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How often is the finding of Hiatal Hernias in patients with established diagnosis of panic disorder and its interpretation?

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Abstract: Anxiety disorders are the most common of all psychiatric illnesses and result in considerable functional impairment and distress. Working with patients who have an anxiety disorder can be highly gratifying for the informed psychiatrist, because these patients, who are in considerable distress, often respond to proper treatment and return to a high level of functioning. A) What is Panic disorder? B) What is Hiatal Hernia? C) Biological theories of panic disorder prominent in the psychiatric literature. D) Neurocircuity of fear. E) Symptoms of Anxiety Disorder Due To Another Medical Condition. F) Methodology. G) Results and Conclusion.
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Keywords: Panic disorder; Hiatal Hernia; Biological theories; psychiatric Neurocircuity

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Introduction

Anxiety disorders are the most common of all psychiatric illnesses and result in considerable functional impairment and distress. Working with patients who have an anxiety disorder can be highly gratifying for the informed psychiatrist, because these patients, who are in considerable distress, often respond to proper treatment and return to a high level of functioning.

One of the major anxiety disorders is panic disorder.

DSM-4 clarified several issues regarding the diagnosis and differential diagnosis that remained obscure earlier.

Panic attacks are well known to occur not only in panic disorder but also in other anxiety disorders (e.g., specific phobia, social phobia, PTSD). In these other disorders, panic attacks are situationally bound or cued -that is they occur exclusively within the context of the feared situation. DSM-4 clarified this confusion by explicitly presenting the definition of panic attacks independently of panic disorder.

While other psychiatric disorders that involve pathological anxiety can make the differential diagnosis of panic disorder difficult, other medical conditions that mimic anxiety are usually easily ruled out; if, most importantly are kept in mind.

Medical conditions that are to be considered are: Hyper and hypothyroidism, mitral valve prolapse, hyperparathyrodism, cardiac arrhythmia, coronary heart disease, pheochromocytoma, hypoglycemia, true vertigo.

In this study, the accent is made on establishing the relationship between the existence of hiatal hernias and the occurrence of panic attacks and/or panic disorder, in a sample of 22 patients. The premises is that this condition occurs more frequently in patients with panic disorder.

Key words during the research:

Panic disorder, panic attacks, anxiety disorders, hiatal hernias.

A) What is panic disorder?

Patients with panic disorder frequently present to the emergency department (ED) with chest pain or dyspnoea, fearing that they are dying of myocardial infarction. They commonly report a sudden unexpected and spontaneous onset of fear or discomfort, typically reaching a peak within 10 minutes. DSM-5 criteria for panic disorder include the experiencing of recurrent panic attacks, with 1 or more attacks followed by at least 1 month of fear of another panic attack or significant maladaptive behaviour related to the attacks. A panic attack is an abrupt period of intense fear or discomfort accompanied by 4 or more of the following 13 systemic symptoms:

- Palpitations, pounding heart, or accelerated heart rate.
- Sweating
- Trembling or shaking
- Shortness of breath or feeling of smothering
- Feelings of choking
- Chest pain or discomfort
- Nausea or abdominal distress
- Feeling dizzy, unsteady, lightheaded, or faint
- Chills or heat sensations
- paraesthesia (i.e., numbness or tingling sensations)
- Derealisation (i.e., feeling of unreality) or depersonalization (i.e., being detached from oneself)
- Fear of losing control or going crazy
- Fear of dying
- During the episode, patients have the urge to flee or escape and have a sense of impending doom (as though they are dying from a heart attack or suffocation). Other symptoms may include headache, cold hands, diarrhoea, insomnia, fatigue, intrusive thoughts, and ruminations.

Patients with panic disorder have recurring episodes of panic, with the fear of recurrent attack resulting in significant behavioural changes (e.g., avoiding situations or locations) and worry about the implications of the attack or its consequences (e.g., losing control, going crazy, dying).

Panic disorder may result in changes in personality traits, characterized by the patient becoming more passive, dependent, or withdrawn.

It is important to assess precipitating events, suicidal ideation or plan, phobias, agoraphobia, and obsessive-compulsive behaviour. Exclude involvement of alcohol, illicit drugs (e.g., cocaine, amphetamine, phencyclidine, amyl nitrate, lysergic acid diethylamide [LSD], Yohimbine, 3,4-methylenedioxymethamphetamine [MDMA, or ecstasy]), cannabis, and medications (e.g., caffeine, theophylline, sympathomimetics, anticholinergics).

