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Brief Clinical Reports

FAINTING, PANIC, AND MEDICAL CONDITIONS: A CASE STUDY

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Abstract. This case describes the cognitive-behavioural treatment of a 40-year-old woman with a 22-year history of infrequent fainting episodes. It highlights three features: 1) the potential importance of undiagnosed medical conditions in people presenting for anxiety disorders; 2) a somewhat unusual comorbid diagnosis of Agoraphobia Without History of Panic and Panic Disorder with Agoraphobia; and 3) the importance of modifying attitudes about performance and responsibility to reduce the chance of relapse.

Keywords: Panic, CBT, fainting, medical conditions.

Introduction

Fear of fainting is a common symptom in panic disorder that is usually associated with faintness, dizziness or light-headedness (e.g., Rapee, Sanderson, McCauley, & Di Nardo, 1992) although actual fainting is uncommon, presumably due to the elevation of blood pressure during panic attacks (e.g., Margraf, 1990). Cognitive therapy, targeting the misinterpretation of physical signs and symptoms, has proved to be an effective treatment for panic disorder (e.g., Clark, 1996). Anderson, Taylor and McLean (1996) reported a case where panic disorder was complicated by pre-existing vaso-vagal reactions (fainting) to blood-injury stimuli. The authors demonstrated that understanding and treating the functional relationship between the two conditions was essential in obtaining a successful resolution. The present uncontrolled case study illustrates the role of a history of fainting (not as a reaction to blood-injury stimuli) in later onset panic disorder.

Background

Ms N., a 40-year-old pay supervisor for a large public sector organization, was referred for CBT by a general practitioner for panic disorder and depression. Detailed history taking and

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behavioural analysis revealed a 22-year history of infrequent fainting episodes (approximately one per year), normally in the morning. They were not originally associated with anxiety and even over the previous few years there was little conscious fear of fainting. There had, however, been a slowly developing pattern of agoraphobic avoidance over 15 years, with increasing avoidance in the last 4 years, extending to the theatre, cinema, and restaurants. There had also been an increase in episodes of dizziness and depersonalization over the previous 3 years.

Ms N. reported numerous psychosocial stressors in the previous 5 years affecting both her immediate family (infertility, miscarriage, foster-care and adoption) and her extended family (terminal illness of father, near death of mother, suicide attempt by sister). She had also experienced pressure and uncertainty at work (obligation to study, work reorganization and cuts) and financial insecurity (husband lost job followed by periods of unemployment). She had an episode of pneumonia a year before referral. Ms N was a top performer at work, was held up as an example by her superior, and had been targeted for promotion to management. She felt pressured to perform and held down all her responsibilities, and had missed very few workdays despite the events in previous years. Her primary support network of family and friends provided little support due to her father's illness and major events in the lives of two sisters and two friends (two with mental illness, two in violent relationships, one divorce). Ms N. saw herself as always available to others and capable of always performing at a high level. She derived satisfaction from her roles at work and within her family and social network. She did not consider the burden placed on her by the numerous stressors as unusual or unacceptable. Rather, she felt personally inadequate when her resources were infrequently overextended.

Four months before referral she suffered two prolonged fainting episodes that necessitated brief hospitalization. Further medical investigations revealed no anomalies. Uncued panic attacks emerged in the following weeks, with increased avoidance followed by emergence of depressive symptoms in the last 3 months. A month's holiday did not allow any great recuperation and was marked by several intense panic attacks. She reported fatigue, loss of interest in activities, strong emotional reactions to small problems, episodes of weeping, difficulty in concentrating, loss of appetite, and sleep disturbance. On returning to work Ms N. was placed on sick leave after 2 days and was prescribed paroxetine (20 mg) 4 weeks before referral.

Diagnosis

A diagnosis was given of Agoraphobia Without History of Panic, based on the 15-year history of avoidance in response to fainting and later to dizziness, Panic Disorder with Agoraphobia, based on the emergence of panic over the previous 3 months, and Major Depressive Disorder (Single Episode Moderate Severity) (American Psychiatric Association, 1994). There was no Axis II disorder. All previous medical findings over the previous 20 years (including thorough investigation 5 months previously) had been negative except for a slight tendency to hypoglycaemia. Throughout the 22-year-history, fainting episodes had been explained as vaso-vagal shock. Although the patient regained consciousness after several minutes, several days of weakness and fatigue followed. There were numerous psychosocial and environmental factors related to the primary support group and social and occupational environments. Highest level of functioning (Global Assessment of Functioning

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Scale; American Psychiatric Association; 1994) in the previous year was estimated as 70; functioning at referral was estimated at 48.

