Spectrum of Conversion Disorders: Clinical Perspective for a Neurologist

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ABSTRACT
Conversion disorders are a borderland between psychiatrist and neurologist. These are known from ancient time and witnessed a variety of nomenclature and theories. Manifestations of conversion disorders are widely variable and may range from psychogenic movement disorders, psychogenic sensorimotor deficit to nonepileptic psychogenic seizures. Though advancement in diagnostic techniques helps in diagnosis of a number of neurological disorders, still diagnosis of conversion disorders is mostly clinical. For a neurologist, it is important to know breadth of conversion disorders and salient clinical features to differentiate from organic disorders to avoid fallacious diagnosis and its serious consequences. Treatment options include psychotherapy, hypnotic therapy, pharmacotherapy and recently transcranial magnetic stimulation. All patients of conversion disorders may not have milde course.

Keywords: Conversion disorder, Neurologist, Psychiatrist

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INTRODUCTION
Conversion disorders witnessed a colorful history for long time. Prior to 16th century, the hysteria was linked with aniliness of the uterus or to explanations such as witchcraft or demonic possession. By the early 17th century, psychological basis of hysteria was postulated. Later, Freud suggested that the emotional charge of painful experiences would be consciously repressed as a way of managing the pain, but this emotional charge would be somehow “converted” into the neurological symptoms. Till mid of last century, psychological theories were well established for conversion disorders. Recently, newer imaging modalities provide new insight to understanding of neurobiological basis of conversion disorders. In DSM-IV TR, conversion disorders have been kept as a different category. Conversion disorders may have varied manifestations ranging from psychogenic movement disorders, nonepileptic psychogenic seizures to sensorimotor deficit. In an outpatient clinic, they may mimic a number of neurological disorders. Without knowing the spectrum of these disorders, they may be misdiagnosed, and can lead to wrong diagnosis and therapies. On other hand, some of neurological disorders may mimic conversion disorders and can lead to delay in diagnosis and further serious consequences. It is very important to know spectrum of conversion disorders and differentiation from organic neurological disorders. Here, in this article we have tried to highlight the important conversion disorders that can help in day to day neurology practice.

Definition and DSM IV Criteria of Conversion Disorders
Conversion disorder is a motor-sensory deficit after a stressful event without an organic etiology. Historically, conversion disorder is described as patient exhibiting “la belle indifference,” a characteristic lack of normal concern about their deficit. Conversion disorders may be confused with somatization disorder; however, conversion disorder is distinguished by its relatively acute nature and narrower symptom complex. The American Psychiatric Association DSM-IV-TR defines diagnostic criteria for a conversion disorder (Box 1).

Dimension of Conversion Disorders
Conversion disorders may have varied manifestations ranging from motor or sensory deficit to psychogenic movement disorders to psychogenic sensorimotor deficit to nonepileptic psychogenic seizures. Though advancement in diagnostic techniques helps in diagnosis of a number of neurological disorders, still diagnosis of conversion disorders is mostly clinical. For a neurologist, it is important to know breadth of conversion disorders and salient clinical features to differentiate from organic disorders to avoid fallacious diagnosis and its serious consequences. Treatment options include psychotherapy, hypnotic therapy, pharmacotherapy and recently transcranial magnetic stimulation. All patients of conversion disorders may not have milde course.

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### Psychogenic Movement Disorders

Psychogenic movement disorders are amongst the most frequent psychogenic symptoms. Psychogenic movement disorders include dystonia, tremor, myoclonus, tics, hemiballismus, chorea, parkinsonism and a host of bizarre gait and stance disturbances. Psychogenic disorders that eventually turned out to be a physical illness have been reported in 6-30% of the patients. The diagnosis of idiopathic movement disorders can be done just by history and physical examination. Absence of a specific diagnostic laboratory test or any neuroimaging findings makes idiopathic movement disorders more susceptible to wrong diagnosis or its delay in diagnosis. To further complicate the matter, organic neurologic disease has been reported in as many as 28% of psychogenic neurologic disorders.

### Diagnosis of Psychogenic Movement Disorders

Diagnosis of psychogenic movement disorders must be made by a neurologist based on neurologic observations, though psychiatric analysis is essential. A comprehensive history and neurological examination as well as appropriate diagnostic studies (including MR imaging of the neuraxis, serum copper and ceruloplasmin levels, thyroid function test, parathyroid hormone or cerebrospinal fluid analysis) should be performed in proper clinical setting to exclude an organic etiology.

