

REVIEW ARTICLE

Delusional Infestation: State of the Art

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Patients with a delusional infestation (DI) have an overwhelming conviction that they are being infested with (non) pathogens without any medical proof. The patients need a systematic psychiatric and dermatological evaluation to assess any possible underlying cause that could be treated. Because they avoid psychiatrists, a close collaboration of dermatologists and psychiatrists, who examine the patient together, seems to be a promising solution. It helps to start a trustful doctor–patient relationship and motivates the patient for psychiatric treatment. We here review diagnostic criteria, classification of symptoms, pathophysiology and treatment options of DI. Antipsychotic medication is the treatment of choice when any other underlying cause or disorder is excluded. Further research is needed to assess the pathophysiology, and other treatment options for patients with DI.
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Patients with delusional infestations (DI) are totally convinced that some living or non-living organism or fibres have infested their body (1, 2). Like any other delusional disorder, patients do not have insight into the psychiatric origin of their beliefs and do not doubt reality of their convictions. The obsessionality or involuntary engagement with their symptoms causes a great deal of suffering. There is a high burden of disease and many patients consult multiple doctors during the same illness period (3) (Box).

Box. *A representative case history of delusional infestation*

A 38-year-old man, visited our psychodermatology outpatient clinic at the Department of Dermatology of the Academic Medical Center in Amsterdam, with severe symptoms. For the previous 8 years he had been suffering from the sensation of little animals continuously crawling under his skin. He sometimes saw them coming out of his skin as little black fibers. With little tweezers he tried to get the animals out of his body and he showed elaborate skin defects on his arms, back, and legs. He needed to take a shower for at least two hours every day to become clean and get rid of his symptoms. He was unable to continue work for the last 4 years and was socially isolated.

Originally, patients with DI believed that they were infested by parasites or other pathogens like bacteria, viruses, or worms. However, other patients are convinced that they are infested with materials like fibres, filaments, threads, and particles (4). Patients describe associated tactile hallucinations like continuously biting or stinging and itching at one or several spots in or under their skin, or auditory hallucinations like buzzing (5). To get rid of the symptoms they use all kinds of creams, but also needles, knives, or toxic cleaning agents. As a consequence, patients show large excoriations, prurigo nodularis, ulcerations, or secondary infections (6, 7). Many patients, varying between 25–75%, will bring the pathogen to the clinician to prove their conviction, which is known as the “specimen sign” (8–10). Other terms for the specimen sign are “matchbox sign” or “baggies sign”, because patients will use different types of containers to transport the specimen. When one examining the brought sample with a microscope one normally finds cutaneous debris or strings, lint, plant material, or insects (10). Many patients are desperate because of the chronicity of their symptoms (11), and the misunderstanding within their social context and with many physicians. A recent report in patients with delusional disorders shows suicidal behaviours in 8–21% of the patients, which is similar to patients with schizophrenia (12). Patients not only visit dermatologists but also veterinarians, pest control specialists, or entomologists (13).

HISTORY AND DIAGNOSTIC CRITERIA

The initial description of patients with this disease was by Thibierge (14) in 1894 with the name “les acarophobes”. Les acarophobes had the false conviction that they had scabies, although some of them never had it and others were cured.

During the following years other acronyms were used, including “Dermatozoenwahn” (Ekbom’s syndrome), delusion of infestation and parasitophobia. Delusional parasitosis (DP) was the term introduced in 1946 by Wilson & Miller (15) to describe patients who were convinced they were infected by parasites. Because an increasing number of patients did not believe they were infested by parasites but by another living or non-living material, Bewley et al. (4) proposed the term delusional infestation (DI) in 2010. The term DI

includes patients with a delusional belief that they are infested with any kind of living or inanimate pathogen. It could include patients with the so-called “Morgellons disease” (16). Patients with Morgellons disease have similar symptoms like patients with DI including crawling sensations under the skin; spontaneously appearing, slow-healing lesions; hyperpigmented scars when lesions heal; intense pruritus; seed-like objects, black specks, or “fuzz balls” in lesions or on intact skin; fine thread-like fibres of varying colours in lesions and intact skin; lesions containing thick, tough, translucent fibres that are highly resistant to extraction; and a sensation of something trying to penetrate the skin from inside out. However, there is an ongoing discussion about the aetiology of Morgellons disease. According to some authors, patients with Morgellons disease do show signs of dermatitis and the presence of microscopic subcutaneous factors (17). These authors are convinced that Morgellons disease is not a delusional disorder and they present arguments why earlier studies included not the appropriate study subjects and were not able to exclude a physiologic cause of dermatopathy in their patients (18).

Morgellons is not included into the Internal Classification of Diseases 10th Revision (ICD-10). Within psychiatry, DP/ DI is diagnosed as a somatic delusional disorder (DSM-IV/5) (19).

