# Cardiopathica fantastica

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The drama and publicity associated with recent advances in cardiovascular diagnosis and therapy have led to an increased incidence of the cardiovascular presentation of Münchausen syndrome. Although Asher[1,2] in his original description pointed out three common presentations: abdominal pain (laparotomaphilia migrans), bleeding (haemorrhagica histrionica), and neurologic (neurologica diabolica), it was not long before a number of other forms were described including a variety of cardiovascular presentations, eventually termed cardiopathica fantastica.[3] The admission of two patients with this diagnosis to the coronary care unit of the University of Michigan within 1 year prompts the present report. The increasing incidence of this problem and the disruption that these patients cause in the coronary care unit demand an awareness of its existence, to avoid undue admission to the unit, or if this fails, to formulate appropriate methods of dealing with it.

## DEFINITION

Münchausen syndrome is currently considered to be a subset of factitious disorders.[4] The essential feature is the presentation of factitious physical symptoms, so convincing that the patient is able to gain and sustain multiple hospitalizations characterized by extensive medical work-ups, often including invasive procedures. It is often thought that this illness is under voluntary control, since the symptoms are deliberately produced. However, this behavior pattern cannot be controlled, and these individuals cannot refrain from their obviously self-destructive actions. This is in contrast to the malingering patient who voluntarily controls both his symptoms and behavior pattern for the achievement of a specific goal that can usually be identified.

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Münchausen syndrome patients present in a fashion so dramatic that physicians respond with intense and emergent medical care. Their histories include wide travels and detailed descriptions of grandiose experiences. Their early lives are often deprived and at the time of admission they seem to have no social support systems. Frequently, they are in trouble with the law. They have had multiple hospitalizations and are familiar with medical terminology and hospital routine. Yet, when pressed for details regarding their present lives and presenting problems, they become increasingly vague and their stories inconsistent. They are eager to submit to painful procedures. If their demands for extensive evaluations and medications are not met, they become management problems because of aggressive, demanding, unruly, and evasive behavior. When confronted or challenged, they indignantly leave against medical advice, only to move on to the next emergency room or hospital.

## EXEMPLARY PATIENTS

**Case No. 1.** P.F., a 52-year-old black male, was admitted to the coronary care unit with a 3-hour history of precordial chest pain, to rule out myocardial infarction. He stated that he had symptoms of angina pectoris and previous admissions to other hospitals to rule out myocardial infarction. During a previous admission for an episode of chest pain he underwent coronary arteriography. The coronary arteriogram revealed a proximal left anterior descending coronary lesion. The patient subsequently underwent coronary artery bypass graft surgery of the left anterior descending coronary artery. This was complicated by an anterior myocardial infarction and by an episode of cardiac arrest 1 week after surgery.

The patient stated that he was a nuclear physicist with the Nuclear Regulatory Commission and while investigating the Three-Mile Island nuclear accident had been exposed to a massive dose of radiation. He related that he was then hospitalized in Harrisburg where he had a cardiac arrest. He said he was successfully resuscitated and after myocardial infarction was ruled out he was transferred to the

National Institutes of Health in Bethesda, Md., for further evaluation. After discharge from the National Institutes of Health, he noted weight loss and anorexia but had not had any chest discomfort until the day of admission, when while investigating a "nuclear spill" in Michigan, he noted the occurrence of severe precordial chest pain.

On physical examination his blood pressure was 142/102 mm Hg supine, his pulse was 116 bpm, and respirations were 52/min. There was no jugular venous distention. Examination of the patient's chest revealed scattered rhonchi and a few expiratory wheezes but no rales. There was a well-healed median sternotomy scar. Examination of his heart revealed that the first heart sound was of normal quality, but it was felt that the pulmonic component of the second heart sound was increased. There was a fourth heart sound but no third heart sound. There were no cardiac murmurs or rubs. There was a well-healed surgical scar on the abdomen. Examination of the extremities failed to reveal any evidence of peripheral edema. Peripheral pulses were equal and present throughout. Neurologic examination was within normal limits. The patient's initial ECG revealed evidence of an anterior myocardial infarction with ST segment elevation in leads $V_1$ to $V_3$. His chest x-ray examination was within normal limits. Serial ECGs and serum enzymes including creatine kinase (CK) and lactate dehydrogenase (LDH) failed to show evidence of acute myocardial infarction. Chest x-ray examination confirmed the presence of a sternotomy with wire sutures in place.

