

Insulin-overdose- A Potential Trigger of Ventricular Fibrillation with Takotsubo Cardiomyopathy?

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I have no conflict of interest.

Insulin-overdose- A Potential Trigger of Ventricular Fibrillation with Takotsubo Cardiomyopathy?

Author(s): Balga I

Abstract

A 46 years-old diabetic man lost consciousness because of an insulin-overdose (Glasgow Coma Scale 3, blood-glucose 1.5mmol/L). He regained consciousness after infusion of glucose 5% 100ml/10minutes (Glasgow Coma Scale 15, blood-glucose 3.8mmol/L). An additional infusion of glucose 5% 100ml/10minutes was given (blood-glucose 5.2mmol/L). 5minutes after the end of glucose infusion, he suddenly lost consciousness because of ventricular fibrillation and immediate cardiopulmonary resuscitation for 15minutes restored a sinus rhythm. The cardiac catheterization proved a takotsubo cardiomyopathy without significant coronary artery stenosis. A hypokalemia of 2.5mmol/L, presumably caused by the insulin-overdose, was present. Perhaps, insulin-overdose may trigger ventricular fibrillation with takotsubo cardiomyopathy, has not been reported in this context, but may surprise medical staff working in and out of hospital. Consequently, these patients need quick hospital admission, cardiovascular monitoring, echocardiography and cardiopulmonary resuscitation on standby until normalization of all dysrhythmic factors. More case reports or a study in the future are necessary to support this hypothesis.

Introduction

Takotsubo cardiomyopathy (TC) may be caused by hypoglycemia¹⁰⁻¹². Hypoglycemia may produce a proarrhythmic state¹³⁻¹⁵. VF with TC after insulin-overdose has not been reported in this context. The case of a diabetic patient, who survived ventricular fibrillation (VF) with TC after insulin-overdose is reported.

Case Report(s)

Prehospital case report

The ambulance team, an anesthetist and a paramedic, was called for an unconscious 46 years-old patient (weight 85kg, height 170cm) at a snack-bar. On arriving, the man lied on the floor. The observers told,

that the man came by car, suddenly lost consciousness before eating and was caught up without injuring himself. The first physical examination showed a normal spontaneous breathing (oxygen saturation 99% in air), a regular radial pulse 117/minute, a blood pressure 120/70mmHg, a Glasgow Coma Scale (GCS) 1/1/1 and isochorous, mid-dilated pupils reacting normally to light. The measured blood-glucose was 1.5mmol/L (27mg/dl). After fractional intravenous (iv) glucose 5% 100ml/10minutes, the man regained consciousness (GCS 15) and a blood-glucose of 3.8mmol/L (68mg/dl). Additional iv glucose 5% 100ml/10minutes was given, then a blood-glucose of 5.2mmol/L (94mg/dl) was measured. The history revealed a non-compliant diabetic (type II) patient. He had injected himself estimating 40U of short-acting instead of long-acting insulin one hour ago. The patient negated to suffer from a heart disease, angina pectoris or dyspnoea. The cardiac and pulmonary auscultation was assessed inconspicuously. The man denied to be hospitalized. The ambulance team tried to convince the man, that a hospitalization in his case was mandatory. 5minutes after the end of glucose-infusion, the man suddenly lost consciousness, stopped breathing and no carotid pulse was felt. The ambulance team started with cardiopulmonary resuscitation (CPR). The electrocardiogram (ECG) presented VF as first rhythm and one biphasic shock of 200J was fired (Illustration 1). Then pulseless electrical activity followed. Three times 1mg iv epinephrine was applied each 5minutes. Following differential diagnosis came into consideration: hypokalemia, hypoglycemia, acute myocardial infarction, pulmonary embolism¹. It was not possible to gain a blood drop for blood-glucose measurement during the CPR. Because of possible rebound hypoglycemia, 50ml iv glucose 5% was blindly administered. After 15 minutes of successful CPR, a tachycard sinus rhythm (Illustration 2) was restored and the patient reflexlessly intubated (capnography, initial CO₂ 6.2kPa). During intubation, regurgitation was seen. Blood-glucose was measured 16.6mmol/L (299mg/dl) after CPR and 10minutes later 11.5mmol/L (207mg/dl). The patient was brought without further complications and without the need of vasopressive drugs to the intensive care unit.