Fann and Williams proposed four levels of certainty for the diagnosis of psychogenic dystonia which can be applied to all types of movement disorders which are summarized in Table 1. Other diagnostic criteria have been proposed by Shill-Gerber and revision of Fann-Williams criteria by Gupta and Lang. The Shill-Gerber criterion haslacune as it gives more importance towards historical information rather than clinical characteristics of movement disorders and so has been criticized. Gupta and Lange proposed the revision of Fann-Williams criterion by making a new category of clinically definite by merging documented and clinically established categories and laboratory supported definite.

### Levels of certainty for the diagnosis of psychogenic movement disorders (PMD)

| Level               | Criteria                                                                 |
|---------------------|--------------------------------------------------------------------------|
| 1. Documented PMD   | Movements are relieved by psychotherapy, psychological suggestion, administration of placebos and the patient must be witnessed to be free of symptoms when supposedly unobserved. |
| 2. Clinically established PMD | Movements are inconsistent over time or incongruent with the classical symptomatology (i.e. a patient complaining of posturing of the limbs resists passive and active movement but easily grooms himself daily). In addition, one or more of the following present: other neurological signs that are definitely psychogenic (false weakness or sensory finding and self inflicted injuries), multiple somatizations or a documented psychiatric illness. |
| 3. Probable PMD     | The movements are inconsistent or incongruent with the classical disorder but other features in support of psychogenicity are lacking. |
| 4. Possible PMD     | A suspicion for a psychogenic basis for the movements is based only on the presence of an obvious emotional disturbance. |
Knowledge of clinical features of organic dystonia is helpful
to differentiate it from psychogenic dystonia. Features like
sudden onset, fixed posture since begging, excessive pain, associated bladder/bowel symptoms, response to placebo, lack of
“Gesteantagoniste” and other associated functional motor deficit
or psychogenic sensory loss favor psychogenic etiology of dystonia.
Over diagnosis of psychogenic dystonia should be avoided as these
have been reported only in 2.6% of the dystonias in one study.8

**Psychogenic Tremor**

Tremor is defined as a rhythmic, bidirectional oscillating movement
owing to contraction of antagonist muscles that may occur at
rest, with posture, or with movement. Psychogenic tremors have
been reported to be the most common type of PMD, accounting
for approximately 50% of the patients.13 Difference between
organic and psychogenic tremor is summarized in Table 2.12

| Features                  | Organic tremor                      | Psychogenic tremor                      |
|---------------------------|-------------------------------------|----------------------------------------|
| Onset                     | Insidious                           | Sudden                                 |
| Postural variation        | Amplitude varies with different     | Amplitudes remain same in all          |
|                           | postures                            | postures                               |
| Clinical inconsistencies  | No                                  | Yes                                    |
| Distractibility           | May worse with distraction          | Lessens                                |
| Attention                 | May reduce the amplitude            | Increased                               |
| Changing pattern          | Rare                                | Usual                                  |
| Spontaneous remission     | Rare                                | Usual                                  |
| Response to anti-tremor   | Yes                                 | No                                     |
| drugs                     |                                     |                                        |
| Remission with psychotherapy | No                              | Yes                                    |

**Psychogenic Myoclonus**

Myoclonus is defined as sudden, brief, shock-like movement
casted by either muscle contraction or by lapses in posture.
Psychogenic myoclonus have been reported as the most common
type of nonorganic movement disorder, accounting for 8.5% of
all myoclonus and 20.2% of all psychogenic movement disorders
in one movement disorders clinic.14 Features of Psychogenic
myoclonus are summarized in Box 5.14

**Psychogenic Parkinsonism**

Parkinsonism is a symptom complex of resting tremor, rigidity,
bradykinesia, and impaired postural reflex. The clinical presentation
of parkinsonism may vary and no definitive diagnostic test exists.
Some salient features are highlighted in Table 3 which may help
to differentiate psychogenic parkinsonism.15 This differentiation
is challenging and requires considerable experience.