The original description suggests similarities with a phobia, which is an anxiety disorder consisting of irrational fear for heights, animals, or small rooms, but Thibierge (14) already described patients with DI who have overvalued ideas or are completely convinced they are infested by pathogens without having panic attacks or insight into the irrational.

Diagnostic criteria for DI according to the review of Feudenmann & Lepping (1) are: (i) conviction (from overvalued idea to delusion) of being infested by animate or inanimate pathogens without any medical or microbiological evidence of a true infestation, and (ii) abnormal cutaneous sensations explained by the first criterion. Additional symptoms are visual illusions or hallucinations. Location is on, in, or under the skin and any part of the body can be affected.

PREVALENCE

The prevalence of DI is unknown in the general population because of a lack of population-based epidemiological research. However, one recent paper showed results of a population-based study in Olmsted county, USA. The age- and sex-adjusted incidence was 1.9 (95% confidence interval (CI): 1.5–2.4) per 100,000 person-years. Mean age at diagnosis was 61.4 years (range 9–92 years). The incidence of DI increased over 4 decades from 1.6 (75% CI 0.6–2.6) per 100,000 person-years to 2.6 (95% CI 1.4–3.8) per 100,000

person-years between 1976 and 2010. Three surveys and another population-based study (6, 19–21) showed prevalence estimates of 0.148–4.225 per 100,000 person-years, and only one other survey study found an incidence estimate of 0.845 per 100,000 person-years (22). In patients aged less than 50 years there is an equal distribution between males and females but above 50 years 2.5 times more females are affected (4). Moreover, around 8–14% of the patients have a family member or close friend with similar symptoms, which is called *folie à deux* or shared psychotic disorder (23–26). If the patient believes that a close family member is suffering rather than him – or herself, then it is called DI by proxy. A recent paper showed that in a survey to 32,663 veterinary clinicians, 2.3% of them reported that they had seen a case of DI by proxy among pet owners presenting mainly their dogs or cats infested by arthropods or worms to veterinary clinics. One third of the pet owners claimed to be infested themselves, which Lepping et al. called “double delusional disorder” (27).

DIAGNOSTICS AND COMORBIDITY

Within the literature on DI one often sees the distinction between primary and secondary psychosis (see Box, (28)). Patients are diagnosed with “secondary psychosis” when they have DI symptoms with any underlying disorder or drug/ medication that is causing the psychotic symptoms and with “primary psychosis” when there is no underlying cause.

Therefore, when a patient presents with symptoms of DI, it is important to start with ruling out any other underlying disorder, cause, or medication that is causing the symptoms. The main underlying disorders or causes are: (i) true infestation; (ii) medical condition with pruritus like endocrine, renal, hepatic, malignant, rheumatoid, and neurological disorders; (iii) pregnancy; (iv) schizophrenia; (v) psychotic depression; (iv) dementia; (iiv) obsessive-compulsive disorder; (iiiv) skin picking; (ix) trichotillomania; (x) hypochondriasis; (xi) dermatitis artefacta; (xii) psychosis elicited by drugs like cocaine, amphetamines, and cannabinoids; (xiii) medication that enhances dopamine: direct agonists (piribedil, ropinidrol, carbegoline, pramipexol) or NMDA antagonists: amantadine, topiramate; (xiv) drugs which cause pruritus; and (xv) bacterial skin superinfection caused by self-therapy.

The elaborate differential diagnosis requires a thorough history taken by both a dermatologist and psychiatrist to exclude any other underlying cause or disorder, which is easily facilitated within a psychodermatology outpatient clinic where both medical specialists see the patient together. After history taking, a general and dermatological skin examination is required followed by laboratory testing (Table I) and a

Table I. Laboratory testing in patients with suspected delusional infestation

Inflammation markers
Complete blood cell count
Erythrocyte sedimentation rate
Glucose
C-reactive protein
Serum creatine
Electrolytes
Liver function, hepatitis serologies
Thyroid Stimulating Hormone
B vitamins
Folate
Pregnancy test
Borrelia serologies
Human Immunodeficiency virus (HIV) serologies
Vasculitis screening
Allergy testing

skin biopsy. Furthermore, an MRI brain scan is needed to exclude any brain abnormality such as ischaemic lesions or brain tumours.

One of the major limitations in scientific literature on DI up till now is the shortage of standardized, elaborate psychiatric and dermatological testing of large cohorts of these patients. Therefore, we do not know in detail how many patients have primary or secondary DI or any combination of disorders causing secondary DI.

In one meta-analysis of DI case reports, Trabert (25) concluded that 60% of the DI patients have underlying psychiatric disorders. In a more recent review of 107 DI patients from the Mayo clinic, 40 of the 54 patients (76%) who were willing to participate in structured psychiatric evaluation had additional psychiatric diagnoses. The most prevalent diagnoses were depressive disorders (24%), anxiety disorders (19%), and drug use disorders (19%). Only, a minority had personality disorders (4%) (10).

