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CASE REPORT

Sudden death in anorexia nervosa: exploring the mechanism of death

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Summary

Introduction: Anorexia nervosa (AN) is a severe chronic psychiatric disorder with often underestimated prevalence. Patients suffering from anorexia nervosa can die from natural causes, such as severe heart problems, organ failure, or malnutrition, as well as from unnatural causes, such as suicide. However, the mechanism of death in anorexia nervosa is poorly understood.

Patient review: Our paper describes a case of sudden death in a 22-year-old woman suffering from anorexia nervosa. The autopsy showed severe malnutrition. Upon opening the abdominal cavity, extremely dilated stomach (without signs of wall necrosis or rupture) was noted. Biochemical findings showed that the vitreous chloride, sodium and potassium levels were markedly low, suggesting antemortem significant hypokalemia. There were no biochemical signs of severe hypoglycemia and dehydration. As cardiac disease is a significant cause of death in AN, aggravation caused by concurrent biochemical derangement must be taken into account. It is possible that gastric dilatation has a role in the occurrence of death, but this remains questionable.

Conclusion: In this case, the possible mechanism of death could have been a disruption of heart rhythm in an arrhythmogenic state due to an electrolyte imbalance or vagal activation related to gastric dilatation.

Key words: anorexia nervosa, gastric dilatation, arrhythmia, sudden death, autopsy

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INTRODUCTION

Anorexia nervosa (AN) is a severe chronic psychiatric disorder with often underestimated prevalence. The mortality rate of the patients suffering from AN from all causes of death is higher than that of the general population (1).

AN patients can die from natural causes, such as severe heart problems, organ failure, or malnutrition, as well as from unnatural causes, such as suicide. However, mechanism of death in AN is poorly understood (2, 3). Many patients with AN struggle with various digestive symptoms. In this case, we noticed gastric dilatation in autopsy. Since the first description of acute gastric dilatation in 1833, there has been an ample amount of cases documented in literature (4). Generally, the cause of gastric dilatation is difficult to pinpoint (5). This condition may be the result of mechanical and/or neurogenic factors and may appear in individuals with physical and mental impairment (AN). In addition to eating disorders, gastric dilatation has been reported to result from superior mesenteric artery syndrome, volvulus of hiatal hernias, trauma resuscitation, medications, air swallowing due to neuromuscular incoordination, diabetes mellitus, and other conditions (3,6,7,8,).

In this paper, we shall present a case of a young anorectic woman who died suddenly and discuss possible mechanisms of death.

CASE REPORT

A 22-year-old female was found dead in her bedroom bed. According to heteroanamnestic data, she had suffered from anorexia nervosa for five years. That day, according to her mother's statement, she had a meal for the first time in two days. She rapidly consumed porridge (approximately 400 ml) and one apple and she drank about 600 ml of tea. A few minutes later (without history of any symptoms) the woman lost consciousness and died. The autopsy was performed two days later.

The deceased was extremely underweight – she weighed only 23 kg and was 161 cm tall (BMI 8.9 kg/m2) **(Figure 1)**. There were no signs of external or internal injuries. The abdomen was not distended. Upon opening the abdominal cavity, extremely dilated stomach was noted **(Figure 2)**. It completely filled the abdominal and partially the pelvic cavity (extending from the xiphoid to the bladder). Its vertical diameter was around 40 cm, while its horizontal diameter amounted to around 30 cm. When the stomach was opened, it revealed around 1.2 l of undigested content. The gastric wall was of usual color, without necrosis. The gallbladder was highly distended. The intestines contained a small amount of semisolid fecal content. There were no obstructions, ascites, peritonitis or depositions of fibrin in the digestive tract. The diaphragm was not elevated. All organs were small in size, yet without obvious signs of dehydration.

Figure 1. The patient's body was poorly nourished with body mass of only 21 kg (BMI 8,6 kg/m^2).

Toxicological testing was negative for alcohol and drugs. Biochemical findings showed vitreous chloride, sodium and potassium in the following concentrations: 78 mmol/l, 116 mmol/l and 9.86 mmol/l, respectively. The vitreous glucose, and urea levels were 4.1 mmol/l and 28 mmol/l, respectively; no ketones were found.

Finally, the underlying cause of death was AN, and the possible mechanism of death could have been a disruption of heart rhythm due to an electrolyte imbalance or vagal activation related to gastric dilatation.

