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Unraveling belly dancer's dyskinesia and other puzzling diagnostic contortions: A narrative literature review

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Abstract:

Belly dancer's dyskinesia (BDD) is characterized by involuntary abdominal wall movements that are rhythmic, repetitive, and dyskinetic. The present study aims to review BDD's etiology, pathophysiology, and management. We searched six databases to locate existing reports on BDD published from 1990 to October 2023 in electronic form. A total of 47 articles containing 59 cases were found. The majority of the patients affected by BDD were female, accounting for 61.01% (36/59) of the cases. The mean and median ages were 49.8 (standard deviation: 21.85) and 52 years (range: 7–85), respectively. The BDD was unilateral in only 3.38% (2/59). The most commonly reported causes associated with BDD were 17 idiopathic, 11 drug-induced, 11 postsurgical procedures, 5 pregnancies, and 4 Vitamin B12 deficiencies. BDD is a diagnosis of exclusion, and other more common pathologies with similar presentation should be ruled out initially. Differential diagnostic reasoning should include diaphragmatic myoclonus, cardiac conditions, truncal dystonia, abdominal motor seizures, propriospinal myoclonus, and functional or psychiatric disorders.

Keywords:

Belly dancer dyskinesia, belly dancer's sign, dyskinesia, movement disorder

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Introduction

Belly dancer's dyskinesia (BDD) is a rare disorder that was first characterized in 1990. Iliceto *et al*. were the first to coin the term BDD, which they described as involuntary abdominal wall movements that were rhythmic, repetitive, and dyskinetic.[1] In 1976, Nichols reported the "belly dancer's sign" on a newborn with phrenic nerve palsy.[2] However, the case described by Nichols *et al*. resembles more diaphragmatic myoclonus than BDD.[2]

Nevertheless, some authors believe that BDD is a completely different clinical and pathophysiological entity from diaphragmatic myoclonus.[1] In BDD, the movements are described as slow, with characteristic writhing and contorting features. On the other hand, a diaphragmatic myoclonus is a semirhythmic jerking of the abdominal wall.[3] However, in the literature, most manuscripts did not depict the phenomenology of abdominal movements in detail. Furthermore, specific electrodiagnostic studies were only performed in a minority of the studies. In this context, the present review will only assess data from articles that reported individuals with probable BDD based on clinical criteria. It is important to underline that the term belly dancer syndrome has been unadvisedly used in many studies as a synonym of BDD and diaphragmatic

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myoclonus, probably due to the rarity of these conditions, lack of established clinical definitions, and misinterpretations of previously published literature.

BDD is a rare movement disorder characterized by involuntary and often rhythmic contractions of the anterior abdominal wall muscles. These contractions can be painful, predominantly bilateral, and exhibit slow writhing patterns resembling athetosis.[4] Furthermore, the movements result from contractions occurring in several muscle groups, including rectus abdominis, internal and external oblique, transverse abdominis, pectoralis major, and perineum muscles.[5] Associated symptoms involving other systems are usually related to the repercussions of abnormal abdominal muscle contractions, such as epigastric pulsations, sleep disturbances, dyspnea, hiccups, belching, and pyrosis. Interestingly, different respiratory patterns can affect abdominal movements. In this way, different breathing rhythms may decrease abdominal muscle contraction frequency, which can especially be observed during sleep.[6]

The pathophysiology of BDD remains unknown. Notwithstanding, there are several hypotheses for pathophysiological mechanisms in BDD, including dysfunction of spinal inhibitory interneurons, local structural neuronal circuitry reorganization, phrenic nerve irritation, basal ganglia lesions, spinal cord injury, and vitamin deficiencies.[7] Notably, BDD secondary to basal ganglia lesions is usually bilateral and subsides during sleep.[8] On the other hand, BDD associated with spinal cord or peripheral nerve lesions exhibit persistent abdominal muscle contractions without the influence of sleep patterns.[7]

Drug-induced BDD is one of the most frequently reported secondary causes of BDD. Some drugs associated with BDD in previous reports were levodopa,[9] antidopaminergics, domperidone,[4] clebopride,[10] and salbutamol.[11] In general, medications affecting the dopaminergic pathways are the most common cause of BDD secondary to drugs.

Further diagnostic methods for suspected cases of BDD may include ultrasound, electromyography (EMG), and videofluoroscopy of the chest, which does not always provide sufficient evidence of BDD. Additional investigations should include magnetic resonance imaging of the brain and spinal cord and computed tomography of the chest and abdomen to rule out structural lesions leading to BDD.[5] It is worth mentioning that, to the present date, there are no standardized tests for a definitive diagnosis of BDD.

Symptomatic treatment is the main strategy for managing BDD patients. Several therapeutic options have been empirically tried in previous studies, including benzodiazepines,[12] beta-blockers,[13] Vitamin B12 supplements,[14] antipsychotics,[7] antiseizure medications,[15] and antidepressants.[16] However, there is uncertainty and controversy surrounding the effectiveness of these drugs in previously reported studies. Botulinum toxin injections have also emerged as a potential option for the management of BDD, but additional evidence and clinical trials are needed to determine their efficacy.[17] Some reports describe combining various treatments to improve motor symptoms. There are no clinical trials, so the therapeutic management relies on case reports and expert opinions.

This review aims to provide an update and critical analysis of the current understanding of BDD pathophysiology and its management. We will describe the mechanisms resulting in variable disease presentations and outline symptomatology phenomena that can help guiding specific clinical management strategies.