Hospital case report

All laboratory results of the patient are summarised in Illustration 3. The cardiac enzymes were in the normal range on admission. Laboratory data revealed a hypokalemia 2.5mmol/L and a slight hypocalcemia. Other laboratory data from blood, coagulation, liver and kidney were in the normal range. In the thoracic radiography a bilateral pulmonary edema was visible. The abnormal ECG, a ST-elevation in the wall-leads V2-V4 and III (Illustration 4) was the indication of an immediate cardiac catheterization to exclude a myocardial infarction. The coronary angiography uncovered akinesia in the anterior, apical and septal wall segments of the left ventricle (apical ballooning, Illustration 5 and 6), only a moderately diminished ejection fraction 52%, coronary artery sclerosis without a relevant coronary stenosis (a 30% stenosis in the left anterior descending artery and in the right coronary artery) and no pulmonary embolism or other cardiac pathologies (Illustration 3) could be found. With these clinical findings a TC was diagnosed. The patient suffered from following side diagnoses: arterial hypertension, strong nicotine abuse, depression, restless legs syndrome and psoriasis vulgaris. His daily drugs included: padma, pregabalin for knee pain, long-acting insulin (glargin, 40U) and fast-acting insulin (insulin aspart). A therapy with acetylsalicylic acid, lisopril, atorvastatin, torasis, magnesium as needed, potassium substitution was begun. The pulmonal edema disappeared rapidly and the patient was extubated the next morning (GCS 4/4/6). The cardiovascular support with low-dose norepinephrine could be stopped the next day. On day 3, the patient could be transferred to the ward with a telemetric surveillance. On day 4, a transthoracic echocardiography demonstrated a disorder of relaxation, a moderate diminished ventricular function (ejection fraction 47%), a hypokinesia apical and distal posterior. One week after CPR, no ischemic infarctions appeared in the head computertomography. All pathological diagnostic findings normalized without cardiovascular complication.

Discussion

The diabetic patient in a hypoglycemic comatose state ranks as a common daily prehospital emergency. The most part of hypoglycemic patients recover rapidly without complications after administration of iv glucose. Hypoglycemic coma, neurological disturbance and death from hypoglycemic encephalopathy counts among the well known complications from insulin-overdose. In this case, letal complications as

VF with TC were produced after insulin-overdose and glucose treatment. Prodrome symptoms and significant coronary artery stenosis lacked. As electrolyte disturbances, a hypokalemia and a slight hypocalcemia were present. Which pathological mechanisms in this case might have triggered off VF and TC ?

A hypoglycemic stress reaction resulted from insulin-overdose and might have contributed for developing a TC. TC may arise from stress, elevating plasma catecholamines, of different causes and hypoglycemic stress of other origin than insulin has been published. Otherwise, hypoglycemia induces epinephrine secretion augmenting the intracellular potassium-shift, prolongs the QT-time and intensifies a proarrhythmic state. Insulin shifts serum-potassium into the muscle and fatty cells. The intracellular potassium-shift produced a rapid fall in serum-potassium up to 2.5 mmol/L and was estimated as the main cause of VF. In addition, the glucose infusion as enough substratum may have favoured the common intracellular glucose-potassium-shift. TC as stunned heart may predispose to arrhythmias or to sudden cardiac arrest. The patient suffered from psoriasis vulgaris, a skin disease, that can be associated with hypoalbuminemia and hypocalcemia. Hypocalcemia is able to prolong QT-time and predispose to arrhythmias. Perhaps, the more triggering proarrhythmic factors are available, the higher the risk of VF. The speed and duration of proarrhythmic factors may also play a major role: maybe, the quicker they develop and the longer they act, the higher the risk of VF. Diabetic patients with a coronary artery disease have a greater risk to develop cardiac arrhythmias. As main mechanism in this case, insulin-overdose presumably produced several, rapid triggering proarrhythmic factors like stress, hypoglycemia, stunned heart and precipitated VF and TC. Several pathological mechanisms of TC has been discussed, but the exact origin of TC has not been revealed up today.