**Psychogenic Paralysis**

Psychogenic paralysis is characterized by loss of motor and
sensory function subsequent to a psychiatric stressor.16 Associated
symptoms can include loss of vision or hearing, pseudo-seizures,
ataxia, and genitourinary and gastrointestinal dysfunction.17 The
severity of motor symptoms generally exceeds that of sensory loss;
rectal tone and reflexes are usually retained.18 In contrast, in a small
study it was seen that diminished rectal tone, diminished rectal
sensation, or diminished deep tendon reflexes were present in 12 of
14 patients with psychogenic paralysis. However, bulbo cavernous
reflex was retained in all.19 Rectal tone, rectal sensation and deep
tendon reflexes may be subjected to a degree of voluntary control.
The bulbocavernous reflex is involuntary. These facts explain the
findings. Recovery of psychogenic paralysis is rapid and complete,
with more than 90% of patients showing complete recovery within
1 month.15,18

The diagnosis of a psychiatric disorder is of exclusion. When
examining a patient with paralysis, clinical findings like normal
reflexes, normal rectal sensation and tone, normal bulbocavernous
reflex should raise the suspicion for a psychologic etiology.
Appropriate investigations should be done to exclude any organic
etiology.

**Physical Examination for Psychogenic Paralysis**

There are number of physical examination maneuvers that have
been validated to aid in the identification of psychogenic paralysis
(Table 4).

**Spinal Injuries Center Test**

The Spinal Injuries Center (SIC) Test was described by Yuen et al for
the evaluation of conversion disorder with motor deficit involving
the lower extremities.20 In this test the patient is asked to be in
the supine position and the examiner passively lifts the patient’s
knees to a flexed position with feet flat on the bed. The examiner
releases the patient’s knees; if the patient is able to maintain the
flexed position, he is considered to be SIC test-positive. If the patient
has severe paralysis, he won’t be able to maintain his knees in the
flexed position, and the leg on the paretic side will spontaneously
fall into the flaccid extension position. In this scenario, the SIC test
result is considered negative.

**Barre’s Sign**

Barre’s sign may be used for the evaluation of hemiplegia. The
patient is placed in the prone position with knees flexed at right angles. The examiner releases the knees and asks the patient
maintain the position; the limb on the affected side will fall
into extension in the event of true paralysis. In a patient with
psychogenic hemiparesis, the affected limb will extend rapidly
without contraction of the hamstrings.20
Table 3: Clues suggesting psychogenic parkinsonism

| Feature               | Psychogenic parkinsonism                                      | Organic parkinsonism                                     |
|-----------------------|--------------------------------------------------------------|---------------------------------------------------------|
| Onset                 | Often sudden                                                  | Gradual                                                 |
| Course                | Static, maximum disability early                             | Progressive                                              |
| Laterality            | Dominant side most affected                                  | Any side may be affected                                 |
| Tremor                | May be of any type (Rest, postural, action)                  | Usually resting tremor                                   |
| Variability           | Reduces with distraction/concentration, increases with attention | Worsens with concentration                              |
| Rigidity              | "voluntary resistance", cogwheeling absent, may decrease with distraction | Cog wheeling present                                    |
| Bradykinesia          | No true fatiguing, marked slowness, bizarre features.        | Worsens with time, may cause significant fatiguing       |
| Gait                  | Atypical, arm held stiffly at side, analgic if pain associated.| Short, shuffling gait, tendency to lean forward          |
| Postural stability    | Extreme or bizarre responses to minimal displacement          | Initially minimal, progress with time                    |
| Other neurologic features | False weakness, nonanatomic sensory loss                 | Usually absent                                           |
| Psychiatric symptoms  | Varied, evident but accurate definition not always possible   | May have depression as a part of disease                 |
| Secondary gain        | Litigation, compensation are usually present                 | Usually absent                                           |

Table 4: Different clinical tests for psychogenic motor deficit and their clinical implications

| Clinical tests                  | Clinical implications                                      |
|---------------------------------|---------------------------------------------------------|
| Spinal Injuries Center Test     | For psychogenic lower limbs weakness                     |
| Barre’s Sign                    | For psychogenic hemiplegia.                             |
| Babinski Thigh-Trunk Test       | For psychogenic hemiplegia and paraplegia.              |
| Hoover’s Test                   | For psychogenic hemiplegia. As with Barré’s test, Hoover’s test is of no use in the evaluation of paraplegia. |
| Monrad-Krohn’s cough test       | For psychogenic arm monoparesis.                        |
| Double-crossed-arm pull test    | For psychogenic arm monoparesis.                        |
| Make-a-fist test                | For psychogenic wrist drop.                             |
| Reversed hands test             | For psychogenic hand paralysis.                         |
| Backward displacement test       | For psychogenic foot drop.                              |
| Raimiste’s leg adduction-abduction synkinesis | For psychogenic hemiplegia and leg monoparesis |