Methodology

We searched six databases to locate existing reports on belly dancer dyskinesia published from 1990 to October 2023 in electronic form. Excerpta Medica (Embase), Google Scholar, Latin American and Caribbean Health Sciences Literature (Lilacs), Medline, Scientific Electronic Library Online (Scielo), and Science Direct were searched. The search term was "belly dancer dyskinesia [Table 1]."

Results

A total of 47 articles containing 59 cases of BDD were found [Table 2].[1,5,10,12,18-22] 61.01% (36/59) of the patients were female. The mean and median ages were 49.8 (standard deviation: 21.85) and 52 years (range: 7–85 years old), respectively. The BDD was unilateral in only 3.38% (2/59). The most commonly reported causes associated with BDD were 17 idiopathic, 11 drug-induced, 11 postsurgical procedures, 5 pregnancies, and 4 Vitamin B12 deficiencies.

Etiology and pathophysiology

Several factors can lead to BDD, including central and peripheral nervous system disorders

Table 1: FreeText and Medical Subject Headings search terms in the US National Library of Medicine

Query	Search term	Results
Belly dancer dyskinesia	("bellies"[All Fields] OR "belly"[All Fields]) AND ("dancer"[All Fields] OR "dancer s"[All Fields] OR "dancers"[All Fields]) AND ("dyskinesiae"[All Fields] OR "dyskinesias"[MeSH Terms] OR "dyskinesias"[All Fields] OR "dyskinesia"[All Fields])	35

Table 2: Cases of belly dancer's dyskinesia reported in the literature

Author	Age (year)/sex	Age onset BDD (year)	Cause	Unilateral/ bilateral	Management	Comment
Iliceto *et al*.[1]	25/female	24	Vaginal delivery	Bilateral	Partial improvement after seven months of the BDD onset	BDD frequency worsened with menstruation
	58/male	54	Cholecystectomy	Bilateral	No improvement with several therapeutical choices	BDD disappeared with full inspiration, brisk walking, and sleep
	67/female	65	Cystoscopy removal of renal calculi	Unilateral	No improvement with several therapeutic choices	BDD occurring during sleep, awakening patient
	74/female	71	Several abdominal procedures	Bilateral	Partial improvement with clonazepam	Video available
Caviness *et al*.[18]	52/female	52	Colectomy	Bilateral	No improvement with several therapies	
	68/female	64	Cholecystectomy	Bilateral	No improvement with several therapies	
	71/male	67	Idiopathic	Bilateral	No improvement with several therapies	
	64/female	61	Multiple sclerosis	Bilateral	No improvement with several therapies	
Kono *et al*.[12]	63/male	63	Idiopathic	Bilateral	Clonazepam showed improvement in frequency and amplitude	Spinal myoclonus
Kim *et al*.[19]	76/female	71	Appendicectomy	Bilateral	NA	
	33/male	33	Appendicectomy	Bilateral	NA	
Linazasoro *et al*.[10]	52/female	52	Clebopride	Bilateral	Discontinuation of clebopride. Clonazepam was started Transcutaneous electrical nerve stimulation showed improvement in the symptoms	BDD only occurred in the sitting and lying positions. She also developed akathisia that improved after discontinuation of clebopride
Inghilleri *et al*.[5]	85/female	85	Idiopathic	Bilateral	Carbamazepine was effective in the management of BDD	Spinal myoclonus
Roggendorf *et al*.[20]	35/female	35	Central pontine and extrapontine myelinolysis	Bilateral	NA	
Karthik *et al*.[21]	52/male	52	Idiopathic	Bilateral	No improvement	
	34/female	33	Idiopathic	Bilateral	Partial improvement with clonazepam	
Lim and Seet[22]	50/female	50	Multiple abdominal surgeries	Bilateral	Improvement with botulinum toxin	
Schrader *et al*.[27]	78/female	75	Idiopathic	Bilateral	Deep brain stimulation of the posteroventral lateral part of the globus pallidus internus	She first developed the BDD. Later, orofacial dyskinesia appeared
Carecchio *et al*.[9]	72/female	72	Levodopa	Bilateral	Dopaminergic therapy was changed with partial improvement of the symptoms	Video available
Amin *et al*.[26]	47/female	47	Haemorrhoidectomy	Bilateral	Diazepam and diphenhydramine were prescribed with full recovery	
Bhidayasiri and Tarsy[28]	15/female	15	Spinal cord injury secondary to trauma	Bilateral	No management is described	Video available
Alshubaili *et al*.[29]	67/male	67	Idiopathic	Bilateral	Botulinum toxin with full recovery of BDD	
	61/female	54	Idiopathic	Bilateral	Botulinum toxin with full recovery of BDD	
	32/male	32	Idiopathic	Bilateral	Botulinum toxin with full recovery of BDD	
	59/female	52	Idiopathic	Bilateral	Botulinum toxin with full recovery of BDD	

Contd...

Table 2: Contd...

