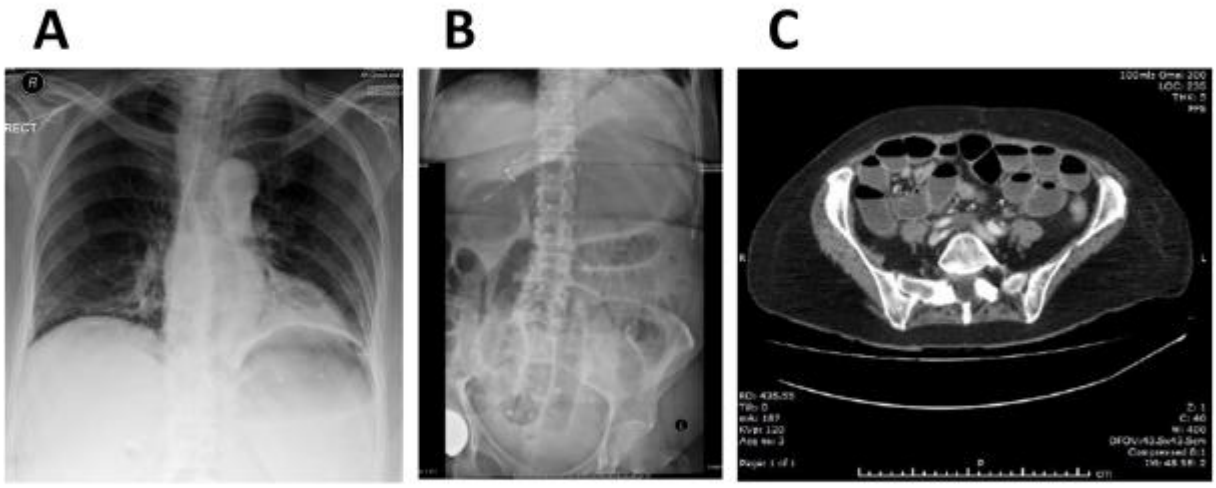

Figure 3. ECG recorded immediately after gastric tube insertion and stomach evacuation, showing complete resolution of PR and ST segment deviation, no baseline drift, and normal QT interval in all leads. Poor R wave transition persists in precordial leads.