

Belly Dancer's Syndrome: Causes, Clinical Presentations, and Treatment Options

Senior Research Project

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Abstract

Belly dancer's syndrome, also known as diaphragmatic flutter, is a rare condition in which rapid myoclonus of one or both hemidiaphragms is present. The abdominal muscles may also be involved. These contractions are uncontrollable and involuntary. Interestingly, the myoclonus often ceases when the afflicted patient sleeps, returning again when he or she awakens. The contractions are not associated with cardiac rhythms or normal respiratory rate. Pain is often associated with these contractions. Pain patterns mimicking myocardial infarction pain may also be present, which may delay accurate diagnosis. A key component of belly dancer's syndrome is a dual respiratory pattern, allowing blood oxygenation through both voluntary ventilation and the involuntary diaphragmatic and abdominal contractions. Arterial blood gas levels are unaffected by belly dancer's syndrome, even when normal respiration is medically arrested. During such time, the fluttering of the diaphragm provides sufficient blood oxygenation to sustain arterial blood gases as normal levels. The cause of belly dancer's syndrome varies, making diagnosis difficult. Such causes include disruptions within the central or peripheral nervous systems, anxiety, nutritional imbalances, and the administration of certain pharmaceuticals. Magnetic resonance imaging, computerized tomography, electrocardiography, and plain film radiography often yield no abnormal findings. Fluoroscopy and electromyography utilizing the Koepke technique are the most advantageous methods of diagnosis. Belly dancer's syndrome is often treated with blocking or crushing the phrenic nerve. Many medications have also proven beneficial in treating the condition.

Keywords: *Belly dancer's syndrome, Leeuwenhoek's disease, moving umbilicus syndrome, diaphragmatic flutter, abdominal myoclonus*

Introduction

Belly dancer's syndrome is a rare condition consisting of involuntary, repetitive, often rhythmic contractions of the diaphragm, causing a vibration of the abdomen reminiscent of the undulating movements of a belly dancer. This condition is more commonly known as diaphragmatic flutter. Other terms describing this phenomenon include belly dancer's dyskinesia, belly dancer's myoclonus, Leeuwenhoek's disease, respiratory myoclonus, moving umbilicus dyskinesia, moving umbilicus syndrome, truncal dystonia, abdominal dystonia, abdominal myoclonus, diaphragmatic tremor, and diaphragmatic myoclonus. These terms shall be used interchangeably in this literature review. Diaphragmatic flutter was first described by Antony van Leeuwenhoek in 1723. He himself was affected by the condition, and correctly discerned the source of his affliction to be his diaphragm.¹ Belly dancer's syndrome is often misdiagnosed at first, owing to its rarity and to its varying causes. Such causes include reactions to pharmaceuticals, neurologic disorders, and injury to the central nervous system, though many cases are idiopathic. Some cases have even been linked to psychosocial factors. Many treatments have been found beneficial, though no single treatment has been proven effective in all cases.

A search was conducted for relevant articles using the following databases: PubMed, DynaMed, Science Reference Center, MasterFILE, EbscoHost, and OvidSP. Keywords utilized in this search include belly dancer's syndrome, diaphragmatic flutter, diaphragmatic tremor, and diaphragmatic myoclonus. Articles included in the search were limited to the English language and translations to English. Case reports and literature reviews were consulted. Due to the

rarity of belly dancer's syndrome, articles selected for review included those published between 1928 and 2011. A total of forty-eight patient cases were included in the articles selected for review.

Clinical Presentation

The most common presentation of belly dancer's syndrome is a visible or non-visible, involuntary, rapid fluttering or rolling of the abdominal wall or epigastrium.² This fluttering is caused by quick contraction and release of the diaphragm. This condition is considered to be a form of myoclonus.³ After conducting a review of 42 cases of belly dancer's syndrome, Rigatto and De Medeiros found that the rate of diaphragmatic contraction ranges between 35 and 480 contractions per minute, with the average rate found to be 150 contractions per minute.⁴ This fluttering can occasionally be so rapid as to shake the bed the patient may be laying on.⁵