Babinski Thigh-Trunk Test

To make an attempt to sit from a supine position, the natural tendency of the lower extremities is to tense in extension by contracting the gluteal muscles so as to provide a stable base. In true hemiplegia, the affected lower extremity will involuntarily exhibit thigh flexion when attempted to sit, because the gluteal muscles are weak and has no extensor tone. In paraplegia, both extremities will exhibit thigh flexion. The patient with psychogenic paralysis will typically maintain both lower extremities in extension position when he attempts to sit, reflexively firing the gluteal muscles.  

Hoover’s Test

In the evaluation of hemiparesis, asking the patient to be in supine position, the examiner lifts the patient’s legs slightly up from the examination table. While the examiner puts his hands below the heels, the patient is asked to lift the affected extremity; the absence of downward heel pressure in the contralateral (uninvolved) limb is indicative of apathy of intent (Fig. 1). As with Barré’s test, Hoover’s test is not useful in the evaluation of paraplegia.  

Monrad-Krohn’s Cough Test

To identify psychogenic paralysis of the arm, the examiner stands behind the patient and grasps the bilateral latissimus dorsi muscles between the thumb and fingers of the right and left hands. The examiner asks the patient to cough forcefully. Both latissimus dorsi muscles synkinetically contact strongly, thus establishing the integrity of the motor pathway through the brachial plexus.  

Fig. 1: Raimiste’s leg adduction-abduction synkinesis
Double Crossed Arm Pull Test
When required to use both sides simultaneously and unexpectedly, the psychogenic patient will generally inadvertently contract the putatively weak side along with the normal side. Patient remains upright with forearms crossed and flexed. If the patient’s arm is completely paralyzed, it will be held in place. While holding the patient’s forearm, patient asks to pull back strongly away from examiner. Usually the patient braces the paretic and nonparetic arms when pulling back.

Make a Fist Test
To distinguish a psychogenic wrist drop from a radial palsy, the patient is asked to extend the arm out straight. The putatively paralyzed wrist hangs limply. The patient is instructed to suddenly make a strong fist. If intact, the putatively paralyzed wrist extensors automatically cock the hand into the “anatomic position” when the patient makes a fist.

Raimiste’s Leg Adduction-abduction Synkinesis
With the patient recumbent, place hands on the patient’s knees and the patient to adduct the legs together strongly. Examiner has to hold hands in place in opposition to the patient’s action. The patient usually braces the putatively paralyzed limb in automatic opposition to the action of the intact limb (Fig. 2). Similarly, patient is asked to press the legs apart strongly against resistance. The putatively paralyzed limb usually will abduct in automatic position.

Difference between psychogenic and organic paraplegia is discussed in Table 5.

Pharmacological Intervention
Hypnotic sedatives have been used in the identification of psychogenic paralysis. The resolution after the administration of Sodium amytal has been reported in many case studies. This short-acting barbiturate has sedative, hypnotic, and anticonvulsant properties. The intravenous administration of 8 mL of 2.5% solution administered over 15 to 20 minutes has been shown to provide an immediate, effective, and lasting cure of psychogenic paralytic symptoms. It is hypothesized that dissociative medication and serial interviews will facilitate the recognition and resolution of symptoms by resolving subconscious (unrealized) conflicts.

Nonepileptic Psychogenic Seizure (NEPS)
Nonepileptic psychogenic seizure has been defined as psychologically determined clinical event that resemble epileptic attack but is not associated with paroxysmal physiologic cerebral dysfunction. NEPS may constitute up to 20% of patients at epilepsy referral centers. To diagnose and treat NEPS is still challenging to the physicians.

Etiology
NEPS may be due to reaction to a specific event. It may an expression of dependency or a wish or may show some attention-seeking needs. Preceding history of sexual or physical abuse can be present. Depression is common and suicide attempts have been reported. Tongue biting and other self-injurious behavior as well as incontinence during NEPS episodes predicts a higher likelihood of suicide attempts.