An other important problem was, that the patient rejected to be hospitalized. Some institutions accept the patient's desire with his signature and he will not be hospitalized. Every institution or hospital should confirm the rules, how to deal with such complex patients and which patients must be hospitalized, not to conflict with the law. A well clearing up for such patients and patience in certain cases are necessary. For paramedic people working without a medical doctor or without the chance of asking a medical doctor's advice, a gradation of patients account to illness in "must be hospitalized" or "must not be hospitalized", may be helpful.

Beside good instructions concerning the insulin therapy, especially a possible cardiac arrest after insulin-overdose and what to do, should be mentioned to diabetic patients and their relatives.

In this case, a diagnostic bedside test of serum-potassium and the therapy of hypokalemia are not available for out-of-hospital. Maybe, some medical prehospital institutions with long transport-times have the capacity. Therefore, a quick hospitalisation and monitored care for patients with insulin-overdose is indispensable. A defibrillator should be within one's grasp for medical stuff and in case of cardiac arrest, the rules of ACLS should be followed. What could we do anything else, if letal arrhythmias occur and hypokalemia as obvious cause is suspected, but neither diagnostic bedside test nor treatment of hypokalemia is disposable out-of-hospital? If indicated, we may facilitate intubation with succinylcholine, provided that no contraindications exist and the patient has reflexes. At this point, an anecdote by experience will be told: a 50-years-old man (GCS 15, angina pectoris) presented with a ventricular tachycardia (VT). The ambulance team decided first an intubation with succinylcholine because of aspiration risk, then to treat electrically the VT. The VT converted spontaneously into a sinus rhythm after muscular fasciculations have stopped and before laryngoscopy (Illustration 7). An extracellular serum-potassium shift due to succinylcholine was assumed. Everybody working out-of-hospital should know the drugs elevating serum-potassium he has at his disposal and these drugs may help in an emergency situation (other drug-example: magnesium).

Conclusion

Every patient with obvious insulin-overdose could potentially suffer from VF with TC. Prodrome symptoms and significant coronary artery stenosis may lack. Perhaps, the risk of letal arrhythmias augments with the number, speed and duration of proarrhythmic factors. Consecutively, these patients may need a quick hospital admission, a mandatory cardiovascular monitoring, bedside echocardiography, CPR on standby until all proarrhythmic causes are corrected. But this is only one case report and in the future, further similar case reports or studies are necessary to support this hypothesis.

Abbreviations(s)

TC: takotsubo cardiomyopathy

VF: ventricular fibrillation

GCS: Glasgow Coma Scale

iv: intravenous

CPR: cardiopulmonary resuscitation

ECG: electrocardiogram

VT: ventricular tachycardia

Authors contribution(s)

The author is responsible for the whole manuscript.

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Illustrations

Illustration 1

The ECG shows VF on the left side and the biphasic shock on the right side.

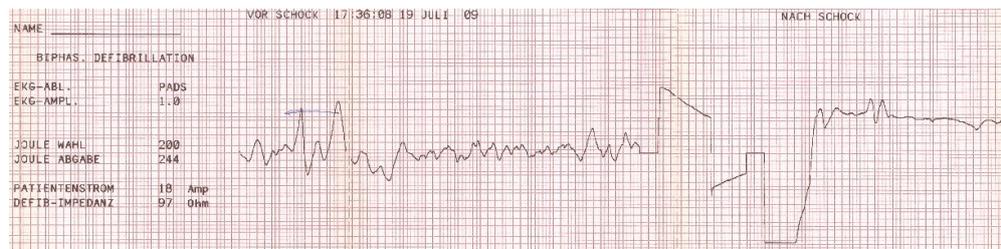


Illustration 2

Tachycard sinus rhythm was restored after the CPR and blood pressure 96/76mmHg measured.