Author	Age (year)/sex	Age onset BDD (year)	Cause	Unilateral/ bilateral	Management	Comment
Valálik *et al*.[30]	36/female	29	Idiopathic	Bilateral	Deep brain stimulation of the posteroventral lateral part of the globus pallidus internus	
Gupta and Kushwaha[4]	23/male	23	Domperidone	Bilateral	Discontinuation of domperidone. Clonazepam and promethazine were given. A complete recovery after two days was observed	Video available
Kelly *et al*.[31]	64/male	64	Thyrotoxicosis	Bilateral	Cabimazole was started with improvement of BDD	The frequency of the abdominal movements was faster than commonly seen in BDD
Meyer *et al*.[32]	37/female	37	Pregnancy	Bilateral	BDD fully recovered with clonazepam and levetiracetam	Video available. Recurrent BDD in past pregnancies
Moreira *et al*.[25]	14/female	14	Cervical spinal cord injury	Bilateral	Chlorpromazine and column stabilization with Jewett vest and physiotherapy	
Rathore *et al*.[33]	12/male	12	Idiopathic	Bilateral	No improvement after several therapeutical choices	Patient presented with BDD 5 days after a diarrhea
Yeh *et al*.[34]	71/female	71	Quetiapine	Bilateral	Discontinuation of quetiapine showed improvement of BDD	
Al-Sibahee and Fawzi[35]	62/male	62	Idiopathic	Bilateral	NA	Video available. He denied further investigation
Aldabbour *et al*.[36]	40/female	40	Pregnancy	Bilateral	A fully recovery with clonazepam was observed	Recurrence of symptoms with further pregnancy
Vasconcellos *et al*.[37]	62/female	62	Haloperidol and risperidone	Bilateral	NA	Video available
Alshurem[38]	34/female	33	Idiopathic	Bilateral	Complete recovery with levetiracetam	Medical history of meningitis
Cavalcante-Filho *et al*.[39]	79/female	79	Levodopa	Bilateral	After 2 weeks of levodopa discontinuation and rasagiline starting, BDD completely improved	Video available
Cavdar *et al*.[40]	68/male	68	Prochlorperazine	Bilateral	Complete recovery 2 weeks after prochlorperazine discontinuation	Video available. Orobuccal stereotypic movements were noted
Dubey *et al*.[41]	62/female	62	Nonketotic hyperglycemia	Bilateral	BDD resolved with normoglycemia	Hemiballism was observed in the right upper limb
Durrani[42]	21/female	21	Appendectomy	Bilateral	Complete recovery after magnesium supplementation	BDD occurred 2 weeks after appendectomy, patient was misdiagnosed with functional disorder
Gure[43]	25/female	25	Pregnancy	Bilateral	Cesarean delivery in the 38th week of pregnancy due to induction without contraction	BDD started around the 22nd week of pregnancy. In her second pregnancy, she did not develop BDD
Hutabarat and Santosa[45]	42/female	42	Systemic lupus erythematosus	Bilateral	Valproate and clobazam were started with improvement of her BDD	Intranuclear ophthalmoplegia was noted
Rodriguez Navas *et al*.[23]	48/male	48	Huntington disease	Bilateral	No improvement	
Nigro *et al*.[44]	73/male	72	Progressive supranuclear palsy	Bilateral	BDD resolved with levetiracetam	
Ossella *et al*.[8]	14/female	14	Idiopathic	Bilateral	BDD improved with diazepam	Possible association with pancreatitis
Wong *et al*.[11]	54/male	54	Salbutamol	Bilateral	BDD improvement after discontinuation of salbutamol	Video available
Ablaza and Salonga-Quimpo[46]	7/female	7	Anti-tuberculosis medication	Bilateral	Carbamazepine was started with complete recovery	Video-EEG

Contd...

Table 2: Contd...

Author	Age (year)/sex	Age onset BDD (year)	Cause	Unilateral/ bilateral	Management	Comment
Bane *et al*.[16]	54/female	48	Cholecystectomy	Bilateral	Amitriptyline, otilonium bromide, pantoprazole, and chlordiazepoxide and clidinium were started. Patient present significant improvement of BDD	Video available
Dominguez *et al*.[14]	83/female	83	B12 vitamin deficiency	Bilateral	Significant improvement after B12 deficiency	
Khosama *et al*.[7]	47/male	46	Idiopathic	Bilateral	BDD full recovery with haloperidol and clobazam therapy	Video available. Facial tics
Rathmann *et al*.[24]	84/male	84	B12 deficiency	Unilateral	Improvement with B12 supplementation and clonazepam	Video available. BDD was not affected by sleep patterns. The patient also had vascular parkinsonism
	52/male	47	B12 lower limit	Bilateral	Improvement with B12 supplementation and clonazepam	Video available. Partial improvement after spinal anesthesia
	56/male	54	B12 deficiency	Bilateral	Improvement with B12 supplementation and clonazepam	Video available
Abraham *et al*.[17]	25/male	19	Idiopathic	Bilateral	A significant improvement with botulinum toxin was observed. Several therapeutical choices were attempted	Patient presented with acute respiratory failure due to worsening of BDD
Cabrera *et al*.[47]	67/male	67	Spinal cord injury	Bilateral	Self-recovery	Video available
Carus *et al*.[13]	24/female	24	Spinal cord injury	Bilateral	No improvement with propranolol and primidone	Patient also had explosive speech and disarticulation, head titubation, and tongue tremor
Divya *et al*.[48]	10/male	10	Salbutamol	Bilateral	Behavioral therapy and clonazepam were started	Functional stressor possibly influencing the development of BDD
Kaga *et al*.[49]	80/male	80	Droxidopa and amantadine	Bilateral	Droxidopa and amantadine were discontinued	Video available
Tafesse Mengesha *et al*.[15]	19/female	19	Pregnancy	Bilateral	Valproate and diazepam were started with delivery. The patient had a full recovery	Video available

BDD: Bely dancer's dyskinesia, EEG: Electroencephalogram, EMG: Electromyography, NA: Not available/not applicable

[Figure 1].[4,7-9,11,13-18,20,22-49] This condition was primarily defined as a peripheral-induced hyperkinetic movement disorder, but several cases of possible central causes were discovered.[38]

It should be taken into account that a recent history of spinal or head trauma may play a role in some cases in the development of BDD.[28] In this context, cervical disorders should be considered in the differential diagnosis when a patient with BDD has a history of recent trauma.