Table 5: Differentiation of psychogenic and organic paraplegia

|                        | Nonorganic paraplegia                                                                 | Organic paraplegia                                                                 |
|------------------------|--------------------------------------------------------------------------------------|----------------------------------------------------------------------------------|
| Onset                  | Usually sudden after a stress                                                        | May evolve slowly or suddenly                                                    |
| Attitude to illness    | May seem indifferent or histrionic                                                    | Appropriate concern                                                              |
| Muscle stretch reflexes| Present and normal                                                                   | Absent in spinal shock or very brisk                                             |
| Plantar response       | Normal plantar flexion                                                              | Dorsiflexion of great toe unless spinal shock is present                         |
| Clonus                 | Absent or unsustained                                                               | Sustained                                                                       |
| Muscle tone            | Normal                                                                               | Flaccid acutely, then spastic                                                    |
| Abdominal/cremastri reflex | Present                                                                       | Absent, depending on level                                                      |
| Umbilical migration    | Absent                                                                              | Upward migration if lesion affects T10                                           |
| Sensory level          | Extends horizontally around waist, variable, differs from motor level                 | Slants obliquely downward, constant border if lesion static                     |
| Inadvertent leg use    | May move legs inadvertently for postural support, in sleep, or with Hoover’s test   | Does not move legs if the paraplegia is complete but may show flexor spasms     |
| Sphincter control      | Present                                                                             | Lost                                                                            |
| Anal wink reflex       | Present                                                                             | Lost in stage of spinal shock                                                   |
| Magnetic resonance imaging, somatosensory evoked potential and cystometrogram | Normal but usually not needed                                                    | Abnormal                                                                        |
Clinical Characteristics of NEPS
NEPS’s are episodes of altered movement, emotion, sensation, or experience, similar to epilepsy secondary to purely emotional causes. Patients are usually young adult but age may range from 4 to 77 years. NEPS episodes have been reported during sleep. However, EEGs show that the patient actually is awake before the episode begins. A variety of symptoms at onset like palpitation, malaise, choking, numbness, peripheral sensory disturbance, pain, odors or tastes, and visual hallucinations or distortions can be confused with an aura of epilepsy.

Some of the motor phenomena that are strongly associated with PNES are episodes with gradual onset or termination; events occurring during “pseudosleep”, movements that are discontinuous; irregular activity; side-to-side head movement; pelvic thrusting; opisthotonic posturing; stuttering; weeping; awareness that is preserved during motor activity; The evolution of the clonic jerks from fast and small-amplitude to slow and large-amplitude movements and the rapid contraction and slow relaxation of the true epileptic clonic jerks are not usually seen in psychogenic seizures. The patient may have upward deviation of eyes, rapid tremulousmovements of the lids and resistance to eye-opening during NEPS episode.

Apparent semipurposeful or purposeful behaviors like swallowing, mouthing, chewing, licking, smacking of the lips, looking about, picking up or moving objects, and undressing can occur and may mimic partial seizures. Behavior may be violent and includes biting, striking, kicking, slapping, pushing, obscene gesturing, clawing at the face or clothes, and directed rage. Vocalizations can occur during the episode. Autonomic changes can accompany psychogenic seizures and include light headedness; acral paresthesias; palpitations, tachycardia, dyspnea, or chest pain; headache; dysphoria or panic; fatigue or weakness; and muscle cramps, spasms, or tetany secondary to hyperventilation. The combination of widespread motor movements with subsequent ability to recall the details of the episode may be pointer toward diagnosis of NEPS. However, both focal and widespread unilateral motor seizures due to epilepsy can occur without loss of consciousness. Supplementary motor seizures characteristically involve both sides of the body, but patients often maintain awareness during the episode.

Self-destructive behaviors seen in patients with psychogenic epilepsy which is suggested by urinary or fecal incontinence or both. Termination of the episode can be abrupt as in epilepsy or gradual. Psychogenic status or pseudostatus is an important and surprisingly common manifestation. The patients can present with a variety of symptoms, including all of those mentioned above. Intubation can be allowed by the patient. Complications due to emergency interventions for pseudostatus have included respiratory arrest, septicemia, pneumonia, urinary tract infection, cellulitis, and foot drop. Clinical features to distinguish psychogenic nonepileptic seizures from epileptic seizures are summarized in Table 6.
Diagnosis of Conversion Disorders: Clinical Perspective for a Neurologist

Diagnosis and Laboratory Testing of Psychogenic Seizures

Diagnosis of NEPS is challenging but crucial. Delay in diagnosis has been reported from months to years with mean time of 7.2 years after initial manifestation to appropriate diagnosis. Delay may result in serious consequences like intubation and mechanical ventilation for pseudostatus to adverse effects of antiepileptic medications. Certain ictal features like gradual onset, bizarre and nonsynchronous movements are helpful in diagnosing psychogenic seizures, but none is absolute.