Therapy

The therapy was based on the cognitive model of panic disorder (Clark, 1986, 1996). The model of panic was integrated within a broad cognitive-behavioural conceptualization of psychosocial stress where assumptions about roles and performance can lead to behaviour that may eventually lead to physical and mental exhaustion. In a state of such exhaustion, physical symptoms are likely to be generated. These symptoms may then be catastrophically misinterpreted as a function of the person's particular history and idiosyncratic beliefs about symptoms and their meaning. Therapy consisted of 1) distinguishing between actual fainting, which occurred with little fear, and panic attacks where fear of fainting was a dominant feature; 2) explanation of the CB model of panic attacks and the general incompatibility between increased anxiety and fainting; 3) graded exposure to previously avoided situations while refraining from safety-seeking behaviours; 4) reframing the emergence of panic and depression in terms of mental and physical exhaustion in response to psychosocial stressors and her particular style of meeting them; 5) modifying attitudes and behaviour to feeling over-responsible toward others and setting personal limits; and 6) modifying perfectionist attitudes and behaviour about performance at work. In all there were 12 sessions of therapy over a 7-month period, with the majority (8) in the first 10 weeks.

Ms N. responded over 3 months to the combined CBT and pharmacotherapy with remission of depressive symptoms, gradual gain of energy, increasing confidence and mobility around the city, and ability to enjoy activities in previously avoided places (shopping centres, theatres, restaurants, etc.). She also learned to place limits on demands by family and friends, although this was not acquired easily.

There were three episodes of nocturnal fainting (i.e., awakening and then losing consciousness while lying down) over a 6-month period that overlapped with the period of panic symptoms and the first 3 months of therapy. The patient was then referred by her doctor to a neurologist. After extensive nocturnal monitoring a probable diagnosis of epilepsy was given, although findings were not clear-cut. It remains unknown whether the earlier fainting episodes were due to vagal shock or were epileptic in origin; however, there is strong suspicion of the latter. Following an additional nocturnal attack, anti-convulsive medication was introduced (Epival). A progressive return to work was planned. An additional nocturnal episode occurred after her first 2-day week. The consequences of this episode (fatigue, weakness, and dizziness) resulted in an incapacity to stay at work in the second week despite valiant attempts to do so. Ms N. reported a loss of confidence, transient depressive symptoms, and re-emergence of agoraphobic avoidance. However, these remitted rapidly with self-directed efforts to confront feared situations.

Ms N. returned to work with the resolve to react appropriately if any additional epileptic episodes occurred by taking sufficient time off for recovery. Recognition of her medical condition relieved some of the pressure she felt in the workplace as accommodations were made to reduce pressure. Reduction of paroxetine is planned to begin after one year at the therapeutic dose. There has been one additional nocturnal epileptic episode but this did not lead to loss of consciousness. The depression is in full remission; there have been no panic attacks nor fear of panic attacks. Of all symptoms at referral only some avoidance remains,

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related to travelling and motorways, but the patient plans to confront these targets over the next 4 months. Periods of fatigue remain a problem but the frequency and intensity of the episodes is decreasing. Current Global Assessment of Functioning is estimated at 80.

Comment

Although this case cannot determine the relative contributions of CBT and medication, it does however highlight three features. First, the role of medical conditions in people presenting for anxiety disorders must not be underestimated. Second, this case illustrates a somewhat unusual comorbid diagnosis of Agoraphobia Without History of Panic (developing slowly from 20 years of infrequent fainting), and Panic Disorder with Agoraphobia (resulting from full-blown panic attacks in the context of physical and mental exhaustion). Third, it was important to modify attitudes about performance and responsibility to reduce ongoing stress and fatigue. It is believed that these interventions are necessary to reduce the possibility of relapse. There are several possible pathways to relapse. For example, relapse could occur through the renewed emergence of physical symptoms such as dizziness or depersonalization due to exhaustion. In a vulnerable emotional state associated with exhaustion there is greater likelihood of misinterpretation, loss of confidence, and increased avoidance. Alternatively, exhaustion could lead to increased frequency of epileptic episodes. Even in the absence of catastrophic misinterpretation, avoidance could then increase through reality-based fears of losing consciousness although the subjective probability and gravity of fainting may be inflated. Although her interpretation of the physical symptoms is currently quite adequate and avoidance is minimal, it is not known whether increased frequency or intensity of physical symptoms or recurrent epileptic episodes would lead to more safety-seeking behaviour. The client must continue to live with the possibility of further epileptic episodes if medication is unsuccessful in completely controlling symptoms.

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