Central nervous system disorders may also play a role in the development of BDD. Roggendorf *et al*. reported a patient who experienced central and extrapontine myelinolysis (CPM/EPM) 5 months before acquiring BDD.[20] CPM/EPM was already associated with dysarthria, dysphagia, quadriparesis, oculomotor abnormalities, and locked-in syndrome, depending on the site of involvement in the pons.

Some medications have been linked to the development of BDD. Linazasoro *et al*. described BDD associated with long-term clebopride.[10] Carecchio *et al*. reported an elderly woman with Parkinson's disease who presented dyskinetic movements of her abdominal muscles after levodopa therapy.[9] In this way, these reports suggest a significant association of BDD with the dopaminergic system. Interestingly, BDD was related to both agonists and antagonists of postsynaptic dopaminergic receptors.

Another reason for BDD may be metabolic abnormalities. Durrani reported a case of BDD secondary to hypomagnesemia.[42] One of the possible explanations for this case is that total body magnesium deficiency can lead to increased acetylcholine release, causing

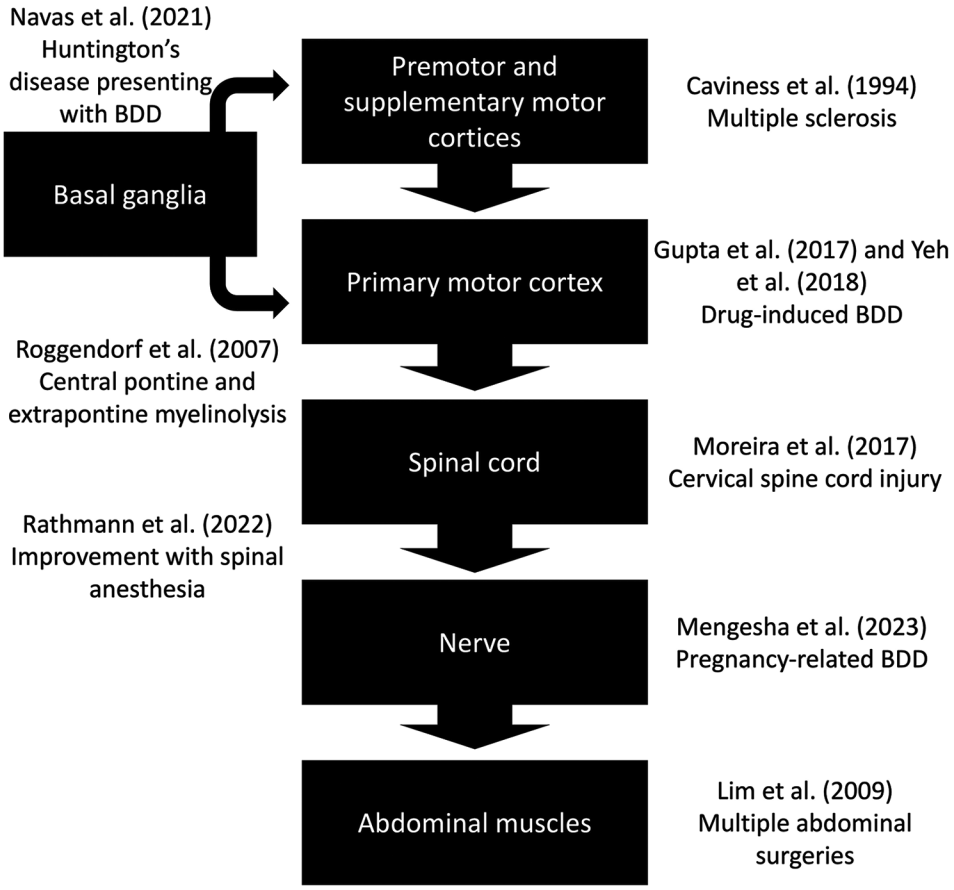

Figure 1: Proposed pathophysiology of belly dancer's dyskinesia. The references correspond to the possible lesions related to the development of belly dancer's dyskinesia. BDD: Belly dancer's dyskinesia

neuromuscular excitation and hyperactivity of alpha and gamma motor neurons. Dogan and Yuruten reported that low serum Vitamin B12 may cause spinal myoclonus.[50] Also, there are cases in the literature of spinal myoclonus with abnormal movements resembling BDD.[12] In this context, it is difficult to define the movement disorder in individuals with B12 deficiency who present with BDD. It is also important to consider that the deficiency of this vitamin is commonly associated with degenerative lesions in the dorsal and lateral columns of the spinal cord due to demyelination.[24]

BDD can occur in pregnant patients. The main explanation is that the pregnancy-related changes related to the uterus and vascular system lead to compressive effects on the thoracic cord and nerve roots, causing abnormal movements.[43] This theory can explain those pregnant patients presenting with BDD in the second and third trimester. However, this hypothesis is less likely to explain the presentation of BDD during the first trimester of pregnancy. It is possible that in the early stages of pregnancy, the development of BDD is related to an autoimmune response or even a disbalance in the electrolytes.[32] In BDD associated with pregnancy, many patients present with recurrence in a second pregnancy.[32,43]

BDD usually presents with two main characteristics: Fast or slow abdominal movements. BDD presenting with fast abdominal movements probably has myoclonic pacing, and some characteristics strongly suggest this hypothesis. First, abnormal abdominal movement improvement with benzodiazepines is a common feature of myoclonus.[51] Second, the worsening of the intensity and frequency of the movement with sympathomimetic medications is also a characteristic feature of myoclonus. It has been proposed that dose-dependent excessive stimulation of the catecholaminergic receptors of the central nervous system and skeletal muscles induces myoclonus.[52]