EEG: The combination of a normal EEG with an episode of loss of responsiveness is virtually diagnostic of a psychogenic seizure, though there are following important drawbacks.

An EEG can be normal during simple partial seizures especially from the frontal lobe. The use of additional scalp electrodes with anticonvulsant withdrawal, can improve ictal recordings. Focal generalized slowing secondary to diffuse encephalopathy or a cortical lesion is not necessarily an indication of epilepsy. Generalized epileptiform bursts in patients with drug withdrawal such as barbiturate withdrawal, artifacts or normal-variant patterns and definite epileptiform discharges in patients without epilepsy, or in asymptomatic relatives of patients with epilepsy can be confused as an evidence of epilepsy. Interictal epileptiform discharges did not exclude a diagnosis of psychogenic seizures. Syncope might cause loss of consciousness, or a fall and a short lasting episode of shaking of the limbs. The EEG might show rhythmic slow-wave discharges or EEG “flattening” of at the time of the episode. This is an epileptiform EEG abnormality. Finally, it is difficult to comment on EEG that has many movement artifacts during ictal period and in such situation it may become difficult to comment on epileptiform discharges.

Long video-EEG monitoring documenting spontaneous and provoked typical episode is the “gold standard” test with advantage of simultaneous correlation of behavior and the EEG pattern. Recorded event can be shown to patient, family members or care givers so that they can compare with prior episodes. However, video EEG does not exclude the possibility of coexisting epilepsy and it can be misinterpreted or can mislead.

Serum prolactin: A several-fold increase in prolactin level relative to baseline within 15 to 30 minutes after the episode is suggestive of epileptic episode. Cut off level of 500 IU/ml for serum prolactin was found to be useful and value greater than this was reported in 90% of patients after generalized tonic clonic seizures and in 60% after complex partial seizures. Prolactin level elevation is more common after generalized tonic-clonic or temporal lobe seizures than frontal lobe or other simple partial seizures. Both false-positive and false-negative results can occur. False positive result has been shown after syncope. Prolactin levels are not significantly increased after psychogenic seizures. Absence of postictal prolactin elevation is not a reliable indicator of a psychogenic seizure.

Seizure Induction Protocols

Because episodes do not necessarily occur spontaneously in the laboratory, there has been wide interest in induction procedures. Compression of body parts, photic stimulation, verbal suggestion, placement of a tuning fork or moistened patches on the skin, and intravenous administration of saline or other placebos are the method utilized to provoke psychogenic seizures. Induction of an episode should be done with emotional support and dignity of the patient should be maintained. Induction method has similar importance as neurologic examination maneuvers that are utilized to elicit neurological signs has.

The concept of beneficence as applied to medical ethics dictates that the physician should not only respect and avoid harming a patient, but also should make positive contributions toward the patient’s well-being and towards the removal of possible harmful conditions, maximizing the benefits and minimizing the risks of a particular treatment. An induction procedure has important contribution to this by avoiding the potential hazards of an inappropriate diagnosis and inappropriate treatments. It also facilitates the appropriate treatment for the patient’s condition. In response to induction not every patient with psychogenic seizures will have an episode and in the course of an induction procedure it might be possible that an episode of epilepsy occurs.

Organic Disorders with Bizarre or Subtle Neurologic Manifestations often Mistaken for Psychogenic Illness

Several neurological, metabolic or immunological disorders may have variable neurological presentations. If these symptoms vary in severity, inconsistency on repeated neurological examinations or if they have unexplainable presentations, they can mimic conversion or somatoform disorders. Porphyria, multiple sclerosis, myasthenia gravis, complex partial seizures and collagen vascular diseases are among the commonest mimickers. Early presentations of these disorders may be misinterpreted as a conversion or somatoform disorders and it may delay specific therapy of these organic disorders. One systematic review reported higher rate of misdiagnosis of conversion disorders before 1970 and consistently low rate of 4% after every decade since then. Commonly epilepsy, movement disorders and multiple sclerosis were misdiagnosed as conversion reaction. Possible causes of misdiagnosis were bizarre presenting symptoms and associated psychiatric history. It can cause devastating effect on outcome. Clinician should be more vigilant before considering unexplainable symptoms as nonorganic. In case of any doubt, patient should be thoroughly investigated.