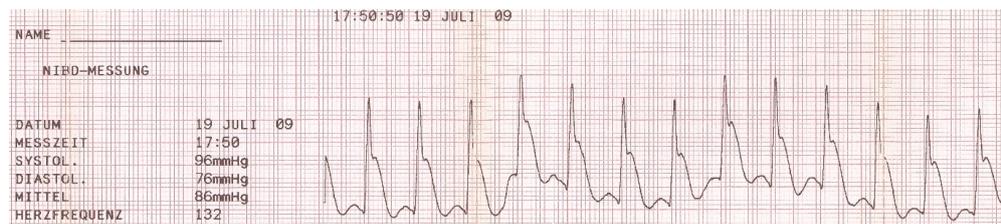


Illustration 3

Results from blood sample and cardiac catheterization on admission.

Laboratory data	normal range	patient's results
Troponin T	< 0.1 µg/L	0.02
Myoglobin	< 110 µg/L	116
Creatine kinase-MB	≤ 5 µg/L	5.0
Creatine kinase	< 195 U/L	85
B-type natriuretic peptide	< 33 ng/L	18
Potassium	3.4-4.5 mmol/L	2.5
Calcium total/ionized	2.15-2.55/1.15-1.35 mmol/L	2.07/0.98
Magnesium	0.70-1.05 mmol/L	1.03
Alcohol	< 0.1 ‰	< 0.08
thyroid-stimulating hormone	0.27-4.20 mU/L	2.13
Blood-glucose	3.9-6.4 mmol/L	15
C-reactive protein	< 5 mg/L	< 5
Body temperature	°C	34.2
pH	7.370-7.450	7.173 arterial

pCO ₂	4.70-6.10 kPa	8.39 arterial
pO ₂	9.50-13.90 kPa	10.4 arterial
Base excess	-2-+3 mmol/L	-7.3 arterial
Bicarbonate	21.0-26.0 mmol/L	18.5 arterial
Oxygen saturation	95.0-98.5 %	92 arterial
Oxygen saturation	70-75 %	69.1 venous
Lactate	0.5-1.6 mmol/L	5.7

Diagnostic values of the cardiac catheterization (patient intubated, ventilated)

Left ventricular volume	endsystolic/endiastolic	115/55 ml
Left ventricular pressure	endsystolic/endiastolic	127/16 mmHg
Aortic pressure	systolic/diastolic/mean	105/70/84 mmHg
Right ventricle	endsystolic/endiastolic	34/10 mmHg
Pulmonary artery pressure	systolic/diastolic/mean	30/17/22 mmHg
Right atrial pressure	mean	8 mmHg

Illustration 4

12-lead-ECG. ST-elevation in the wall-leads V2-4(>2mm) and III(>1mm) was detected.

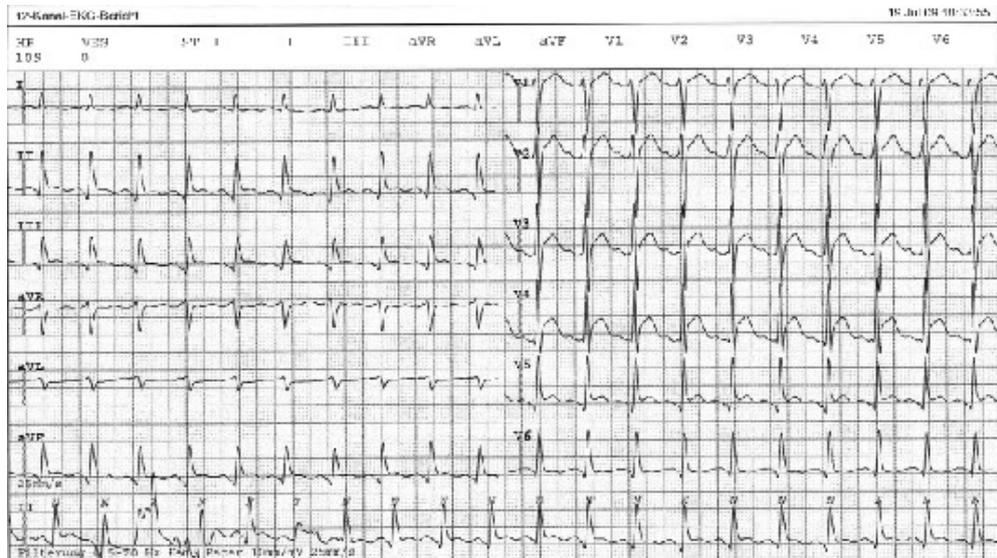


Illustration 5

Cardiac catheterization, left ventriculography. The illustration 5 shows the heart during diastole.