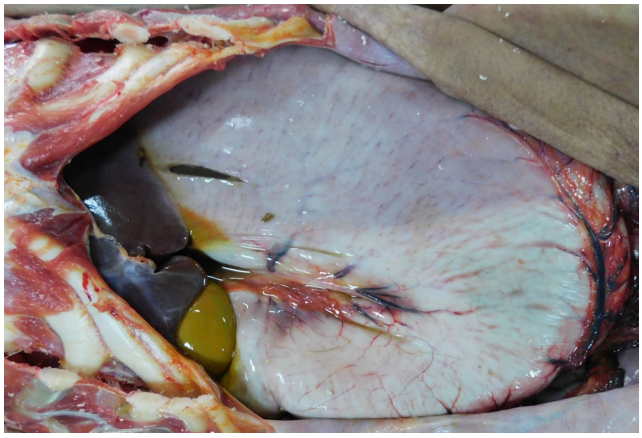

Figure 2. Abnormally distended stomach.

DISCUSSION

Long-term complications of AN include cardiovascular, gastrointestinal, endocrinological, hematological, psychiatric, fertility and pregnancy disorders (9). Recognized cardiovascular complications in these patients include numerous structural, hemodynamic, conduction, repolarization and peripheral vascular changes (9). Although ischemic heart disease and acute coronary syndrome are sparsely described in AN patients, definitive data in support of an increased risk of atherosclerotic vascular disease in anorexia nervosa population are lacking. Literature does suggest that anorexia nervosa could be associated with sudden death (8, 11). As precise etiology behind the increased risk of sudden cardiac death in anorexia nervosa remains unclear, the prolongation of the QT interval has been suggested as a potential cause because of its well-known association with torsade de pointes (12, 13). However, more recently it has been reported that anorexia nervosa might not be inherently associated with QT prolongation; rather, when it is present on electrocardiography, it should point towards secondary causes, such as electrolyte aberrations (hypokalemia, hypomagnesemia), or medications known to prolong the QT interval (9). Therefore, cardiac disease is a significant cause of death in AN, aggravated by concurrent biochemical derangement due to poor food intake and/or purging. That being said, arrhythmogenic complications due to severe hypokalemia that was proven in this case cannot be excluded and, may have, indeed, played a significant role in causing the young woman's death. Furthermore, autonomic dysfunction in AN has been in the focus of numerous studies. Some researchers showed that the cardiac vagal tone was higher in AN patients compared to the healthy control group (14). However, autonomic dysfunction and heart rhythm disorders cannot be proven in a postmortem examination.

In case of starvation, the immediate cause of death could be dehydration. Hypoglycemic coma is also reported in literature as one of the immediate causes of death (15). The vitreous levels of sodium, chloride and potassium, glucose level and ketones were measured and, even though it is well-known that the concentrations of many natural chemical substances in the corpse are rapidly distorted by postmortem autolysis (16), certain postmortem values of electrolytes might be indicative of their antemortem levels. The concentrations of sodium and chloride decrease after death, while the potassium level rises. Elevated vitreous levels of chloride (>135 mmol/l) and sodium (>155mmol/l) are indicative of antemortem dehydration, which is not the case in this paper. The level of potassium was low, even for a 2-day postmortem period, suggesting antemortem hypokalemia that, as we have previously stated, could have been the cause of arrhythmia. Vitreous glucose level was 4.1 mmol/l, while the analysis showed no ketones. In relation to hypoglycemia, vitreous glucose level below 1.4 mmol/l was taken by Sturner et all. to be an indication of low antemortem glucose level (16).

Anemia, leukopenia and thrombocytopenia are common findings in AN patients. In those patients, the diagnosis of infection could be delayed due to the absence of inflammatory and febrile response. In our case, we did not find any signs of infection (17).

In this case, the finding of gastric dilatation was very interesting. Acute gastric dilatation is recognized and described as gastrointestinal complication of AN (8). The symptoms of gastric dilatation can be vague, patients often present with emesis and gradual abdominal distention with pain, but in this case there were no data about prominent gastrointestinal symptomatology.