The syndrome is associated with three different respiratory patterns, each demonstrated in various case reports: tachypnea; diaphragmatic flutter with apneustic respiration; and a dual respiratory pattern wherein the high frequency fluttering respiration is superimposed upon a much slower, normal respiratory pattern.⁵ In most cases, the flutter is present during inspiration and expiration, although some reports demonstrate flutter during only one of the two phases of respiration. When the latter is the case, all relevant studies have described an inspiratory flutter. This fluttering respiration is typically not suppressed when the patient holds his or her breath.⁶ Upon EMG studies, these movements are found to be due to myoclonic contractions of one or both hemidiaphragms. Approximately two thirds of those with the disorder experience involvement bilaterally. The majority of cases of unilateral belly dancer's

syndrome present in the left hemidiaphragm.⁴ Dyspnea is frequently present.⁷ Heart rate is typically normal.⁸ In most cases, the diaphragmatic fluttering ceases during sleep.^{5,9} Laboratory studies, plain film radiographs, electrocardiograms, computerized tomography (CT) scans, and magnetic resonance imaging (MRI) remain within normal limits. Diaphragmatic fluttering is frequently associated with pain or discomfort in the wall of the upper abdomen or lower chest, although it can present without pain.^{3,10} Patients may present with shortness of breath or fatigue.³

Despite disrupted spontaneous respiration, arterial blood gas levels typically remain within normal ranges. Even when voluntary respiration is arrested, a patient with belly dancer's syndrome will maintain adequate blood oxygenation. In a report of a patient who was administered 30 mg pethidine, completely suppressing her spontaneous ventilation for a period of 15 minutes, her diaphragmatic flutter continued unabated. Despite the lack of spontaneous ventilation, the patient did not become cyanotic, nor did her arterial blood gases fall below normal levels. It is speculated that the rapid diaphragmatic fluttering allows enough blood gas exchange to remain within normal limits.⁵ This phenomenon is strong evidence supporting the presence of a dual respiratory pattern, a key feature in patients with diaphragmatic flutter. This dual respiratory pattern consists of the high frequency of diaphragmatic flutter superimposed on the much lower frequency of normal respiration.^{7,8} Tamaya, Kondo, and Yamabayashi have postulated that the dual respiratory pattern often presenting with diaphragmatic flutter may be due to the respiratory center receiving separate impulses, requiring both a fast and slow respiratory rate simultaneously.⁵

Belly dancer's syndrome can be intermittent, lasting minutes or hours.⁸ It may be a chronic, recurrent affliction, or it may have a more isolated or sporadic presentation.¹¹ While most cases depict this condition to be episodic in nature, some cases have been reported of long term continuous diaphragmatic flutter for several months together.³

The rate of diaphragmatic fluttering in belly dancer's syndrome can be identified through needle electromyogram (EMG) studies. Rates identified in this manner have ranged between 0.5 and 15 Hz, with the majority of cases presenting with contractions at rates between 4 and 6 Hz.¹²

Patients experiencing belly dancer's syndrome may complain of severe pain in the left chest wall and into the left arm, a pain pattern typically indicative of myocardial infarction. However, electrocardiography rarely demonstrates objective evidence to support any cardiac abnormality. The similarity of these symptoms to acute myocardial infarction can confuse the patient and his or her physicians, delaying correct diagnosis and treatment.¹³

Diagnostic Tools

Fluoroscopy is frequently utilized when the patient's symptoms indicate diaphragmatic flutter. This diagnostic tool is an x-ray in motion, demonstrating in real-time the movement of the patient's skeleton, soft tissues, and air masses. Analysis of these structures during fluoroscopy will provide evidence of the patterns of movement of the diaphragm. Fluoroscopy will show the presence of both unilateral and bilateral diaphragmatic flutter, even if only unilateral signs and symptoms are present.^{2,5,14}

When diaphragmatic flutter is suspected, but not visible, electromyogram of the diaphragm can prove the presence of myoclonic activity.⁸ An excellent method for evaluating the diaphragm through the use of electromyography is the Koepke technique. When using this technique, monopolar electrodes are inserted through the chest wall, directly into each hemidiaphragm. This method allows separate and simultaneous evaluation of each individual hemidiaphragm, unlike the utilization of an esophageal electrode, which cannot differentiate between the hemidiaphragms.¹⁵

Another way to diagnose diaphragmatic flutter is through the use of a volume-time spirogram. This diagnostic method provides evidence of the dual respiratory rhythms common in diaphragmatic flutter.⁸ In one example of diaphragmatic flutter diagnosed through this method, one respiratory rhythm was shown to have a large, slow rate of 10-15 breaths per minute, with a tidal volume of 1500-2000 ml. This patient was also found to have a second wave with a smaller, faster rhythm of 180 breaths per minute and a 200 ml tidal volume.⁵