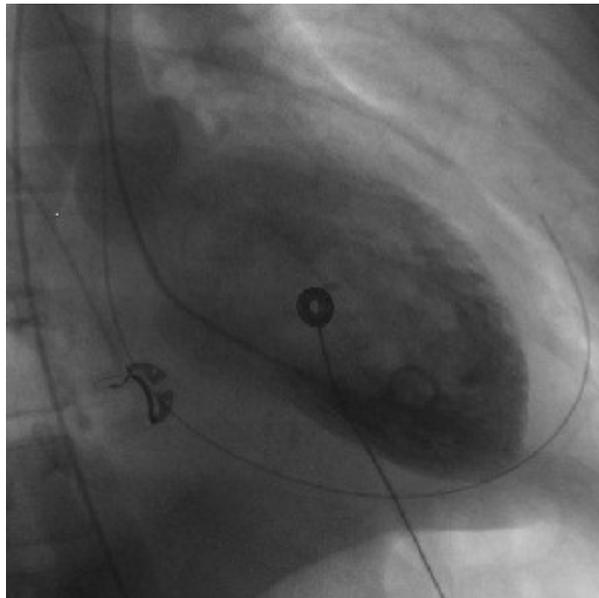


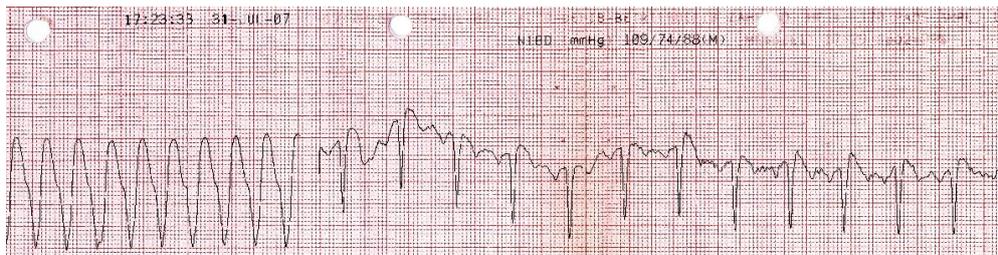
Illustration 6

Left ventriculography. The illustration 6 reveals an apical ballooning during systole, a TC.



Illustration 7

VT (left side) converted spontaneously into a sinus rhythm (right side) after iv succinylcholine.



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Reviews

Review 1

Review Title: Insulin-overdose, A Potential Trigger Of Ventricular Fibrillation With Takotsubo Cardiomyopathy ?

Posted by Dr. Yen-Wen Liu on 05 Dec 2010 08:26:40 AM GMT

1	Is the subject of the article within the scope of the subject category?	Partly
2	Are the interpretations / conclusions sound and justified by the data?	Partly
3	Is this a new and original contribution?	No
4	Does this paper exemplify an awareness of other research on the topic?	Yes
5	Are structure and length satisfactory?	Yes
6	Can you suggest brief additions or amendments or an introductory statement that will increase the value of this paper for an international audience?	Yes
7	Can you suggest any reductions in the paper, or deletions of parts?	No
8	Is the quality of the diction satisfactory?	Yes
9	Are the illustrations and tables necessary and acceptable?	No
10	Are the references adequate and are they all necessary?	Yes
11	Are the keywords and abstract or summary informative?	Yes

Rating: 7

Comment:

Comments:

