Dermatitis artefacta in an intellectually disabled man with monosymptomatic hypochondriacal delusion of HIV infection

Marsus Icaro Pumar and Rajeev Kumar

Aust N Z J Psychiatry 2013 47: 974 originally published online 29 April 2013
DOI: 10.1177/0004867413488219

Dermatitis artefacta in an intellectually disabled man with monosymptomatic hypochondriacal delusion of HIV infection

Marsus Icaro Pumar[1] and Rajeev Kumar[2]

[1]Adult Mental Health Unit Team, Toowoomba Base Hospital, Darling Downs Health Service District, Toowoomba, Australia
[2]College of Medicine, Biology and Environment, The Australian National University, Canberra, Australia

Corresponding author:
R Kumar, Academic Unit of Psychiatry, ANU Medical School, Building 4, Level 2, Canberra Hospital, Garran, ACT 2606, Australia.
Email: Rajeev.Kumar@act.gov.au

DOI: 10.1177/0004867413488219

To the Editor

The prevalence of psychiatric disorders occurring in dermatology outpatient clinics was estimated to be around 30% (Hughes et al., 1983). A range of psychiatric disorders has been reported from an outpatient setting; these include neurotic excoriation, trichotillomania, monosymptomatic hypochondriacal psychosis (MHP), and dermatitis artefacta (Ehsani et al., 2009). MHP, also known as delusional disorder-somatic type is a form of psychosis characterized by a delusion about a particular somatic or hypchondriacal concern. Although the main type of MHP reported in the literature is delusional parasitosis, other forms have also been reported (Wang and Lee, 1997). Dermatitis artefacta is a psychocutaneous disorder in which patients deliberately inflict cutaneous lesions of almost all kinds. It is often associated with childhood sexual abuse, obsessive compulsive disorder, depression, psychosis, intellectual disability, malingering, and factitious disorder (Gupta and Gupta, 1996). We present a case of a 28-year-old male with mild intellectual impairment who presented with dermatitis on his hands and face secondary to a delusion of being infected with HIV.

"Mr L" was a 28-year-old man who presented to the hospital emergency department with infected erythematous skin rashes on the dorsum of his hands and face. He claimed that "HIV was coming out of the hand lesions" and his "blood needed to be drained". He also reported that he had unprotected sex with two women about 3 weeks prior to the onset of the skin rash. On physical examination, he was afebrile and had normal vital functions. The routine blood tests, HIV serology, anti-double-stranded DNA antibodies, extractable nuclear antibody, and anti-neutrophil cytoplasmic antibodies tests were all negative and an MRI brain scan showed no abnormalities. The rashes on the dorsum of the hands and face were warm, tender, and red while some areas displayed signs of infection. He also had erythematous rashes on his face. A diagnosis of dermatitis was made and he was given intravenous flucloxacillin 2 g at the emergency department cubicle. About few hours later, he removed the intravenous line by himself and wanted to leave the hospital. A psychiatric opinion was sought and he was subsequently admitted to an acute psychiatric unit under an involuntary treatment order for further assessment and treatment. Of note, there was no past or family history of any psychiatric disorders. He lived independently with some support and never had any behavioural problems associated with his intellectual disability.

On mental state examination, he appeared as a skinny young man, with poor personal hygiene. His behaviour was characterized by an over-concern with his hands, looking constantly at them and shaking and frequently raising them in the air. He appeared anxious and perplexed with his speech rapid and slightly pressured. He had a severe morbid belief of being infected with HIV and was not amenable to any reasoning. He was not relieved despite repeatedly washing his hands and he did not have the characteristic resistance against a subjective compulsion. Our patient clearly had a well-systematized hypochondriacal delusion of acute onset, which may have been triggered by the fear and anxiety following unprotected sexual contacts. His mild intellectual disability might have been a predisposing factor. His delusional belief of being infected with HIV resulted in repeatedly washing and rubbing his hands to get rid of the virus leading to the development of severe dermatitis affecting initially his hands and later his face.

He was started on olanzapine 10 mg daily and continued on oral flucloxacillin. He responded well to olanzapine 15 mg per day with significant improvement in his skin condition and delusion at discharge. Following his discharge from the hospital, he was assessed by the community mental health service on two occasions reporting good remission.

The most commonly used pharmacological treatment for MHP is the antipsychotic medication pimozide. However, there have been many reported side effects with this medication including cardiac (Q-T interval prolongation) and extrapyramidal symptoms. Several case studies have shown good results with atypical antipsychotics such as risperidone, quetiapine, and olanzapine. Olanzapine has been reported to be effective in previous case reports on MHP (Weintraub and Robinson, 2000; Fawcett, 2002). We chose olanzapine for its immediate sedative effect due to our patient's agitation.