Differential diagnosis of Panic disorder:

- Consider symptomatology of other medical disorders, which may manifest with anxiety as a primary symptom.
- Angina and myocardial infarction (e.g., dyspnoea, chest pain, palpitations, diaphoresis)
- Cardiac dysrhythmias (e.g., palpitations, dyspnoea, syncope)
- Mitral valve prolapse
- Pulmonary embolus (e.g., dyspnoea, hyperpnoea, chest pain)
- Asthma (e.g., dyspnoea, wheezing)
- Hyperthyroidism (e.g., palpitations, diaphoresis, tachycardia, heat intolerance)
- Hypoglycaemia
- Pheochromocytoma (e.g., headache, diaphoresis, hypertension)
- Hyperparathyroidism (e.g., muscle cramps, paraesthesias)
- Transient ischemic attacks (TIAs)
- Seizure disorders

B) What is Hiatal Hernia?:

Hiatal Hernias are characterised by a protrusion of the stomach into thoracic cavity through the widening if the right crus of the diaphgram. Hiatal hernias (H.H.) have been reported to affect anywhere from 10% to 50% of the population.

There are 4 types of oesophageal H.H.:

- Sliding (type I Hernia) accounts for more than 85% of diaphragmatic hernias and the most common symptom is the co-existence of gastro-oesophageal reflux, that may lead eventually to ulceration with resulting haemorrhage. However, it still remains uncertain whether HHs are a cause of reflux or a result of it.
- Paraesophageal (type II Hernias) incidence varies between 3.5 %and 5% of all operated hiatal hernias. These patients will rarely experience reflux symptoms. However they may complain of fullness after meals, palpitations, pain, shortness of breath, dysphagia, regurgitation and may experience peptic ulcers of the stomach. Ingestion of food provides a bolus that entered.
- Mixed hiatal hernias (type III) are a combination of sliding and paraesophageal hernias and therefore include the symptoms of both.
- Hiatal Hernias (type IV). Giant paraesophageal hernias that include more than half of the stomach herniated through the hiatus.

The diagnosis of hiatal hernias is made by barium swallow contrast radiography. Hiatal hernias can appear as a shadow in the posterior mediastinum.

The herniated stomach, causing distension. This distension results in pain which has been described as "crushing" in nature and can be confused with Angina.

C) Biological theories of panic disorder prominent in the psychiatric literature.

1. The sympathetic system:

For many years the possibility that panic attacks are manifestations of massive discharge from the beta-adrenergic nervous system has been considered.

The beta-adrenergic hypothesis received further support from studies claiming that beta-adrenergic blocking drugs, such as Propronolol, have an ameliorative effect on panic attacks and anxiety. However, only modest antianxiety effects can actually be demonstrated.

The Locus Coeruleus has also been implicated in the pathogenesis of panic attacks. This nucleus is

located in the pons and contains more than 50% of all noradrenergic neurons in the entire CNS. Drugs known to be capable of increasing Locus Coeruleus discharge in animals are anxiogenic, whereas many drugs that curtail Locus Coeruleus firing and decrease central noradrenergic turnover are antianxiety agents. However the multiple experiments using a diversity of pharmaceutical agents suggested that the Locus Coeruleus my be involved in arousal and response to novel stimuli rather than in anxiety (Aston-John's et al1984).

2. The panicogen sodium lactate and carbon dioxide hypersensitivity theory:

It has been replicated that the infusion of sodium lactate provokes a panic attack in most patients with panic disorder but not in healthy controls. Also, in a comparable rate to sodium lactate, CO2 inhalation provokes panic attacks in patients with panic disorder. (Gorman et all 1989).

CO2 constitutes the common metabolic product of both lactate and bicarbonate. This CO2 selectively crosses the blood -brain barrier and produces transient cerebral hypercapnia. This hypercapnia then sets off the brain- stem chemoreceptors leading to hyperventilation and panic. Thus, a "false suffocation alarm" theory has been formulated (D.F.Klein 1993).

This suggests a brain-stem dysregulation (Abelson et al 2001) and hypersensitivity of the medulla. On the other hand, Gorman et al. (2000), argued based on several lines of evidence, that the brain-stem respiratory centres constitute a secondary mechanism by which panic attack symptoms relating to respiration manifest, as one if several pathways that are activated by central excitation of the amygdala.

3. GABA -benzodiazepine system:

It has been observed that there is a decrease in benzodiazepine receptor binding on single photon emission computer tomography (SPECT) in the prefrontal cortex and in hippocampus of patients with panic disorder.

The panicogenic effect of Flumazenil, the benzodiazepine antagonist was found in patients with panic disorder, but not normal subjects, suggesting a deficiency in endogenous anxiolytic ligand or altered benzodiazepine receptor sensitivity in panic (Roy-byrne at all 1990).

4. The serotonergic system.

It is thought that this system at least indirectly modulates deregulated response in panic disorder.

Indirect evidence for this is provided by the high efficacy of SSRIs in treating panic. It is thought that it acts via desensitization of brain fear network through inhibiting projections from the raphe nuclei to various cortical and subcortical formations responsible for panic.