The diagnosis of diaphragmatic flutter may be confirmed by respiratory inductive plethysmography.¹⁶ Hoffman, Yahr, and Krieger reported four cases of respiratory inductive plethysmography detecting diaphragmatic flutter in patients who had previously undergone coronary artery bypass grafting (CABG), but were unable to successfully wean from mechanical ventilator support.¹⁷ This method of testing can also detect diaphragmatic flutter incidentally when no associated symptoms exist. Adams, Zabaleta, and Sackner reported the unexpected detection of diaphragmatic flutter in three infants undergoing respiratory inductive plethysmography to monitor apnea known to be commonly associated with respiratory

syncytial virus (RSV), with which these patients were afflicted.¹⁸

Causes of the disorder

Belly dancer's syndrome has many causes. These include disruptions in the central nervous system, the peripheral nervous system, organic disorders, spinal tumors or cysts, disc herniations, and nutritional imbalances. The most common of the causes involving the central nervous system is encephalitis. When the syndrome is caused by irritation of the phrenic nerve, the heart is the most frequent source of the irritation. This can result in diaphragmatic flutter synchronous with systole. Additional causes of diaphragmatic flutter include pleurisy, peritonitis, or ischemia of the diaphragm.⁴ Previous lung disease or surgery, irritation of the phrenic nerve or diaphragm from such conditions as pleurisy and peritonitis, and cardiomegaly in the presence of rheumatic heart disease may also be implicated.⁸ In the case of a 13 year old female, symptoms of belly dancer's syndrome developed after her bronchitis and cough-variant asthma failed to improve.³ Psychogenic factors are also frequently suspected due to the symptoms only presenting while the patient is awake.⁵ When diaphragmatic flutter is present bilaterally, a central origin is more likely.

A recent history of trauma to the spine or head has been an associated factor in some cases of belly dancer's syndrome, and should be taken into consideration as contributing to the onset of the disorder. One patient had experienced head trauma during a diving accident 16 months prior to presentation with belly dancer's syndrome. However, at the time of injury, she was found to have no concussion, skull fracture, or spinal injury, although she was found to have multiple muscle spasms in the neck, back and shoulders and was required to wear a

Philadelphia collar for a period of six weeks. The source of this patient's case of belly dancer's syndrome may have been trauma to the cervical spine, which can result in damage to the root of the phrenic nerve.³ While a physician may not be able to directly associate the onset of the symptoms of belly dancer's syndrome with the exact moment of spinal or cranial injury, the disorder should be included in the differential when a patient presenting with diaphragmatic flutter has a history of recent trauma.

Disorders of the central nervous system may also be a causative factor. Roggendorf et al report a case in which a patient developed belly dancer's syndrome five months after experiencing central and extrapontine myelinolysis (CPM/EPM), suggesting that the basal ganglia lesions implicated in EPM may be a generator of belly dancer's syndrome.¹⁹

Diaphragmatic flutter may be a complication of coronary artery bypass grafting (CABG). Reports exist which demonstrate four patients contracting diaphragmatic flutter after undergoing CABG. Diaphragmatic dysfunction has been proven to be a common complication of this procedure, so it stands to reason that diaphragmatic flutter may be a plausible complication as well.¹⁷

The onset of belly dancer's syndrome has been attributed to the administration of certain pharmaceuticals. In one case, an 80 year old male patient developed diaphragmatic flutter after beginning a course of galantamine for the treatment of Alzheimer's disease. This medication was identified as the source of his affliction by the condition's abatement upon cessation of medication.²⁰ Linzasoro et al reported another instance of belly dancer's syndrome being induced by the patient's chronic use of clebopride, a drug utilized in the treatment of digestive

disorders.²¹

Metabolic deficiencies may also be implicated in the cause of belly dancer's syndrome. Dogan and Yurten speculate that spinal myoclonus could be brought on by a deficient level of serum vitamin B12. While there have been few cases concerning this relationship, a report exists of an 85 year old female with belly dancer's syndrome, in whom no other MRI findings or blood analysis results indicated a cause for the disorder. She was, however found to be deficient in serum B12 levels.²² While there is not sufficient evidence to confirm the role of low serum B12 levels in cases of belly dancer's syndrome, it would be prudent for physicians to take this relationship into consideration.