Clinical presentation

The abdominal wall movements in BDD are produced by variable combinations of rectus abdominis and oblique muscle contractions and accompanied by paraspinal and perineal muscle activity. The pattern of muscle contraction includes EMG bursts of 400–1,000 ms duration and superimposed jerky movements,

suggesting a dystonic character.[3] Lateralized abdominal movements that spread to thoracic and lumbar paraspinal muscles with stimulus sensitive myoclonus were described as BDD due to spinal reflex myoclonus. Undulating BDD can also be driven by an autonomous spinal generator, as suggested by the persistence of the dyskinesias during sleep and the lack of influence of descending inputs on the movements in a case of an intramedullary spinal tumor.[3]

An uncontrollable contorting or rolling movement of the abdomen wall or epigastrium, whether visible or not, is the most common manifestation of BDD. In this context, the easiest way to approach the abnormal movements of the abdominal musculature is to observe the umbilicus's movements, which commonly have a multidirectional movement in bilateral cases and unidirectional in unilateral cases. It is worth noting that there are only two cases of unilateral BDD in the literature.[1,24]

The majority of the reports in the literature suggest a progressive development of dyskinesia, but there are reports of abrupt onset, especially in spinal cord injury.[28] Furthermore, most patients do not have pain related to the movements. However, they are usually uncomfortable and can disrupt their sleep pattern, significantly influencing their daily life.

No cardiac abnormalities in the rate or rhythm have been reported with BDD, which helps differentiate this condition from diaphragmatic myoclonus.[53] Laboratory tests, neuroimaging, and electrocardiograms are within normal limits. BDD can be painless. However, that is not generally the case, as it is commonly accompanied by upper abdominal or lower chest wall pain or discomfort. Patients may also complain of dyspnea, even though arterial blood gas levels often stay within acceptable limits despite disturbed spontaneous breathing.[41] Normal oxygen saturation levels have been observed even if voluntary breathing is stopped in those individuals, probably due to autonomic control of breathing patterns and central regulation of respiratory rates by the medulla oblongata. Nevertheless, there was an atypical report of a patient with a long-term diagnosis of BDD who had an acute exacerbation of the abdominal muscle dyskinesia, leading to acute respiratory distress.[17]

BDD might be sporadic, lasting for a few minutes or extending to many hours. It could occur as an isolated or a recurring condition. While most cases show that BDD is episodic, there have been isolated reports of long-term continuous dyskinetic movements for several months.[29] BDD may also be the presenting sign of cardiac pathology, and individuals with BDD may report excruciating pain in the left arm and chest wall that could be a sign of coronary artery syndrome.[1] In these situations, cardiac enzymes and electrocardiography should be requested to rule out cardiac conditions. These symptoms' resemblance to those of acute myocardial infarction might confuse patients and their clinicians, delaying proper primary care and management.

Diagnostic tools

An appropriate medical history should be obtained from the patient. The majority of the individuals will have a history of recent medication changes, spinal cord injury, or abdominal surgery. In this context, diagnostic tools are requested to rule out other causes that could mimic BDD symptoms.

Laboratory tests should be performed with special attention to thyroid profile,[31] Vitamin B12 levels,[14] and glycemic levels with urine ketones.[41] Brain and spinal neuroimaging should be obtained to exclude possible demyelinating diseases,[18] structural damages, or signs of possible spinal cord lesions. Electrodiagnostic studies with electroencephalogram (EEG) and EMG should be performed to evaluate the characteristics of the abnormal movement and the "*Bereitschaftspotential*."

Fluoroscopy should be requested when the patient exhibits signs of possible diaphragmatic myoclonus, as the patient's bones, soft tissues, and air masses are shown to move in real-time on this diagnostic modality.[54] The diaphragm's movement patterns will be revealed by analysis of these structures during fluoroscopy. Even if only unilateral signs are present, fluoroscopy will reveal both bilateral and unilateral diaphragmatic myoclonus. Myoclonic activity can be demonstrated by an electromyogram of the diaphragm when diaphragmatic myoclonus is suspected but not evident.[55]

The Koepke approach is a good way to assess the diaphragm activity using EMG. Monopolar electrodes are placed through the chest wall and into each hemidiaphragm while utilizing this procedure.[56] Therefore, an independent evaluation of each diaphragm can be performed with this technique.

Volume-time spirometry can be used as another method of diaphragmatic myoclonus diagnosis. This method of diagnosis offers proof of the dual respiratory cycles typical of diaphragmatic myoclonus. A slow rate of 10–15 breaths/min and a tidal volume of 1,500–2,000 ml was identified in one case of diaphragmatic myoclonus diagnosed using this technique. A second wave was also discovered in this patient, with a smaller, quicker rhythm of 180 breaths/min and a tidal volume of 200 ml.[57] The diagnosis of diaphragmatic myoclonus can be supported by performing plethysmography. Hoffman *et al*. described cases of diaphragmatic

myoclonus associated with coronary artery bypass surgery in which plethysmography was performed, and the discontinuation of the postoperative ventilator support was difficult.[58] When no obvious symptoms exist, plethysmography might inadvertently identify diaphragmatic myoclonus. Adams *et al*. reported the unexpected discovery of diaphragmatic myoclonus in three newborns undergoing plethysmography to monitor apnea.[59]

Management and treatment

Most of the therapeutical choices for BDD in the literature are based on expert opinions and case series. There are some standard approaches for those drug-induced BDD. In these specific situations, the offending drug should be discontinued. Some authors reported the prescription of antiseizure medications and benzodiazepines if the patient did not improve after the discontinuation of the offending drug.[12] It is worth mentioning that the improvement with benzodiazepines may suggest myoclonic rather than dyskinetic movements.