While in the coronary care unit, the patient was noted to be evasive and doubts arose as to the veracity of his past history. The Nuclear Regulatory Commission was contacted and sent an investigator who determined that the patient was not a nuclear physicist and had not been associated with the agency. The patient was found to have had several previous hospitalizations for precordial chest pain requiring narcotic medication to control his symptoms during which the diagnosis of Münchausen syndrome was raised. The patient was subsequently transferred to a psychiatric service for further evaluation. Since his hospital discharge, a previous report of Münchausen syndrome including this patient was discovered in the psychiatric literature.[5] He has a massive Veterans Administration record for similar complaints and we are aware of at least one other non-VA Hospital admission for chest pain in Minneapolis.[6]

**Case No. 2.** R.M., a 29-year-old white male, presented to the emergency room of the University Hospital with a complaint of recurrent substernal chest pain exacerbated by stress and sometimes relieved by sublingual nitroglycerin. He stated that a recent evaluation at another hospital had revealed an abnormal ECG and that his father had died 2 months previously of a myocardial infarction. Risk factors included a 20-pack year history of smoking, current work-related and social stress, and a significant family history in that his father's first myocardial infarction had occurred at age 31. He had first had an episode of exertional precordial chest discomfort 4 years previously and more recently had had an episode of severe precordial chest discomfort described as heavy pressure with radiation to the left arm and jaw associated with nausea, shortness of breath, and diaphoresis. He had been seen at a local emergency room for these symptoms, been told that he had a normal ECG, and was refused admission. The present episode had begun 3 days earlier and was described as progressive tightening in the chest and dyspnea on exertion, culminating in his inability to walk 100 yards without shortness of breath. The chest discomfort and shortness of breath were relieved by sublingual nitroglycerin. Social history included the information that he was currently on parole and working 85 hours a week to prove his ability to stay out of jail. He had traveled in Mexico, Spain, Portugal, and Bermuda. An ECG taken in the emergency room was interpreted as showing questionable inferior myocardial ischemia. The patient was admitted to the coronary care unit to rule out myocardial infarction. An admission portable chest x-ray examination revealed questionable left ventricular prominence.

Initial physical examination revealed blood pressure 122/84 mm Hg, pulse of 86 bpm, and respirations 18 bpm. The chest was clear to auscultation and percussion. Examination of the heart revealed the heart sounds to be of normal quality. There was a fourth heart sound but no third heart sound. There was a grade I/VI systolic ejection murmur along the left sternal border without radiation to the neck. There were no diastolic murmurs. Examination of the extremities failed to reveal any evidence of peripheral edema. Peripheral pulses were equal and intact. An ECG revealed evidence of left ventricular hypertrophy by voltage criteria and nonspecific ST-T wave changes. A chest x-ray examination was within normal limits.

The patient's course in the coronary care unit was characterized by repeated episodes of precordial chest discomfort typical of angina pectoris, associated with shortness of breath and diaphoresis.

ECGs taken during the episodes of chest discomfort failed to reveal any significant changes suggestive of myocardial ischemia. The patient's episodes of precordial chest discomfort were only partially relieved by sublingual and intravenous nitroglycerin. They were eventually relieved by morphine sulfate. Although the patient had no ECG or serum enzyme evidence of myocardial ischemia or infarction, his repeated episodes of typical precordial chest discomfort with only partial response to intravenous nitroglycerin, his age, and his previous history were thought to justify urgent coronary arteriography. Because of the frequency and severity of his chest discomfort and the unresponsiveness to nitrate therapy, an intra-aortic balloon was inserted prior to the planned arteriography. After insertion of the intra-aortic balloon and onset of balloon augmentation, his chest pain was relieved.

Prior to the planned arteriography, it was discovered that the patient had had a recent cardiac catheterization at another hospital which revealed normal coronary arteries and left ventricular function. The patient was confronted with this information, which he had not previously revealed. He insisted that an error had been made and that the cardiac catheterization had been performed on someone else with a similar name. At the same time a nurse overheard a visitor refer to contacting the patient's father, who presumably had recently died of a myocardial infarction. A diagnosis of Münchausen syndrome was entertained, the intra-aortic balloon was removed, and the coronary angiography was cancelled. The patient did, however, undergo ergonovine administration during thallium-201 myocardial imaging to rule out the possibility of coronary artery spasm. After an initial test dose of 0.05 mg ergonovine maleate intravenously, the patient received a dose of 0.2 mg. There were no symptoms, ECG, or thallium-201 changes of myocardial ischemia associated with the dose of ergonovine.