5. Hypothalamic-pituitary-adrenal axis:

There is some evidence for uncoupling of noradrenergic and HPA axis activity in patients with panic disorder (Caplan et al. 1995). However, cortisol responses in lactate induced panic have suggested HPA axis involvement in anticipatory anxiety, as it is known to occur in other anxiety and stress states, but not in the panic attacks (Hollander et al. 1989).

D) Neurocircuity of fear:

The most recently proposed model of panic attempts to integrate neurochemical, imaging, and treatment findings in the disorder, coupled with mostly clinical work in the neurobiology of conditioned fear responses (Coplan and Lydiard1998; Gorman et al. 2000).

The model postulates that panic attacks are to a degree analogous to animal fear and avoidance responses and may be manifestations of dysregulations in the brain circuits underlying conditioned fear responses.

Panic is speculated to originate in an abnormally sensitive fear network, cantered in the amygdala. Input to the amygdala is modulated by both thalamic input and prefrontal cortical projections, and amygdalar projections extend to several areas involved in various aspects of the fear response, such as the locus coeruleus (involved in arousal), the brain-stem (respiratory activation), the hypothalamus (activation of the HPA stress axis), and the vortex (cognitive interpretations). The model is thought to explain why a variety of biologically diverse agents have panicogenic properties (by acting at different pathways or neurochemical systems of this network); It is proposed that the respiratory brain- stem nucleus could not be directly triggered by such a variety of agents (Gorman et al. 2000). Thus, dysregulated "cross-talk "between the various neurotransmitter systems previously described, such as serotonergic, noradrenergic, GABA-ergic, CRF, and others, may underlie the pathogenesis of panic (coplan and Lydiard1998).

This is a very comprehensive and theoretically exciting biological model of panic, which is still in great need of empirical validation.

When a person suffers from anxiety disorder due to another medical condition, the presence of that medical condition leads directly to the anxiety experienced. The anxiety is the predominant feature and may take the form of panic attacks, obsessive-compulsive behaviour, or generalized anxiety.

E) Symptoms of Anxiety Disorder Due To Another Medical Condition:

In anxiety due to another medical condition, the most frequently displayed symptom is anxiety in some form, even though there is another medical condition present that underlies and leads to the anxiety. General characteristics of anxiety include muscle tension, heart

palpitations, sweating, dizziness, or difficulty catching the breath. In addition to these physical symptoms, anxiety in general also leads to restlessness, possibly a fear of something impending that will be catastrophic, or fear of being embarrassed or humiliated.

Anxiety due to another medical condition may exhibit several symptom pictures. For example, if the anxiety shows itself as panic disorder, symptoms may include sudden onset of terror with no specific precipitating event (NIMH, n.d.). Along with the terror, a pounding heart, sweating, feeling faint, or dizziness may be experienced. The patient with panic may have physical symptoms that suggest a heart attack, also. These include feeling chilled, numbness in hands, nausea, chest pain, and feelings of smothering. A sense of loss of touch with reality, fear of some impending doom, and fear of losing control add to the impact of panic. Many people who experience panic attacks are convinced they are having a heart attack and seek medical attention at emergency rooms.

If the anxiety is experienced as generalized anxiety, a feeling of increased worry and tension with little or no precipitating factor prevails. These people expect disaster to occur and have increasing concern about health, money, family problems, or work. They can't relax, have an exaggerated startle response, and can't concentrate well. Physical symptoms that accompany generalized anxiety include feeling tired, headaches, muscle aches, irritability, sweating, nausea, feeling lightheaded, and difficulty breathing.

If the anxiety is felt as obsessive-compulsive symptoms, there will be intrusive thoughts that the person doesn't want to have and that bring on anxiety. This leads the person to perform certain behaviours or rituals that decrease the anxiety temporarily. The rituals may reach the point of controlling the person's behaviour. Checking things, touching things in a certain order, and counting are among the most common of these rituals. The thoughts that trigger anxiety many times have to do with harming loved ones, performing sexual acts that are unacceptable to the person, or thinking about things that go against the person's religious beliefs.

In order to give this diagnosis to a patient, there must be evidence that shows the anxiety, regardless of the way it is exhibited, is due to the direct physiologic effects of another medical condition (American Psychiatric Association, 2013). History, physical examination, or laboratory findings are used to establish this direct effect. Anxiety due to another medical condition is not better explained by another mental disorder and does not occur only during the course of delirium. Clinically significant distress must be present, and the functioning of the person in social, occupational, or other areas of life must be impaired.

Careful and thorough medical evaluation must be conducted to determine the presence of the medical condition that leads to the anxiety (Gagarina, 2011). Some of the medical conditions that may be involved in this disorder are hyperthyroidism, hypothyroidism, hypoglycaemia, and hyperadrenocorticism. Heart related problems may also underlie this disorder. Some of these conditions are congestive heart failure and arrhythmia. Breathing problems such as COPD, pneumonia, and hyperventilation also can initiate anxiety. Neurological conditions like encephalitis or neoplasms can lead to anxiety (Bourne, 2014).