Supplementation should be performed when a metabolic deficiency is found. There are some cases of Vitamin B12 deficiency or Vitamin B12 levels near the lower levels that the supplementation showed to significantly improve the patient's motor symptoms.[24]

Cases resistant to the above approaches should be managed with antipsychotics.[7] However, it is important to take into account that the dyskinetic symptoms could worsen, especially in individuals whose BDD was associated with medications related to the dopaminergic pathway.[37] Furthermore, a significant number of adverse effects are usually reported with antipsychotics. In these cases, botulinum toxin injections can be attempted. Alshubaili *et al*. published a case series of patients with idiopathic BDD who had complete recovery with botulinum toxin, especially after the second session of injections.[29]

In individuals who do not have any improvement with oral medications and ultrasound-guided botulinum toxin injections, deep brain stimulation can be attempted. There are two cases of deep brain stimulation targeting the globus pallidus internus with significant improvement of the dyskinetic motor symptoms.[27,30]

Related disorders and differential diagnoses

Diaphragmatic myoclonus

Diaphragmatic myoclonus is also known as Leeuwenhoek's disease, diaphragmatic chorea, palpitations of the diaphragm, convulsions of the diaphragm, diaphragmatic cramps, rhythmic spasms of the diaphragm, clonic spasms of the diaphragm, tremor of the diaphragm, diaphragmatic rumble, flutter fibrillation of diaphragm, diaphragmatic myoclonia, diaphragmatic tic, seesaw movements of the thoracic wall of noncardiac origin, pseudopulsations, or respiratory myoclonus and diaphragmatic flutter.[3] Interestingly, the first report of diaphragmatic myoclonus was written in 1723 by Anthonie van Leeuwenhoek, who developed symptoms of possible diaphragmatic myoclonus himself. He described it as a "violent movement" of the diaphragm.[60]

The clinical presentation of diaphragmatic myoclonus is characterized by forceful movements that can lead to abdominal wall motion, compromise gas exchange, or cause gastro-esophageal problems. This condition is often manifested by jerky abdominal movements that are described as epigastric pulsations or oscillations, or thoracic jitter. It may also produce an audible succussion splash or gurgling noises, and a shuffling precordial murmur may be auscultated. The fluttering diaphragm is easily visible on fluoroscopy, and it contracts at frequencies of 0.5–8.0 Hz in either inspiration or expiration. The recruitment of intercostal, scalene, paraspinal, and abdominal muscles, or epiglottis is also described. Breath holding and deep inspiration can suppress the movements or decrease their frequency.[3]

The diaphragm quickly contracts and relaxes, which causes the "fluttering" appearance. Therefore, some authors classify these movements as a myoclonus. Rigatto and de Medeiros studied 42 patients with diaphragmatic myoclonus, and they found that the rate of diaphragmatic contraction ranges from 35 to 480/min, with an average rate of 150/min.[54] The patient's bed may occasionally tremble due to this fluttering when it happens quickly enough.

There are three commonly reported respiratory patterns with diaphragmatic myoclonus: Tachypnea, diaphragmatic flutter with apneustic respiration, and dual respiratory pattern. In this context, the dual respiratory pattern is characterized by a high-frequency fluttering respiration superimposed upon a much slower, normal respiratory pattern. In some studies, myoclonus was documented during just one of the two stages of breathing. Nonetheless, in most cases, it occurs throughout both inspiration and expiration, and an inspiratory myoclonus has been documented in all of these individuals. Usually, fluttering respiration is not suppressible when patients attempt to hold their breath. The diaphragmatic myoclonus, which occurs at a high frequency, overlaps regular breathing at a much lower frequency in this dual respiratory pattern.[57] Tamaya *et al*. have proposed that the dual respiratory pattern that can concomitantly occur with diaphragmatic myoclonus may be caused by the different inputs received by the respiratory center, which may result in both a high and slow breathing rate simultaneously.[61]

EMG tests have shown that these movements result from myoclonic contractions of one or both hemidiaphragms. Bilateral involvement is experienced by about two-thirds of the patients with diaphragmatic myoclonus. The left hemidiaphragm is where unilateral diaphragmatic myoclonus most frequently occurs. Dyspnea is a common complaint with diaphragmatic involvement.[62]

Diaphragmatic myoclonus is synchronous with the systole in individuals with phrenic nerve irritation.[53] Also, these individuals commonly complain of pain associated with the fatigue of the abdominal muscle musculature and characteristic acute chest pain of cardiac origin. Moreover, the peripheral nerve lesions are related to abnormal muscle movements that usually do not subside with the sleep pattern and could play a role in the pathogenesis of this condition.

When pharmaceutical treatments fail to control the symptoms of unilateral diaphragmatic myoclonus, phrenic nerve block should be attempted. The symptoms usually immediately disappear after the procedure.[63] Additionally, through the course of neural regeneration, it has been demonstrated that this technique enables a subsequent recovery to typical diaphragmatic function. Pulse oximetry and pulmonary function tests are usually within normal limits until the phrenic nerve's regeneration is complete. The surgical procedure may contribute to respiratory difficulties and decreased lung volumes in certain cases. It is important to ensure that the phrenic nerve is intact, allowing the hemidiaphragm to function properly so that the individual can sustain oxygenation at acceptable levels for rest and exercise. It's interesting to note that the signs generally disappear after diaphragmatic function returns to normal, corroborating this pathophysiological hypothesis.