Some cases of belly dancer's syndrome may be caused by biochemical abnormalities. Baker, Kenny, and Zuberbuhler report a case supporting this possibility. In this case study, a seven year old was reported to have developed belly dancer's syndrome after the onset of a malabsorption syndrome. Interestingly, the symptoms of belly dancer's syndrome resolved upon resolution of the malabsorption abnormality through the correction of serum magnesium, calcium, and potassium levels. This patient also had several other conditions presenting simultaneously with the belly dancer's syndrome and malabsorption syndrome. These conditions include idiopathic hypoparathyroidism and Addison's disease. It may be prudent to consider these conditions when evaluating patients presenting with diaphragmatic flutter.²³

Because the causes are so varied, belly dancer's syndrome appears to have no predominance in any one population. The cases reviewed have described a wide variety of affected patients, including men, women, Caucasian, Japanese, Saudi Arabian, and African American individuals.

The youngest patient in the reviewed cases presented with belly dancer's syndrome at just four months old.⁹ The oldest patient in the cases included for review was a 93 year old woman.²⁴

Treatment options and outcomes

Due to the varying causes of belly dancer's syndrome, it follows that many methods of treatment have been shown effective.

Several pharmaceuticals have been utilized in the treatment of belly dancer's syndrome. Diphenylhydantoin is frequently used to treat this disorder, but it is not effective in all cases.⁵ One case of successful treatment with diphenylhydantoin reported that the patient was given 200 mg of the drug intravenously, with symptoms of abdominal flutter being abolished within ten minutes of injection. Relief in this case was still present at the patient's one-year follow-up.⁶

Another pharmaceutical which has proven beneficial in the treatment of diaphragmatic flutter is diazepam. Ruser reported a case of a 45 year old male afflicted with diaphragmatic flutter paired with rapid rhythmic nodding of the head. The patient's signs and symptoms were able to be kept under complete control through the administration of diazepam.²⁵

Haloperidol has also been utilized in the treatment of belly dancer's syndrome. However, its use has been associated with some side effects. Haloperidol has been known to induce sleep in some patients. It has also been reported to cause temporary fixation of the eyes. Haloperidol can be given by intramuscular injection or orally. A dose of 2 mg orally has been shown to be effective in cases of belly dancer's syndrome.⁵

Phenytoin can also be a useful treatment. Young, Clap, Salcedo, and Whitlock reported a case of a woman with bilateral diaphragmatic myoclonus obtaining relief of her symptoms after treatment with intravenous phenytoin. Upon follow-up 12 months later, this patient reported being symptom free for the duration of that time.²

Yet another medical treatment for belly dancer's syndrome is the administration of clonazepam. This medication has been shown to reduce both the frequency and amplitude of the abdominal contractions associated with this condition, and in some cases, completely reverse the symptoms of diaphragmatic flutter.^{26,27} However, one report described a case where the cessation of clonazepam therapy was directly followed by a return and progressive worsening of the syndrome. This patient was initially treated for unilateral belly dancer's syndrome with clonazepam. After clonazepam therapy ended, his abdominal contractions on the previously afflicted side returned with higher frequency and amplitude than prior to treatment, and the opposite abdominal wall became involved. Paraspinal muscles began to present with myoclonic jerks, resulting in twisting of the trunk. The patient's myoclonus also became inducible by tendon tapping at any location of the body.²⁷ Given these findings, it would be prudent to conduct further research into the effectiveness and adverse effects of clonazepam in the treatment of belly dancer's syndrome prior to its administration.

When pharmacologic therapies prove ineffective, phrenic nerve block or crushing can alleviate symptoms of unilateral diaphragmatic flutter. Relief of symptoms is instantaneous.^{3,5} This procedure has been shown to allow for a return to normal diaphragmatic function at a later date through the process of neurological regeneration.²⁸ For the period of time prior to the

completion of the regeneration of the phrenic nerve, pulse oximetry and pulmonary function tests typically remain within normal limits, although in some cases the procedure can result in decreased lung volumes.^{3,29} It can be ascertained that the intact phrenic nerve, enabling the full function of one hemisphere of the diaphragm, can maintain oxygenation at adequate levels for both rest and exercise states. Interestingly, after diaphragmatic function returns to normal, the symptoms of belly dancer's syndrome do not return.³