Many DI patients do not want to acknowledge a psychiatric disorder or accept psychiatric medication; dermatologists use their therapeutic privilege and do not disclose the diagnosis before sending a patient to a psychiatrist or starting psychiatric medication. A recent ethical paper discussed this issue and concluded, however, that disclosure of the diagnosis itself is not known to have immediate negative results, so the risk consists of non-improvement and not of worsening. Furthermore, convincing a patient to start treatment or avoiding the risk of them refusing treatment is not considered enough justification for therapeutic privilege. Patients have the right to refuse treatment, even if the results are severe (19).

To improve clinical care and knowledge about DI patients and reduce dropout of patients sent to a psychiatrist, we need a close collaboration between dermatologists and psychiatrists to structurally evaluate all DI patients and collect elaborate clinical information together. At the Academic Medical Center in Amster-

dam (AMC) we have a psychodermatology outpatient clinic where a dermatologist and psychiatrist screen all patients with symptoms of DI and when necessary send the patients to our Psychiatry-Medical Unit for a more elaborate psychiatric and/or somatic screening.

NEUROBIOLOGICAL FINDINGS

As discussed in the last paragraph on Diagnostics; within the literature on DI one often sees the distinction between primary and secondary psychosis. Patients are diagnosed with “secondary psychosis” when they have DI symptoms as a consequence of any underlying disorder or use of drug/medication that is causing the psychotic symptoms and with “primary psychosis” when there is no underlying cause. However, in primary DI, which is one of the psychotic disorders, we nowadays have increased neuroscience-based knowledge showing mainly functional distortions in related networks of the brain that may be responsible for the symptoms. Interestingly, Huber et al. (28) hypothesized a decreased striatal dopamine transporter (DAT)-functioning (corresponding with an increased extracellular dopamine-level) as an aetiologic condition for DI. The DAT is a key regulator of the dopamine-reuptake in the brain, especially in the striatum. The association between DAT and DI is supported by many case reports showing that medication that inhibits the DAT (methylphenidate, cocaine, and amphetamines) can induce DI. Furthermore, many disorders associated with DI (such as schizophrenia, major depressive disorder, Parkinson, Huntington, multiple system atrophy, diabetes, traumatic brain injury, cerebrovascular diseases, hyperuricemia, human immunodeficiency virus, and iron deficiency) do show a decreased striatal DAT-functioning (28). The first small structural MRI (sMRI) study in patients ($n=9$) with DP pointed to distortions in the dopaminergic innervated dorsal striatum/putamen, especially in patients with DP secondary to another (non-psychiatric) medical disorder. Furthermore, most of these patients also showed generalized brain atrophy (29). In 2011, Huber et al. (30) extended their DI sample to 17 patients and summarized results from sMRI/cerebral computed tomography (cCT). All 8 patients with DI secondary to a non-psychiatric brain disorder or another medical condition showed lesions in the basal ganglia, mainly the striatum, and 4 of these cases also showed generalized brain atrophy. Moreover, all 5 cases with DI without another (non) psychiatric medical condition showed no striatal lesions, but all of them showed generalized brain atrophy. A more recent sMRI and source-based morphometry (SBM) study suggested prefrontal, temporal, parietal, insular, thalamic, and striatal dysfunction in 16 DI patients versus 16 controls. Grey matter volume abnormalities

were similar between different causes of DI, but white matter volume abnormalities were restricted to patients with another (non-psychiatric) medical disorder (31).

To date, a few other studies have assessed neural correlates of DI. The only autopsy study in a patient with DI secondary to a hypophyseal tumour suggested a thalamo-cortical disconnection that was responsible for the symptoms (32). The first cCT study in 7 DI patients suggested cortical or subcortical atrophy (33). A review of case reports suggested frontal, temporo-parietal, striato-thalamal dysfunction (1).

Interestingly, one voxel-based morphometry MRI paper assessed neural correlates of somatic delusions and hallucinations in 75 patients with schizophrenia compared to 75 healthy controls, and confirmed structural differences in grey and white matter of the fronto-thalamic region in schizophrenia patients with somatic delusions compared to schizophrenia patients without somatic delusions and controls (34). Furthermore, neuro-imaging studies in patients with itching and skin manipulation because of chronic skin diseases also show abnormal activity of striato-thalamo-orbitofrontal regions (35, 36). Therefore, the proposed neurobiological model of DI consists of a disrupted medial prefrontal control over somato-sensory representations (9, 37).

TREATMENT

The optimal treatment for DI is: (i) building a trustful therapeutic relationship, and (ii) antipsychotic medication. It is a challenge in many cases of DI to develop a trustful patient–doctor relationship. Most of the patients have bad experiences with other doctors in their medical history, do not feel that they are taken seriously about their suffering, and reject any psychiatric diagnosis or treatment.