Cardiac conditions

Acute chest pain or discomfort is the most frequent complaint when the phrenic nerve is the culprit of the syndrome, which can cause a systolic diaphragmatic myoclonus.[53] There may also be a relation between cardiomegaly and rheumatic heart disease, prior lung disease or surgery, and irritation of the phrenic nerve or diaphragm from disorders including pleurisy and peritonitis.[60]

Individuals presenting with chest pain radiating to the left upper limb should have a full workup for pain from cardiac origin. BDD and diaphragmatic myoclonus are diagnoses of exclusion in these situations. In this way, BDD and diaphragmatic myoclonus should be considered a potential etiology in patients complaining of thoracic pain with negative diagnostic workup for coronary artery disease with a low pretest probability of cardiovascular conditions.

Aortic conditions

A common finding in thin patients, when they lie flat, is abdominal pulsations. This finding can be observed in healthy individuals, and it is called "false abdominal aortism" or "aortism." However, this can also occur in individuals with aortic aneurysm, in which there is an association between the size of the abdominal aortic aneurysm and the probability of abdominal pulsation.[64]

The fact that the abdominal pulsation occurs simultaneously with the cardiac systoles can help differentiate it from BDD. Furthermore, the clinician in the abdominal palpation can feel a mass corresponding to the aortic aneurysm, which is not observed in patients with BDD. Moreover, abdominal aorta abnormalities usually affect only the epigastric region, but BDD usually involves the entire abdomen.

Truncal dystonia

Truncal dystonia is characterized by dystonia affecting paraspinal, abdominal, and chest muscles.[65] BDD can be differentiated from abdominal dystonia by some features. First, abdominal dystonia rarely affects only the abdominal muscles. The most common presentation of abdominal dystonia is a mixture of different degrees of involvement of the truncal muscles. Second, the movements of abdominal dystonia are usually more sustained than BDD's, leading to significant trunk movement. Furthermore, abdominal dystonia is a postural flexion movement semiologically different from BDD.

Abdominal motor seizures

The fast abdominal, diaphragmatic, and epigastric spasms of BDD can mimic seizures. Noteworthy, seizures presenting with isolated abdominal movements were rarely reported. To be more specific, there are 17 cases of seizure-induced isolated abdominal movements in the literature [Table 3].[66-76] Localization of the epileptogenic foci and symptomatogenic zones was reported in multiple sites, suggesting a possible pathway. In this way, the possible seizure foci involve the mesial brain regions spreading to the mesial cortex and, after, going to the primary sensorimotor region corresponding to the trunk.

The most significant differentiation between these specific types of seizure affecting abdominal musculature and BDD is the seizure activity in the EEG. Another significant finding found in almost all individuals is a previous history of seizures. However, electrodiagnostic studies should be requested to differentiate BDD from seizure because some individuals only presented abdominal symptoms without any other focal sign or impairment of consciousness.

Table 3: Cases of abdominal movements secondary to seizures reported in the literature

Reference	*n*	Age, sex	Seizure location	Seizure type
Nathanson *et al*.[66]	4	63 (mean), 3 males +1 female	Brainstem	Focal motor with or without loss of awareness versus status epilepticus
Matsuo[67]	3	42.3 (mean), 1 male and 2 females	Contralateral parietal	Focal motor
Rosenbaum and Rowan[68]	1	65, male	Right frontoparietal	FMSE
Chalk *et al*.[69]	1	66, male	Right parasagittal	FMSE
Fernández-Torre *et al*.[70]	1	77, female	Right frontal	FMSE
Dafotakis *et al*.[71]	1	62, male	Unknown	FMSE
Tezer *et al*.[72]	1	25, female	Left mesial parieto-occipital	FMSE
Oster *et al*.[73]	1	59, male	Left mesial frontoparietal	Focal motor onset with and without secondary generalization
Ribeiro *et al*.[74]	2	72 (mean), 2 males	Left occipital; right occipital	FMSE
Aljaafari *et al*.[75]	1	26, male	Left mesial parietal	Focal motor onset with and without secondary generalization
Lizarraga *et al*.[76]	1	77, male	Left mesial parietal	Focal motor onset with and without secondary generalization

FMSE: Focal motor status epilepticus

Propriospinal myoclonus

Propriospinal myoclonus (PSM) is a movement disorder that affects axial muscles, which usually occurs in the trunk, hips, and knees in a fixed pattern. The term propriospinal refers to neurons that originate and have axons that terminate within the spinal cord, and which help coordinate motor activity between spinal segments, in contrast to the supraspinal projections such as corticospinal, rubrospinal, reticulospinal tract, and vestibulospinal tracts.[77]

Due to specific temporospatial distribution, BDD can be distinguished from PSM with electrodiagnostic studies. The characteristic findings of the EMG in the idiopathic PSM are slow conduction velocity (≤15 m/s), short burst duration (<300 ms), no synchronicity, and absent BP. Another clinical feature particularly observed in individuals with PSM is that lying supine and the sleep-wake transition period can exacerbate myoclonic activity, especially in those cases of idiopathic rather than functional origin.[78]

In a review of the literature that identified 179 patients with PSM (55% male), the mean age at onset was 43 years (range: 6–88 years) and a functional movement disorder was diagnosed in 104 (58%) cases. In 12 cases (26% of reported secondary cases, 7% of total cases), a structural spinal cord lesion was found. Because it is rare for a structural lesion to underlie PSM, the authors recommended polymyography to assess recruitment variability combined with a BP recording in all cases.[78]

Functional

BDD is an uncommon condition that is challenging to diagnose. The overlap with several neuropsychiatric conditions makes the diagnosis of this disorder difficult. Furthermore, even if a functional component is not identified, the symptoms of BDD could resemble those of anxiety-induced hyperventilation. Although abnormal abdominal movements are commonly associated with functional movement disorders, this diagnosis should be made after consideration of other secondary causes of BDD.[79] Clinicians must carefully investigate already reported causes before concluding that the condition has a psychosomatic origin.