1. 1. Dr. Balga reported that patients with insulin overdose could potentially suffer from ventricular fibrillation (Vf) with takotsubo cardiomyopathy. Actually, hypokalemia can induce Vf, especially in those with impaired LV function, including takotsubo cardiomyopathy, and hypokalemia can be resulted from insulin treatment. Therefore, it will be illogical to state insulin-overdose may trigger Vf. Although the author discussed this in the discussion, yet the title and abstract will make the readers confused. In my opinion, the title, abstract, and conclusion should be revised more clearly.
2. 2. The statement of "lethal complications as VF with TC were produced after insulin –overdose and glucose treatment" should be revised. It is not logically.
3. 3. Please submit the coronary angiography pictures and the followed-up cardiac function data at discharge. I can not find out any data showing the heart function was normal after discharge. Among patients with takotsubo cardiomyopathy, the cardiac function will be recovered without any sequela.
4. 4. Should every insulin-overdose patients need bedside echocardiography? I do not think so. Although they are at risk of hypokalemia and the potential fetal arrhythmia, yet in my opinion, it will be overstated that every such patient needs bedside echocardiography. In fact, many hypokalemia patients will not have Vf.
5. 5. There are some minor mistakes in this literature:
 - 1) Some spelling errors: eg. letal
 - 2) The reference numbers should be re-ordered. I can not find the "reference 1", but there were two "reference 2". The cited references in the session "introduction" were 10-12, and 13-15, but should they be "1-2", and "3-5"?
 - 3) 12-lead ECG DPI is not enough. By the current figure 4, ST seemed to be not elevated in lead III. Please re-submit a more clear figure.

Competing interests: nonw

Invited by the author to make a review on this article? : No

Experience and credentials in the specific area of science:

I stated my experience in the manuscript : Atypical presentation of "takotsubo cardiomyopathy" without ST segment elevation: a case report. Cases J. 2008;1:309.

Publications in the same or a related area of science: Yes

References: Liu YW, Chen JY, Tsai WC, Chen JH. Atypical presentation of "takotsubo cardiomyopathy" without ST segment elevation: a case report. *Cases J.* 2008;1:309.

How to cite: Liu Y. Insulin-overdose, A Potential Trigger Of Ventricular Fibrillation With Takotsubo Cardiomyopathy ? [Review of the article 'Insulin-overdose- A Potential Trigger of Ventricular Fibrillation with Takotsubo Cardiomyopathy? ' by]. *WebmedCentral* 1970;1(12):REVIEW_REF_NUM194

Review 2

Review Title: Insulin overdose, a potential trigger of ventricular fibrillation with takotsubo cardiomyopathy?

Posted by Dr. Ida Pernicova on 25 Oct 2010 06:39:55 PM GMT

1	Is the subject of the article within the scope of the subject category?	Partly
2	Are the interpretations / conclusions sound and justified by the data?	Partly
3	Is this a new and original contribution?	No
4	Does this paper exemplify an awareness of other research on the topic?	Yes
5	Are structure and length satisfactory?	Yes
6	Can you suggest brief additions or amendments or an introductory statement that will increase the value of this paper for an international audience?	Yes
7	Can you suggest any reductions in the paper, or deletions of parts?	No
8	Is the quality of the diction satisfactory?	Yes
9	Are the illustrations and tables necessary and acceptable?	No
10	Are the references adequate and are they all necessary?	Yes
11	Are the keywords and abstract or summary informative?	Yes

Rating: 0

Comment: This article reports a very interesting case of a successfully managed prehospital cardiac arrest following treatment for non-intentional insulin overdose in presence of possible takotsubo cardiomyopathy [TC]. The author comments rightly on the electrolyte disturbance associated with the insulin overdose and the need for an extended monitoring. The message regarding routine unavailability of stat potassium out-of-hospital and its potential implication is also highly relevant. I would perhaps advise more caution about some of the wording in the text. There is much debate regarding the diagnosis, aetiology and management of TC. Reports are on a rise. Despite it (and despite this particular case), most smoker diabetic males who suffer ventricular fibrillation (whether or not in association with hypoglycaemia) do not have TC. As such, thinking about TC by a paramedic and/or A&E team remains less important than considering a myocardial infarction and electrolyte disturbance. TC remains a diagnosis of exclusion. Has a possibility of phaeochromocytoma please been ruled out on the follow-up? This case illustrates well that severe insulin overdose can be deadly and may have an unpredicted development requiring monitoring. In summary, this is a highly interesting case-report with a clinically relevant message. P.S. It would be helpful if The WebmedCentral Team could help out the English-non native speakers contributors with the texts:)