Acute gastric dilatation is defined as a condition in which the gastric wall rapidly loses its tension, and the gastric lumen is filled with gas and secretions, leading to rapid distention in the absence of structural obstruction in the stomach and/or duodenum (18). In the presented case, there was no structural obstruction in the stomach and/or duodenum. Literature describes deaths related to severe stomach dilatation followed by wall necrosis. In the described cases, high intragastric pressure overwhelms gastric venous pressure, producing ischemia, necrosis and finally the perforation of the gastric wall (8,11). In the presented case, there were no such complications. Fatalities following acute gastric dilatation without wall necrosis and rupture are rarely described and discussed, which is why the question is raised about the cause of death in those cases. Acute gastric dilatation accompanied by in-

creased intra-abdominal pressure and compression of the inferior vena cava may result in the congestion of bilateral lower limbs. In the case reported by Sincina, the inferior vena cava was compressed between dilated duodenum and vertebral bodies (19). Overextension of the gastric wall could induce peritoneal stimulation and vasovagal reflex, contributing to neurogenic shock (11, 19, 20). This is an example of distributive shock resulting from imbalance of sympathetic and parasympathetic regulation of vascular smooth muscles and heart rate. Also, acute gastric distension accompanied with increased intra-abdominal pressure could lead to diaphragmatic elevation and consequent limitation of respiratory movement. Space-restrictive disturbances in the thoracic cavity may also lead to the fatal outcome (6, 21). Therefore, all of the above suggests that acute gastric dilatation could be considered the cause of sudden death. However, the patient presented in this paper did not have any of the previously described complications. The role of gastric dilatation in the occurrence of death remains questionable.

The cause and mode of death in the presented case can be discussed. The absence of peritoneal irritation, ischemia and necrosis of the stomach wall, as well as fibrin deposits associated with liquid blood, suggest rapid death. It is possible that the previous heavy cachexia contributed to faster death; therefore, "there was not enough time" for the development of intestine necrosis and subsequent perforation and peritonitis.

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CONCLUSION

Finally, even though abnormalities in the heart rhythm cannot be proven postmortem, arrhythmogenic state caused by hypokalemia, could be considered as the mechanism of death in the presented case. Although the role of gastric dilatation in this case remains unclear, it could theoretically contribute to the occurrence of death. This possibility should be considered when determining the cause and manner of death in similar cases in the future.

Ethical approval

This article does not include any studies involving human participants or animals conducted by the authors.

Author contributions

All authors have evenly contributed to the conception of the work, interpretation of data and preparing the draft of the manuscript or interpretation of revised version of the manuscript.

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NAPRASNA SMRT U SLUČAJU ANOREKSIJE NERVOZE: RAZMATRANJE MEHANIZAMA UMIRANJA

Bogdanović Milenko[1], Popović Vesna[1], Atanasijević Tatjana[1], Radnić Bojana[1], Petrović Tijana[1], Milošević Veljko[1]

Sažetak

Uvod: Anoreksija nervoza je teško hronično psihijatrijsko oboljenje čija tačna učestalost nije poznata. Pacijenti sa anoreksijom nervozom mogu umreti od prirodnih uzroka smrti, poput kardiovaskularnih bolesti, popuštanja organa, pothranjenosti, ili usled nasilnih uzroka smrti, kao što je samoubistvo. U svakom slučaju, mehanizam umiranja kod anoreksije nervoze nije do kraja istražen.

Prikaz slučaja: Opisujemo slučaj iznenadne smrti mlade žene stare 22 godine koja je bolovala od anoreksije nervoze. Na obdukciji je pokazana teška pothranjenost. Neposredno po otvaranju trbušne duplje uočeno je da je želudac izrazito dilatiran (bez postojanja znakova nekroze ili rupture). Biohemijskom analizom uzorka tečnosti staklastog tela pokazano je da su vrednosti hlorida, natrijuma i kalijuma izrazito niske, ukazujući na značajnu antemortalnu hipokalijemiju. Biohemijski nisu pokazani znaci ozbiljnije hipoglikemije i dehidratacije. Imajući u vidu da su kardiovaskularne bolesti značajan uzrok smrti kod osoba sa anoreksijom nervozom, pogoršanje kardiovaskularne bolesti je moglo biti izazvano poremećajem elektrolita što se mora uzeti u razmatranje. Potencijalno je moguć uticaj dilatacije želuca na pogoršanje kardiovaskularne bolesti i smrtni ishod i kao takvog ga treba uzeti u razmatranje, ali ovo ostaje nejasno.

Zaključak: Mogući mehanizam umiranja u ovom slučaju bi mogao biti poremećaj srčanog ritma nastao usled proaritmogenog stanja izazvanog poremećajem elektrolita ili usled vagalne aktivacije izazvane dilatiranim želucem.

Ključne reči: anoreksija nervoza, dilatacija želuca, aritmija, iznenadna smrt, obdukcija

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