To summarize, early detection and treatment of the primary psychiatric condition can have a significant impact on the positive outcome of comorbid cutaneous disorders.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

Ehsani AH, Toosi S, Shahshahani MM, et al. (2009) Psycho-cutaneous disorders: an epidemiologic study. *Journal of the European Academy of Dermatology and Venereology* 23: 945–947.

Fawcett RG (2002) Olanzapine for the treatment of monosymptomatic hypochondriacal psychosis. *Journal of Clinical Psychiatry* 63: 169.

Gupta MA and Gupta AK (1996) Psychodermatology: an update. *Journal of the American Academy of Dermatology* 34: 1030–1046.

Hughes JE, Barraclough BM, Hamblin LG, et al. (1983) Psychiatric symptoms in dermatology patients. *British Journal of Psychiatry* 143: 51–54.

Wang CK and Lee JYY (1997) Monosymptomatic hypochondriacal psychosis complicated by self-inflicted skin ulceration, skull defect and brain abscess. *British Journal of Dermatology* 137: 299–302.

Weintraub E and Robinson C (2000) A case of monosymtomatic hypochondriacal psychosis treated with olanzapine. *Annals of Clinical Psychiatry* 1: 247–249.

Liver toxicity with clozapine

Peter Tucker

Concord Centre for Mental Health, Concord, Australia

Corresponding author:
Peter Tucker, Concord Centre for Mental Health, Hospital Road, Concord, NSW 2139, Australia.
Email: Peter.Tucker@sswahs.nsw.gov.au

DOI: 10.1177/0004867413488224

To the Editor

Sporadic cases of serious clozapine liver toxicity appeared in the 1990s (Kellner et al., 1993; Markowitz et al., 1997). Two reports of fatal acute liver failure have been published by MacFarlane et al. (1997) and Chang et al. (2009). We report a case of fulminant liver toxicity with good outcome.

The patient was a 21-year-old male university student. He had symptoms of social withdrawal and declining academic performance over the previous 2 years, and the development in recent months of auditory and visual hallucinations, as well as disorganised behaviour including posturing and catatonia. He had been tried on olanzapine with no clear benefit.

On admission he displayed extreme perplexity, thought blocking, flatness of affect, and detachment from those around him. He reported distressing hallucinations, torment and thoughts of suicide.

Investigations for pathology other than schizophrenia were negative. Liver function tests were normal.

Following trials of other antipsychotics, the use of clozapine was proposed. The patient was keen for this. He was enrolled in the clozapine monitoring program.

Clozapine was started at 12.5 mg daily and ramped up gradually in accordance with the recommended standard protocol. He tolerated this well apart from some early sedation and moderate tachycardia without electrocardiogram changes, effectively treated with atenolol. There was significant improvement in his mental state after a week, and this continued to improve.

About 3 weeks after commencing clozapine the patient reported somnolence, which increased over the ensuing week. Liver tests were done: alanine aminotransferase (ALT) was 794 U/L (normal 5–55), aspartate aminotransferase (AST) was 305 U/L (normal 5–55), bilirubin 25 μmol/L (normal 21 or less), albumin and gamma-GT were normal, white cell count (WCC) was 12.1 × 10^9/L (normal 4–10) with neutrophilia, and haemoglobin (Hb) 120 g/L (normal 130–170).

These were repeated several days later with the following results: ALT 1914, AST 830, bilirubin 76, albumin 35 g/L (normal 38–48), gamma-GT 99 U/L (normal 60 or less), WCC 21.4 and Hb 107. Clozapine, which by this stage was at a dose of 250 mg per day, was stopped immediately and urgent gastroenterological advice sought, resulting in the patient's transfer to a gastroenterology unit for observation.

Blood tests were repeated daily. Transaminase peaks (ALT 2282, AST 1056) occurred on the day after cessation, and gamma-GT peaked at 164 the following day. Bilirubin peaked at 112 on day 3, and international normalized ratio (INR) was raised to 1.3 for 5 days. Albumin was below normal for 2 weeks after drug cessation. C-reactive protein (CRP) peaked at 45.5 mg/L the day after cessation. Haemoglobin reached its nadir of 96 g/L 11 days after cessation. The WCC dropped to normal levels after 4 days and all enzymes had returned to normal levels 3–4 weeks after clozapine had been ceased, bilirubin being the last indicator to normalise.

The patient was restarted on amisulpride and within a few weeks he was discharged to the care of his parents on a dose of 200 mg twice daily with ongoing support in the community by the First Episode Psychosis team. Liver function tests were within normal limits.

The case illustrates the treacherous nature of this rare fulminant hepatotoxic reaction. Our patient complained only of sedation in the context of increasing doses of a very sedating drug. As with other cases reported, there was no history or biochemical evidence of pre-existing liver disease, or indeed any indicator of vulnerability, and the liver damage progressed very rapidly.

Our patient appears to be the youngest case described in the literature, which may have contributed to his good recovery after such marked liver test abnormalities. Had recovery not occurred, a liver transplant would need to have been considered. It is of