Further information was obtained from his parole officer which revealed that he had had normal work-ups at other hospitals where he had been admitted with similar symptoms. At this point psychiatric consultation was obtained. Although the patient stuck to his story despite confrontation, he did admit to feeling depressed and overwhelmed and agreed to a transfer to the Psychiatric Unit of the hospital. While awaiting the results of serum enzymes that were in progress to definitively rule out myocardial infarction, the patient was noted to be somnolent and difficult to arouse. A toxicity screen revealed opiates, barbiturates, and benzodiazepines in his urine, and phenytoin and phenobarbital in his serum. The following day he was transferred to the Psychiatric Service.

## COMMENTS

The first patient ever reported with a cardiovascular presentation of Münchausen syndrome was a 42-year-old male with a history of an anterior myocardial infarction and atrial fibrillation.[7] He had multiple subsequent admissions for precordial chest discomfort suggestive of myocardial infarction and an ECG showing atrial fibrillation. Davidson[7] pointed out the difficulty in managing such patients, stating "this is a genuine case of Münchausen Syndrome in a man who had a myocardial infarction and now has paroxysmal fibrillation. Should he present himself at another hospital—as he almost certainly will—with a similar story, it would require a bold casualty officer to refuse him admission."[7] Spiro[8] in 1968, in an extensive review of Münchausen syndrome, found 38 patients, four of whom presented with cardiac symptoms. A later review in 1978[9] added 43 more cases of Münchausen syndrome, 10 presenting with various cardiac symptoms. As the public had become more knowledgeable and sophisticated about cardiovascular disease, so had the presentation of these patients. One patient flew all over the world diverting the airplanes he was on to unplanned stops, complaining of chest pain and a supposed history of six previous myocardial infarctions and two coronary artery bypass grafts.[10] Another patient had multiple hospital admissions because of a history of "mitral click syndrome,"[11] while yet another repeatedly went from hospital to hospital in New York City with symptoms of an arrhythmia, demanding that he be treated by cardioversion[12]; a fourth patient complained not only of precordial chest pain but also of having the Wolff-Parkinson-White syndrome.[13] Kounis[3] in 1979 observed that cardiac symptoms were increasingly accounting for the presentation of Münchausen syndrome and added the term cardiopathica fantastica to the list of subtypes of Münchausen syndrome. He correctly anticipated that, given the "wide variety of present cardiological problems, their complicated and attractive terminology, as well as the cardiological instrumentation, patients prone to Münchausen Syndrome would appear more frequently with cardiopathica fantastica."

Despite the increasing incidence and attention paid to factitious illness and Münchausen Syndrome in the medical literature,[14,15] it is surprising that this

presentation has not as yet been addressed in the cardiovascular literature. These patients are often admitted directly from emergency rooms to the coronary care unit and as in patient R.M., are often subjected to complex diagnostic and therapeutic procedures, some of which may carry significant risk. In view of the current enthusiasm for early coronary artery bypass graft surgery and/or streptokinase infusion in patients with suspected myocardial infarction, it is likely that further reports will appear in which patients with cardiopathica fantastica are subjected to these procedures as well. In these cases, when the appropriate diagnosis is finally made, the coronary care unit staff will have to face the fact that great resources of time, effort, and equipment have been fruitlessly expended. The resultant anger and frustration will inevitably get expressed to each other as well as to the patient, and may undermine the efficiency of the team's functioning.