Competing interests: none

Invited by the author to make a review on this article? : Yes

Experience and credentials in the specific area of science: Author of a review article on Takotsubo cardiomyopathy. Currently lecturer at the Division of Cardiovascular & Diabetes Research at the Leeds Institute of Genetics, Health and Therapeutics. University of Leeds. U.K.

Publications in the same or a related area of science: Yes

References: Pernicova I, Garg S, Bourantas CV, Alamgir F, Hoye A. Takotsubo cardiomyopathy: a review of the literature. *Angiology* 2010;61:166-73.

How to cite: Pernicova I. Insulin overdose, a potential trigger of ventricular fibrillation with takotsubo cardiomyopathy? [Review of the article 'Insulin-overdose- A Potential Trigger of Ventricular Fibrillation with Takotsubo Cardiomyopathy?' by J. WebmedCentral 1970;1(10):REVIEW_REF_NUM81

Review 3

Review Title: Insulin overdose, a potential trigger of ventricular fibrillation with takotsubo cardiomyopathy

Posted by Dr. Paolo E Angelini on 01 Oct 2010 11:10:11 PM GMT

1	Is the subject of the article within the scope of the subject category?	Partly
2	Are the interpretations / conclusions sound and justified by the data?	Partly
3	Is this a new and original contribution?	No
4	Does this paper exemplify an awareness of other research on the topic?	Yes
5	Are structure and length satisfactory?	Yes
6	Can you suggest brief additions or amendments or an introductory statement that will increase the value of this paper for an international audience?	Yes
7	Can you suggest any reductions in the paper, or deletions of parts?	No
8	Is the quality of the diction satisfactory?	Yes
9	Are the illustrations and tables necessary and acceptable?	No
10	Are the references adequate and are they all necessary?	Yes
11	Are the keywords and abstract or summary informative?	Yes

Rating: 0

Comment: This article reports a nice new example of a clinical scenario accompanied by Takotsubo acute and transient cardiomyopathy. Insulin overdose was apparently the precipitating event, but not the real cause. Of 1 000 000 cases of insulin overdose only an occasional case (1-5, maybe) will be complicated by a similar presentation, while the others do not, and they recover without complications. What is the difference? We believe that the baseline cause of Takotsubo syndrome is endothelial dysfunction: transient and severe. That triggers, in our theory, severe diffuse coronary spasm of most or some of the coronary tree, with acute ischemia. This would lead to persistent left ventricular dysfunction, usually but not only at the apex, of variable duration. (see reference of mine, above, for the "first ever" cases of experimental reproduction of Takotsubo in humans, broght about by acetylcholine challenge, in the cath lab, with echocardiographic demonstration of reversible apical ballooning). So, in many different baseline clinical states, with or without adrenergic challenge) a patient would develop a transient excessive spsticity of the coronay arteries, that leads to sustained ischemia and residual stunning of the affected myocardium. Insulin overdose would realize the same (triggering of takotsubo) only in predisposed patients, with endothelial dysfunction, that can only be demonstarted with acetylcholine testing. An this is the novel message of our paper, we are commenting.

Competing interests: No

Invited by the author to make a review on this article? : Yes

Experience and credentials in the specific area of science: Author of multiple articles on the Takotsubo syndrome. Involved in research on the pathophysioloy mechanism of Takotsubo. main article: Angelini P: Transient left ventricular apical ballooning: a unifying theory at the edge of Prinzmetal angina. J Cathet Cardiovasc Interv 2008;71:342-352

Publications in the same or a related area of science: Yes

References: Angelini P: Transient left ventricular apical ballooning: a unifying theory at the edge of Prinzmetal angina. J Cathet Cardiovasc Interv 2008;71:342-352

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