Treatment

The literature regarding management of conversion disorders is scanty. Only few control trials have been conducted in this area. Most of the studies are conducted with small sample sizes and with inadequate control. The outcome measures (dependent variables) are also poorly defined in most of the studies. Most of the studies lack follow up sessions which are very important in the management of conversion disorders. Associated psychopathology such as depression and anxiety are not addressed in most of the studies. Patients diagnosed with conversion disorder frequently benefit from a team approach to treatment and from a combination of treatment modalities. A team approach is particularly beneficial if the patient has a history of abuse, or if he or she is being treated for a concurrent physical condition or illness.

Explanation and Reassurance

A good explanation of the symptoms to a patient with a conversion disorder is a prerequisite of successful further treatment. In explanation and discussion, terms such as psychogenic or functional should be avoided. These terms give impression of intentional act by patient. Scientific term like conversion disorder should be used and patient should be explained about the nature of its symptoms and role of psychosocial stressors. The manner of explanation should be empathetic. It is also difficult to explain and convince friends,
family, and employers about the diagnosis. Neurologists, are usually not well verse with this entity and they do not know how to further pursue the patient. It can be simply explained to the patient that they do not have any neurological disease.

Psychotherapy

Psychodynamic psychotherapies are historically popular in the treatment of conversion disorders, but they are less used empirically. A recent RCT in patients with psychogenic nonepileptic seizures showed benefit from cognitive behavioral treatment (CBT) compared with standard therapy over a 6-month period.46 Group therapy, preferably in conjunction with concurrent individual therapy, offers advantages of reinforcing psycho educational concepts, while providing the opportunity for patients to learn from each other and also help each other. Two noncontrolled studies have reported benefit of psychotherapy to patients having nonepileptic seizures.47,48 Multidisciplinary in patient treatment may be preferred to patients with severe and prolonged symptoms,49,50 but such resources are not available for all patients.

Psychodynamic psychotherapy is sometimes used with children and adolescents to help them gain insight for their symptoms. Family therapy is often recommended for younger patients whose symptoms may be related to some family issues. Group therapy appears to be particularly useful in helping adolescents to learn social skills and copying strategies, and to decrease their dependency on their families. Recently a Cochrane review was unable to draw a conclusion about potential benefits or harms of psychosocial interventions for conversion disorders from the present studies.46 This emphasizes on the need of more scientific studies and randomized controlled trials to elicit role of psychosocial therapies.

Hypnosis

Hypnosis has been advocated for the treatment of conversionsymptoms since the time of Charcot, Janet, and Freud. Neuroimaging data support the idea that conversionsymptoms involve common neurological pathways, and the high hypnotizability of these patients promotes the use of hypnosis in the treatment. Forty-four outpatients who were suffering from conversion disorder were randomly assigned to a hypnosis or a waiting-list condition. It was seen that there was more improvement in hypnosis-condition patients as compared to baseline and waiting list controls.51 Another study comparing a comprehensive treatment program comprising intensive group therapy, social skills training, creative therapy, sports therapy, and physical therapy with or without hypnosis showed no added benefit from hypnosis for resolving conversion symptoms and no predictive value of hypnotizability for treatment outcome.52 Hypnosis can be a useful adjunctive treatment, but it is not essential for improvement. A comprehensive approach is likely to be the most effective. Hypnosis without other forms of psychiatric treatment may decrease conversion symptoms but have less impact on overall psychopathology.