From the literature a few strategies are suggested for approaching DI patients. When seeing the patient for the first time, it is essential to accept that one will not be able to convince the patient that he or she does not have any animals or pathogens within their skin. Avoid any discussion about the reality of the cause of the symptoms. Moreover, it is essential that a patient can talk freely and that the severity of suffering is acknowledged. The patient also wants a decrease of his or her suffering. Part of building a therapeutic relationship is offering a standard set of diagnostic research including a skin biopsy (see paragraph on Diagnostics). It is still not common sense among dermatologists to systematically offer these diagnostics to exclude any other (treatable) underlying cause of DI. The patient then feels taken seriously, and when any other (treatable) cause is excluded, he or she will more easily accept antipsychotic treatment. Two recently published papers reviewed treatment options of DI and confirmed the importance of building a therapeutic relationship (38, 39).

The next step is prescribing antipsychotics, which needs to be accepted by the patient. Reduction of stress or preoccupation and the itch relieving properties of antipsychotics are medical arguments to convince the patient (38, 40). Interestingly, one study showed reduced growth of parasites by antipsychotics (41). Up till now, only limited evidence confirms effectiveness of antipsychotics in DI patients. Mostly open-label trials and case series/reports do show partial or complete remission in 50–90% of DI patients treated with antipsychotic medication. So far, only one double-blind randomized, placebo-controlled crossover trial, which was poorly designed, assessed effects of antipsychotic medication (pimozide) in 11 DI patients. Pimozide (1–5 mg daily), which is a high-potency first-generation (or typical) antipsychotic agent, was superior compared to placebo for itch delusions, but not for feelings of vermin or excoriations (42). In addition, case series and case reports show improvement of DI symptoms with pimozide (see overview by Generali & Cada [43]) and it has been the drug of choice for years. According to a more recent survey, results show that British dermatologists still prefer pimozide, and prescribe neuroleptics to one third of their DI patients (20). On the other hand, two systematic reviews suggest that the claim that pimozide is a particularly useful treatment for delusional disorder is not based on trial-derived evidence (44, 45). Moreover, pimozide is associated with a higher risk of extrapyramidal side effects, neuroleptic malignant treatment syndrome, and prolongation of the QT-interval compared to atypical or second generation antipsychotics (1).

Therefore, patients with movement disorders should be treated with second-generation antipsychotics. Also, only case reports/series with second-generation antipsychotics (risperidone, olanzapine, quetiapine, sertindole, and paliperidone) in DI patients have been published so far (5, 44, 46). No studies have evaluated any possible differences in effectiveness between typical and atypical antipsychotics in DI patients (44, 47).

One study used several dopaminergic neuroimaging techniques in two DI patients before and after treatment with second-generation antipsychotic medication, and showed that effective treatment was associated with blocking of 63 to 78% of striatal D₂ receptors as well as glucose metabolism changes in the thalamus (9).

There is also limited evidence to guide dose and duration of antipsychotic agents. One small follow-up study with pimozide showed that half of the 14 patients remained in remission 19–48 months after termination of treatment with pimozide. Four patients did not respond at all, and 3 patients (21%) relapsed (48). Another more recent study concluded that prolonged treatment with antipsychotic agents is required because 25% of the patients relapsed within 4 months after cessation of medication (49).

In a few patients with DI, electroconvulsive therapy shows beneficial result (50). One case report showed beneficial results of addition of citalopram to clozapine in a woman with DI and refractory schizophrenia (51).

Besides pharmacological treatment, cognitive behavioural therapy (CBT) could be a treatment option, because patients with schizophrenia show encouraging improvements when treated with CBT with respect to hallucinations and delusions, medication adherence, distress, and relapse. CBT interventions can help the patient to question their fixed beliefs, make connections between their thoughts, emotions, and behaviours, and furthermore help in building an alliance with the patient or improving their social functioning (52–54).

In conclusion, patients with DI suffer severely from their overwhelming conviction of being infested with (non)pathogens without any medical proof. Thorough medical and psychiatric examination is needed to diagnose any contributing cause of symptoms. To improve clinical care and knowledge about DI patients and reduce dropout of patients sent to a psychiatrist, we need a close collaboration between dermatologists and psychiatrists to structurally evaluate all DI patients and collect elaborate clinical information together. Knowledge regarding neurobiological underpinnings of DI is growing and the proposed neurobiological model of DI consists of a disrupted medial prefrontal control over somato-sensory representations. Antipsychotic treatment is the treatment of choice when any other underlying cause or disorder is excluded. Other treatment options like selective serotonin reuptake inhibitors, electroconvulsive therapy and CBT shows promising results. Further research is needed to assess the pathophysiology, and other (long-term) treatment options for patients with DI.

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