There must be a close association between the medical condition and anxiety in order for this diagnosis to be appropriate. That is, the anxiety symptoms must occur close in time to the onset, worsening, or lessening of the medical condition. If the features of anxiety that are seen are not typical for a primary anxiety disorder, and there is a medical condition present, this is an indication that anxiety due to another medical condition may be an appropriate diagnosis.

Differential Diagnosis:

One of the major considerations in diagnosing anxiety due to another medical condition is to be certain the anxiety doesn't occur just during the course of delirium. It is appropriate to make the diagnosis if the anxiety occurs directly due to dementia, however. Differentiation of anxiety due to the effects of continuing substance use or abuse must be considered, also (Gagarina, 2011). Withdrawal from a substance or exposure to a toxic substance would lead to a diagnosis of Substance-Induced Anxiety. Medical examination including drug screens would be useful in this situation. It is possible to have a dual diagnosis of Anxiety Due to Another Medical Condition and Substance-Induced Anxiety disorder if criteria for both diagnoses are met.

A primary anxiety disorder or an adjustment disorder with anxiety are two other conditions that must be differentiated from anxiety due to another medical disorder. In the first of these, there is no direct link to a medical condition that causes the anxiety. In the other, onset of the anxiety at a later age or a family or personal history of anxiety should suggest an adjustment disorder.

F) Methodology:

A total number of 22 patients, with established diagnosis of panic disorder according to the criteria in DSM4 were randomly selected in a clinic in Homs, Syria in 2007-2008. A barium swallow contrast radiographic investigation was performed to all of them, also in Trendelenburg tilt position.

G) Results and conclusion of Study:

20 patients out of 22 had hiatal hernias!

Small HH.	**12 (4 GERD* +, 1 GERD ++)**
Medium sized HH	**4(GERD +++ severe)**
Big sliding HH	**4(GERD + moderate)**
Comorbid depression	**15**
Comorbid agoraphobia	**9**
Maintenance therapy 6 months	**14**
Maintenance therapy 1 year	**4**
Total remission after one year	**18**
No remission after 1 year treatment	**2**

*GERD gastro esophageal reflux disorder

All other psychiatric disorders were ruled out and also, all other medical conditions that might mimic or cause panic attacks or anxiety. All of the patients were not aware of having hiatal hernias. However, most if them had different degrees of abdominal discomfort, flatulence and some of them had gastroesophageal reflux in different degrees as mentioned above.

A very interesting thing is the notion that the majority of studied cases were people who had the habit of taking a nap after the main meal of the day, which is a common practice in the country. And most of the first or recurrent attacks happened upon awakening or even made the person wake up in panic.

This demonstrates the temporal and organic relationship between panic attacks and HH, the symptoms of which are mostly at play in a lying patient with full stomach.

What is more, all of them had the major complain of debilitating panic attacks and multiple visits to emergency units in hospitals, a crippling anticipatory anxiety and 9 of them developed agoraphobia and 15 secondary depression eventually.

As discussed above, the neurocircuity of fear theory postulates a summing up of different manifestations of dysregulation in the brain circuits that underlie conditioned fear responses.

So, while type II, III, IV Hernias supposedly manifest with more obvious physical symptoms that are related directly, and contribute to the hyperventilation pattern of breathing with the cascade of reactions that might lead to panic attacks; still small sliding hiatal hernias exert a different type of action through other presumed pathways, by feeding the thalamic input by noxious symptoms (bloating, distention, or heartburn), that is interacted and modulated by amygdalar and prefrontal cortical projections. Thus, the interpretation of these non significant symptoms, is exaggerated triggering the Locus Coeruleus and brain-stem respiratory activation.

Therefore this circle of fear can be activated starting at any point of the circuit, in an already sensitive nervous system.

Besides the usual medications and psychotherapeutic treatment, patients received education about HH. They were advised against.

Overeating or eating food known to contribute to bloating and gas, such as cabbage, beans, and lentils that may cause bloating and Avoiding or limiting caffeine intake.

Avoiding alcohol, especially hard liquor.

Wearing loose, comfortable clothing around the torso.

Applying physiotherapy like massage and different types of exercises and also relaxation sessions are all of pivotal importance.

In conclusion we can say that Hiatal Hernias might be represented in panic disorder patients more than thought of before, and paying attention to the possibility of it's presence in patients may help clinicians and patients alleviate an important triggering cue of panic attacks and disorder; the thing that would eventually result in shorter and more effective treatment and, off course, shorten the period of maintenance therapy. However, these findings need be replicated with greater number of patients to have more validity and consideration.

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