**Cardiac management.** How can we avoid admitting these patients in the first place and, if they are admitted despite appropriate evaluation, how should the situation be handled? Even in retrospect, patient P.F. in the present report, along with several other previously reported cases presenting with chest pain and symptoms suggestive of acute myocardial infarction and ECG evidence of prior infarction and a previous coronary artery bypass graft, could not be denied admission to the coronary care unit for evaluation. Once admitted, however, caution should be exerted in proceeding with invasive procedures without objective evidence of myocardial ischemia. It is recognized that the ECG may be normal during episodes of myocardial ischemia. If ECG evidence of ischemia is not present in a patient complaining of precordial chest discomfort, the possibility of Münchausen syndrome should be considered and the patient's history should be reviewed for clues as to its likelihood. In retrospect, both of the patients in the present report had a history at least compatible with the diagnosis. Patient R.M. was in trouble with the law and patient P.F. claimed to have been a part of a grandiose event, the Three-Mile Island accident. In this situation objective evidence of myocardial ischemia should be sought. Myocardial ischemia can be diagnosed by the finding of a thallium-201 defect or by the finding of a transient wall motion abnormality on blood pool imaging or echocardiography, if these studies are carried out during or immediately after the episode of chest discomfort due to ischemia.[16] A possible alternative, such as in patient R.M. in the present report, would be to insert a Swan-Ganz catheter into the pulmonary artery and to have required demonstration of an increase in left ventricular filling pressure, which almost always accompanies significant episodes of myocardial ischemia. It must be admitted, however, that even with careful review of the history some of these patients, especially those with objective evidence of prior infarction or coronary artery bypass graft surgery, may not be detected and the apparent urgency of the situation is such that they will be subjected to invasive procedures.

**Psychiatric management.** Once discovered, these patients must be recognized as a psychiatric problem and a consultation-liaison psychiatrist must be called. The reaction that the patient generates is often so intense that the psychiatrist must address not only the problems of diagnosis and disposition for the patient, but also the turmoil among the members of the coronary care unit team that is left in his wake. This includes helping the staff to understand the nature of the syndrome and to handle their feelings of irritation at each other for allowing themselves to be duped.

Unfortunately, these patients prove as difficult and frustrating for the psychiatric team to deal with as for the cardiologist. Attempts to specify the underlying basis for this disorder have not as yet been successful. Patient histories and evaluations give the impression of severe personality disorders characterized by hostility, dependency, imposture, poor impulse control, and self-destructive acting out. In Münchausen syndrome all of these features come together in the uncontrollable urge to adopt the role of the patient and to interact with medical personnel. The difficulty in working up these patients, the short time they stay in any one hospital, the lack of follow-up, and the relatively small numbers that any one group can accumulate contribute to the lack of understanding of this problem. Nadelson[17] underscores the problem of working with these patients and questions the worth of the effort. He recommends that the most appropriate intervention would be to try to limit the damage the patient does to himself and to others and to help the staff by confirming the diagnosis and by encouraging confrontation. The poor past record of trying to interrupt the destructive and disruptive patterns of behavior in these patients may justify this attitude of helplessness. However, the cost of ignoring these patients is great and promises to be even greater unless more successful diagnostic and therapeutic strategies are developed.

**Conclusions.** In patients presenting with chest pain suggestive of myocardial ischemia cardiopathi-

ca fantastica should be added to the differential diagnosis if objective evidence of ischemia is not present and the history provides clues to the presence of Münchausen syndrome such as wide travels, detailed description of grandiose events and persons, lack of social support systems, or trouble with the law. Evidence of previous myocardial infarction or coronary artery bypass graft surgery does not exclude the diagnosis.

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# Intravenous nitroglycerin: Clinical pharmacology and therapeutic considerations

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Synthesized in 1846 by Sobrero, nitroglycerin (NTG) was first described as an antianginal agent in 1879, by Murrell,[1] who established the efficacy of sublingual NTG in relieving acute anginal episodes and in the prophylaxis of angina when used before exertion. NTG and its derivatives and congeners have become well established as primary therapeutic agents in managing patients with ischemic heart disease.[2] Despite its documented efficacy for patients with angina pectoris, the usefulness of NTG for many years was limited by its short duration of action when administered sublingually. Other routes of delivery have been devised to overcome this shortcoming. Topical ointment,[3] sustained-release oral preparations,[4] and even aerosol inhalation[5] have proved to be effective forms of NTG administration.

First reports of the intravenous administration of NTG appeared in the European literature in 1969.[6] The safety, ease, and predictability of intravenous administration of this drug have been firmly documented,[7] and its use has been extended to congestive heart failure (CHF),[8-10] the unstable angina syndrome,[11-13] CHF in acute myocardial infarction (AMI),[14,15] myocardial salvage in AMI,[16-19] hypertension during and after coronary revascularization,[20-23] and controlled hypertension during cerebrovascular surgery.[24] This report delineates the intravenous use of NTG in current clinical practice, with emphasis on pharmacology, physiology, and practical applications.

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