Some cases of belly dancer's syndrome may be resolved without medical intervention. If there is a psychogenic cause, the patient's symptoms may be successfully treated through reassurance alone.⁸ In the case of the three infants afflicted with RSV, mentioned previously, evidence of diaphragmatic flutter was found incidentally through monitoring with respiratory inductive plethysmography monitoring for apnea. In all three of these patients, the diaphragmatic flutter resolved spontaneously and without treatment within four days of onset, with no recurrence reported at one-year follow-up.¹⁸

Related disorders & Differential diagnoses

Belly dancer's syndrome is a rare condition not readily identified by medical practitioners. The signs and symptoms of this syndrome frequently resemble those of other conditions. These differential diagnoses include: diaphragmatic pacing, tachyarrhythmia, transient obstruction of the mechanical valve, pulmonary hypertensive episodes, drug induced hypotension, and hyperventilation related to anxiety. Many of these differentials can be excluded by prolonged monitoring, telemetry, holter tape recording, echocardiography of valve function, and extensive pacemaker interrogation without reproduction of symptoms.⁸

The symptoms of belly dancer's syndrome can mimic those of acute myocardial infarction. A 57 year old white male was reported to have presented with severe pain in the left chest wall, with radiation into his left arm and hand. Interestingly, all cardiac examinations revealed no evidence of a heart condition. After more testing, the patient's symptoms were determined to be due to diaphragmatic flutter.¹³ Lurje and Stern speculate that cardiac symptoms can occur when diaphragmatic hypotonus is present. They believe this allows intra-abdominal pressure to push the diaphragm into the cardiac space, impairing cardiac function enough to resemble the symptoms of a myocardial infarction.³⁰ Due to the similarities of presentation, if a patient complaining of symptoms of cardiac malady demonstrates no objective evidence of such a malady's existence, and is unresponsive to treatment for applicable cardiac conditions, belly dancer's syndrome should be considered as a possible etiology.

The symptoms of belly dancer's syndrome are similar to hyperventilation due to anxiety, even when a psychogenic factor is not the cause of the condition.⁸ Because of this, correct diagnosis and treatment may be delayed. While belly dancer's syndrome is sometimes due to psychogenic factors such as anxiety or hysteria, this is not always the case. Careful consideration must be made by the physician to determine if there is a somatic cause for the condition. If the cause is indeed determined to be psychogenetic in nature, correct treatment can only be achieved through proper diagnosis of diaphragmatic flutter.

The rapid abdominal, diaphragmatic, and epigastric contractions of belly dancer's syndrome may easily be confused as being due to other causes of convulsions. Seizure disorders may be blamed. Stress conversion disorders and Tourette's syndrome also have a similar presentation.

In cases of diaphragmatic flutter associated with inspiratory stridor, vocal cord dysfunction could appear to be the cause. However, the course of treatment for each of these conditions may not resolve the symptomatology. Such treatments include the administration of lorazepam, valproic acid, haloperidol, pimozide, clonazepam, carbamazepine, diazepam, fluoxetine, clonidine patches, and biofeedback. If a condition which typically responds to such treatments is suspected as the cause of the patient's complaints, the failure to respond to the treatments may lead physicians to suspect a diagnosis of diaphragmatic flutter.³

Case Presentations

Case 1

A 12 year old girl developed bursts of abdominal jerks four days after a flexion injury to the cervical spine. These jerks were only present while she was awake. They had been present for one month when the patient sought treatment for them. Neurologic examinations, plain film radiographs, and MRI of the cervical spine showed no abnormal results. A fluoroscopy was performed, examining diaphragmatic movements. The fluoroscopy demonstrated bilateral fluttering movements of the diaphragm primarily coinciding with inspiration. A surface EMG recording of the muscles of the abdominal wall during voluntary respiration demonstrated rhythmic abdominal movements of 4 to 5 Hz, occurring during inspiration. A 20 hour polygraphic-video-EEG study confirmed these findings. The patient was administered 20 mg of carbamazepine daily for ten months. At the end of these ten months, the symptoms of the disorder were absent, and the administration of carbamazepine was progressively tapered until the patient was no longer taking medication. Upon follow-up eighteen months after the initial

cervical spine trauma, she remained asymptomatic and taking no medications for her previous condition of diaphragmatic flutter.¹²