Pharmacotherapy

In the absence of sufficient evidence based data and recommendations, the current practice is to use medications appropriate for the comorbid psychiatric and somatic symptoms. Anecdotal studies report improvement with selective serotonin reuptake inhibitors (SSRIs), betablockers, analgesics, and benzodiazepines.48 An opertional of antidepressants in patients with psychogenic movement disorder and recent or current depression also showed that class of medications to be effective in reducing conversion symptoms.49 Recently a pilot, double-blind, randomized, placebo-controlled trial showed Class II evidence that flexible-dose sertaline up to a maximum dose of 200 mg is associated with a nonsignificant reduction in PNES rate compared with a placebo control arm (risk ratio 0.51, 95% confidence interval 0.25-1.05, p = 0.29), adjusting for differences at baseline.50 If patient is on antiepileptic drugs and diagnosis is likely to be nonepileptic psychogenic seizure, antiepileptic drugs should be stopped in view of their side effects and no therapeutic role. Barbiturates and benzodiazepines should be tapered gradually to avoid withdrawal symptoms. Some antiepileptic drugs have mood stabilizing property like Valproic acid and Lamotrigine, these may be continued if patient has associated bipolar mood disorder. In one study, randomized controlled trial was done to compare effect of immediate versus delayed withdrawal of antiepileptic drugs on outcome. There was a significant reduction in spell frequency and use of rescue medication from baseline to 9 months in the immediate withdrawal group compared to delayed withdrawal group. Emergency health care utilization dropped to zero in both groups by the end of the study.51 No controlled studies have evaluated atypical antipsychotics for the treatment of conversion reactions, particularly in the absence of frank paranoia or psychosis. Reports of the benefits of antipsychotic medications in conversion reactions52-54 are anecdotal.

Transcranial Magnetic Stimulation

More recent anecdotal reports showed benefit of transcranial magnetic stimulation in refractory conversion paralysis55 and somatization associated with posttraumatic stress disorder.56 Frontal-subcortical circuits disruption in these disorders as shown by functional neuroimaging supports the use of transcranial magnetic stimulation in conversion disorders. Future procedures those directly targeting frontal-subcortical circuit will ultimately benefit patients with conversion disorders.

The Patient Who Does Not Improve

All patients with conversion disorder do not respond in meaningful manner. Despite best efforts, if patients does not improve, they can be ensured that best treatment has been given to them for the time being and their symptoms might improve in future if they continue the medication. For time being, therapy should be targeted on present symptomatology like anxiety, depression or other mood disorders. Patient should be explored for other psychosocial stressors or other perpetuating factors. Such patients should be under close follow-up as few organic diseases may mimic conversion disorders as highlighted previously. It is important to protect them from unnecessary investigations and treatment and consideration for referral to specialist center if needed.

Role of Neurologist in Management of Conversion Disorders

Traditionally, neurologists don’t play significant role in management of conversion disorders and refer them to a psychiatrist. However, neurologists can play an important role with further visits to reinforce the explanation and rationale for the diagnosis. After gaining trust in the treating neurologist, it becomes relatively easy to discuss any associated psychological factors with the patient and,
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if needed, a referral can be done to a psychiatrist /psychologist. Significant number of all neurology outpatients have functional symptoms, so it is unlikely to access specialist psychological treatment by all. Psychiatric/psychosocial treatment is not needed for most of the patients and patients with mild symptoms can be managed simply by conversation, explanation and reassurance. If there is no response to appropriate therapy or if there is any doubt in diagnosis of conversion disorder, neurologist plays an important role to look for any missed organic disorder.

Course and Prognosis
Between 50% and 90% of the patients with conversion disorder exhibit short-term resolution of symptoms after reassurance, but as many as 25% of these responders relapse or develop new conversion symptoms over time.\(^\text{51,52}\) Worst prognostic factors are longer duration of symptoms, psychiatric comorbidity, subacute presentation, and tremor. Among patients with nonepileptic seizures, even those with symptomatic improvement may remain disabled.\(^\text{53}\) In one outcome study of 56 such patients, only half of the patients had a resolution of nonepileptic seizures, a mean of 1.5 years after diagnosis, and many still exhibited depressive symptoms, suicidal ideation, and suicide attempts. A patient’s perception of good health and occupational functioning is correlated with resolution, which suggests that interventions that focus on improving functioning and self-esteem could aid treatment.\(^\text{60}\)

In conclusion, this review has limitations as every aspect of conversion disorders cannot be highlighted. Conversion disorders have wide spectrum of manifestation ranging from sensori-motor deficit to movement disorders to nonepileptic seizures. Management of conversion disorders is more challenging as their diagnosis. No evidence based guideline is available as yet. Treatment is mainly targeted to explanation, reassurance and treating associated comorbidities. Relapses and other psychiatric manifestation are common.

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