In the neurological examination of patients with possible BDD, the examiner should notice the effects of distractibility, entrainability, and suggestibility in the presence of abnormal abdominal movements.[79]

One of the possible methods to differentiate functional from involuntary movements is to perform back-averaging of EEG-EMG, which is useful to assess the presence of BP. The BP is an EEG potential that precedes the onset of individual movements. In this context, it is particularly useful for identifying functional jerks. It is also not a measure of voluntary activity since functional movements are not voluntary.

It is worth mentioning that continuous movements will not show the BP, since there are no onsets, so the main evaluation should be at the beginning of the movement.[80]

Another possible clinical clue for suspecting BDD is no improvement of symptoms with standard therapy, which can suggest this uncommon disorder. Considering this fact, it is possible that four of the cases reported by Caviness *et al*.,[18] one of the cases reported by Karthik *et al*.,[21] and one case reported by Rathore *et al*.[33] were functional disorders.

However, it is important to notice that lack of response to medication is not the best strategy to diagnose a functional disorder. Nevertheless, in a narrative review, it is difficult to establish clinical diagnostic criteria for this condition since it is not known the real number of patients with BDD given the lack of electrophysiological

studies performed in these reports. We recognize that back‑averaging of EEG-EMG is probably the most useful diagnostic tool to identify functional movement disorder (FMD) in patients with suitable symptoms. Additional studies must be performed in the future with EEG-EMG to better characterize clinical presentation of these disorders.

Other uncommon movement disorders

Other uncommon movement disorder that should be included in the differential diagnosis include scar dancing syndrome, which is defined as a peripheral trauma-induced involuntary hyperkinesia around a surgical incision. According to Yang *et al*., scar dancing syndrome is characterized by hyperkinesia around the surgical scar, which may or may not be painful, affects muscles right below the scar in long surgical incisions, and usually occurs less than a year after the surgery.[81]

Another peripherally induced movement disorder is dancing dorsal quadrilaterals following surgical injury. In this context, upper spine instrumentation can cause dorsal afferent nerve injury leading to prolonged neuropathic pain and abnormal movements. This can result in the dancing dorsal quadrilaterals syndrome, characterized by repetitive, writhing, and jerky movements affecting trapezius and rhomboids muscles. These movements disappear when lying down or with sensory stimulation, voluntary muscle activation, and sleep.[82]

Another condition is "jumpy stump syndrome," which is defined by dystonic, choreiform, and myoclonic movements that occur in the amputated limb. These patients may also experience associated neuropathic pain, and this condition may begin immediately after initial amputation or years later. Peripheral nerve damage associated with amputation and neuroma formation seem to be involved in the pathogenesis, but this may also be a subtype of spinal myoclonus or conversion disorder elicited by psychological or emotional trauma.[83]

Limitations and future perspectives

There are several limitations in the literature regarding BDD. Most of the reports did not differentiate between diaphragmatic myoclonus and BDD, leading to misunderstandings of the characteristics of these different pathologies. In the present study, only articles presenting BDD cases were included. However, most reports did not perform electrodiagnostic or pulmonary studies to differentiate from diaphragmatic myoclonus. Another significant drawback regarding the BDD is that due to the rarity of this condition, most of the knowledge comes from case reports.

Future studies should clearly describe the phenomenology of the movement disorder, including factors that improve or worsen the abnormal movement. Furthermore, further investigation with electrodiagnostic studies is essential to evaluate motor cortical potential. Moreover, the authors should include all the parameters found in the EMG. Neuroimaging, cardiovascular, and pulmonary function tests should be performed to rule out other more common clinical conditions with similar presentation.

Conclusion

BDD is an uncommon condition that frequently mimics several other disorders. Based on the current understanding of the cases already reported in the literature, there is a phenomenological difference between BDD and diaphragmatic myoclonus, but it is difficult to distinguish these two entities in the literature due to lack of uniformity of terminology and EEG-EMG testing in previously published reports and also in clinical practice due to their similar presentation. BDD might be mistaken for a coronary artery disorder, functional disorder, hyperventilation, seizures, and tremor disorder. The complex mixture of possible etiological factors makes the diagnosis even more challenging. A detailed patient history and careful physical examination are required to diagnose BDD correctly and ensure that the appropriate course of therapy is taken. Not all individuals with this syndrome will have a similar response. Further studies should assess the efficacy of different therapies and the characteristics related to the main therapeutical choices.

Author contributions

JR, AC: Concepts, design, definition of intellectual content, literature search, data acquisition, data analysis, statistical analysis, manuscript preparation, manuscript editing, manuscript review and guarantor; NV, IT, VB: Concepts, literature search, data analysis, manuscript preparation, manuscript review and guarantor.

Ethics approval and consent to participate

We have read the Journal's position on issues involving ethical publication and affirm that this report is consistent with those guidelines.

Data availability statement

Data sharing not applicable to this article as no datasets were generated and/or analyzed during the current study.

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Conflicts of interest

There are no conflicts of interest.

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