Case 2

In 1949, an elderly man collapsed while getting off a bus. Upon admission to the hospital, the patient complained of excruciating, compressing pain in his chest, shortness of breath, and dizziness, all of which were of sudden onset. The patient claimed he was 100 years old, having served in the American Civil War, despite medical examiners recording his appearance to be approximately 65 years of age. The patient was belligerent, argumentative, and constantly moved about. Loud, rapid, irregular sounds were auscultated over the chest and precordium. The rates of these sounds varied from 200 to 240 per minute. Pulse was 72 beats per minute. Blood pressure and respiratory rate were normal. Heart size was within normal limits. There was evidence of moderate emphysema, but no other respiratory abnormalities were present. Visual examination and palpation of the abdomen yielded no abnormal results. The remainder of the physical examination was within normal limits. The patient was diagnosed with myocardial infarction, but ECGs following this diagnosis revealed no evidence of such. A chest radiograph demonstrated moderate emphysema, elevation of the left hemidiaphragm hemisphere, and slight displacement of the mediastinum to the right. Complete blood count and urinalysis were normal. The patient was sedated with morphine, Demerol, and chloral hydrate, but he continued to be belligerent. Given the lack of evidence to support the previous diagnosis of myocardial infarction, the patient was diagnosed with diaphragmatic flutter. He received no treatment for this condition, however, as he was required to leave the hospital

upon discovery that he was using a false name and was thus not entitled to hospitalization at a Veterans Administration hospital.¹⁰ Several reports exist, demonstrating this same patient presenting at various Veterans Administration hospitals across the United States, seeking treatment for the same condition depicted above. Each time, he uses a different false name and generally has a grandiose and fictitious background story. Many physicians who have dealt with this man and authors who have reported cases concerning him attribute his case of diaphragmatic flutter to be of psychogenic origin.^{12,31}

Case 3

A 13 year old Caucasian female contracted a frequent, severe cough during an upper respiratory infection. Previous history included striking the right side of her head on the edge of a swimming pool 16 months prior to contracting the upper respiratory infection, with no concussion, skull fracture, or spinal injury found. She did however have neck, back, and shoulder muscle spasms, and was required to wear a Philadelphia collar for 6 weeks. While suffering from the severe cough during the presenting upper respiratory infection, the patient developed bronchitis and cough-variant asthma, for which she was hospitalized. She did not improve, despite treatment with frequent nebulized beta-agonists, intravenous aminophylline, and corticosteroids. While in the hospital, the patient developed a loud inspiratory stridor which preceded her cough. The cough and stridor increased in frequency. She then abruptly developed a striking breathing pattern consisting of tachypnea and inspiratory stridor, which did not cease while awake, but remitted when sleeping. The breathing pattern was also made worse with anxiety. The patient experienced dyspnea and was limited in speech and physical

activity. Physicians considered the diagnoses of seizure disorder, stress conversion disorder, Tourette's syndrome, and vocal cord dysfunction. To address these differential diagnoses, the patient was administered lorazepam (Ativan). Clonazepam, carbamazepine (Tegretol), diazepam (Valium), fluoxetine (Prozac), clonidine (Catapres) patches, and biofeedback. None of these treatments were effective in alleviating the patient's symptoms. The patient's respiratory rate was 140 to 160 breaths per minute, consisting of respiratory cycles of 13 to 16 shallow inspirations and one normal expiration. She occasionally demonstrated facial tics. Her blood gas values exhibited evidence of hyperventilation, though her blood tests were normal, as were EEG, CT and MRI of the head, CT of the chest, and plain film radiographs of the cervical spine, thoracic spine, and chest. Fluoroscopy of the diaphragm revealed rapid myoclonus of the left hemidiaphragm, present predominantly during inspiration. Diaphragmatic excursion was normal. Because previous pharmacologic treatment was unsuccessful, a nerve block at the left C4 nerve root was performed using bupivacaine (Marcaine). This resulted in paresis of the left hemidiaphragm and temporary resolution of her abnormal breathing pattern. Interestingly, continued nerve blocks at the C4 level for the following two weeks, as well as injection of methylprednisolone (Solu-Medrol) at the same nerve root level failed to provide any further or more lasting relief of symptoms. The left phrenic nerve was then crushed, which resulted in complete relief from all symptoms while retaining pulse oximetry and pulmonary function tests within normal limits. A follow-up fluoroscopy 2 months after the phrenic nerve crush revealed that normal function of the left hemidiaphragm had returned, as expected. The patient continued to be asymptomatic at follow-up one year after phrenic nerve crush treatment.³

Case 4

An 80 year old male presented with bilateral, segmental abdominal pulsations with sudden onset of 24 hours duration. These pulsations occurred in waves of 20 to 30 pulsations per minute throughout wakefulness, but were absent during sleep. Episodes of pulsations lasted 12 to 15 minutes each. These pulsations disrupted respiratory function, predominantly during inspiration. There were slight cervical contractions associated with the abdominal pulsations, which occasionally extended into the left arm. The only position of relief, which was partial, was elevating both arms while seated. Physical and neurological examinations were normal. Cerebral and cervico-dorsal MRIs were normal. EMG study of the trapezius, scalenes, and inferior intercostals revealed involuntary, irregular, and pseudorhythmic activity, especially after active contraction of the muscle being tested. The patient's health history was relevant for hypertension, dyslipidemia, chronic renal failure, and Alzheimer's. Serum creatinine levels were consistent with the patient's previous history of chronic renal failure, but no other metabolic findings were significant. The patient's medications included torasemide for hypertension, gemfibrozil for dyslipidemia, and galantamine for Alzheimer's. The patient was diagnosed presumptively with respiratory myoclonus and galantamine was withheld, as it was the most recent medication added and had only been prescribed for one month prior to presentation with abdominal pulsations. A few days after cessation of galantamine administration, the episodes of abdominal pulsations were greatly diminished. The patient was then given 1,500 mg of valproic acid and 9 mg of clonazepam per day, resulting in further improvement in the patient's condition. Three weeks after the initiation of treatment, the patient was asymptomatic.²⁰

Case 5

A 29 year old athletic male presented with involuntary, painless, pseudo-rhythmic jerking of the abdominal wall and paravertebral musculature, greater on the left than right side. The patient's voice and respiratory actions were unaffected by the condition. This condition developed suddenly two weeks prior to presentation for care, when the patient was lifting a heavy box. In the days that followed onset, the frequency and severity of the condition increased. There was no change with sleep or sensory stimulation, but symptoms became worse with emotional stress. The patient reported having not been taking any medications at the time of onset, nor had he ever taken neuroleptics, lithium, SSRIs, or benzodiazepines. General examination, higher mental function, cranial nerve examination, deep tendon reflexes, Babinski test, strength, sensory function, autonomic function and cerebellar function were all normal. CBC, PCR, electrolytes, creatinine, common liver function tests, vitamin B12, folate levels, thyroid function, and CSF analysis were all normal. CT scan of the brain and abdomen were unremarkable. Needle EMG of the rectus abdominis and paravertebral musculature at the same metameric level was performed. The EMG revealed arrhythmic discharges lasting between 100 and 500 ms. This finding is consistent with myoclonus. A spinal MRI revealed a median-paramedian disc herniation at the T6-T7 disc level, placing a mild compressive effect on the ventral columns, more so on the left. Placebo treatments failed to resolve symptoms. Clonazepam was given in an amount of 2 mg per day, resulting in complete elimination of myoclonus. At follow-up examination two months later, the patient reported no recurrence of symptoms while continuing to take clonazepam.²⁶

Conclusions

Belly dancer's syndrome is a rare disorder, often mimicking several other disorders. This condition may be confused as myocardial infarction, hyperventilation, hysteria, seizures, or tremor disorders such as Tourette's. Contributing further to the confusion of diagnosis is that belly dancer's syndrome has many different causes, including metabolic deficiencies, psychogenic factors, trauma, and most commonly, disorders of the central nervous system. A thorough patient history and careful examination is necessary to obtain a correct diagnosis of belly dancer's syndrome and to ensure that proper treatment is given. The best diagnostic techniques to be utilized in the diagnosis of belly dancer's syndrome include fluoroscopy of the diaphragm and electromyography of the diaphragm through use of the Koepke technique. These testing techniques are seldom used, which may add to the seeming rarity of belly dancer's syndrome. If physicians would more frequently utilize these diagnostic techniques in patients presenting with fluttering motions of the diaphragm or abdominal wall, perhaps more cases of belly dancer's syndrome would be identified.

According to the articles reviewed, the most common treatments administered for patients suffering from belly dancer's syndrome include crushing of the phrenic nerve and mediating with diphenylhydantoin, clonazepam, and haloperidol. While these treatment methods have proven effective in many cases of this syndrome, several side effects have been noted, and success is not a guarantee in all patients afflicted. More research is warranted on the side effects of each of these treatments, both short term and long term, and in their overall efficacy in the treatment of belly dancer's syndrome.

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