SHORT REPORT

Pseudoseizures and asthma

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Background: Sexual abuse and head injury are important risk factors of pseudoseizures, reported in about a third of patients. Clinical experience suggests that asthma is another possible risk factor.

Objectives: To determine the relative prevalence of asthma in patients with pseudoseizures.

Methods: A retrospective record review was undertaken of reported asthma in 102 patients with pseudoseizures and 70 psychotic controls. The pseudoseizure patients were subgrouped according to method of diagnosis: 47 in whom epilepsy was excluded by capturing a typical attack on video-electroencephalographic monitoring (VEEM), and 55 not diagnostically confirmed with VEEM.

Results: Asthma was reported in 26.5% of pseudoseizure patients, compared with 8.6% of the psychotic controls (χ^2 = 8.6; p = 0.003). Asthma was reported at similar rates in the VEEM confirmed (29.8%) and non-VEEM confirmed (23.6%) pseudoseizure subgroups. The significant excess of reported asthma held for both the VEEM confirmed subjects (Pearson's χ^2 = 5.4, p = 0.02) and non-VEEM confirmed subjects (Pearson's χ^2 = 8.9, p = 0.003).

Conclusions: There is an association between pseudoseizures and reported asthma. Various models are proposed whereby somatisation, anxiety hyperventilation, and dissociative elaboration may account for the observed association. Both asthma and anxiety hyperventilation may be important risk factors for the development of pseudoseizures. The reported asthma may itself be psychogenic in origin in a proportion of patients. Confirmatory prospective studies are indicated.

A s many as 20% of patients treated for intractable epilepsy may have pseudoseizures,[1–3] a condition also known as dissociative convulsions (ICD-10) or non-epileptic seizures (NES).[4] These are unintentional paroxysmal episodes of altered sensation, movement, perception, or emotional that clinically resemble epileptic seizures but are not accompanied by epileptiform neurophysiological changes.[5] Patients may suffer considerable disability, but early diagnosis and psychotherapeutic intervention can lead to improvement, reduce undue hospital attendance, and avoid unnecessary anticonvulsant treatment.[6] In spite of characteristic differences in semiology, course, and response to treatment, the distinction between epileptic seizures and pseudoseizures can be extremely difficult, and may ultimately depend on capturing a typical attack during prolonged video-EEG monitoring (VEEM).[2] To compound matters, pseudoseizures and epilepsy often coexist.[3] With incidence rates of 3–5 per 100 000[7 8] (similar to those reported for mania[9]), a high index of suspicion should be maintained to avoid inappropriate, ineffective, and costly treatment.

Research into risk factors for pseudoseizures has shown comorbidity with depression and personality disorder,[10 11] as well as less specific predisposing vulnerabilities such as poor coping strategies (for example, in the case of cognitive deficit), and acute stress or loss.[2] In more recent studies, increased rates of around 30% for sexual and physical abuse[12 13] and previous head injury[14–16] were found, suggesting that those are important risk factors. Our clinical experience with pseudoseizure patients gave us the impression of an excess of reported asthma in such patients. Such an observation may provide further insights into the aetiology and evolution of pseudoseizures.

In the present study we sought to establish whether a reported history of asthma is more prevalent in patients with pseudoseizures than in psychiatric patients or the general population, in a sample representative of pseudoseizure patients seen in neuropsychiatric practice (thereby including the significant proportion of such patients who do not undergo prolonged VEEM, and some who also suffer from true epilepsy); and to discuss the aetiological and clinical significance of such an association.

METHODS

We identified patients diagnosed with pseudoseizures (dissociative convulsions, ICD F44.5) referred over a two year period to the national neuropsychiatry unit in the South London and Maudsley NHS Trust. To ensure compatible retrospective data collection, we wished to use a psychiatric control group which approaches our index group, but which does not include anxiety as a prominent feature. We chose to study a sample of psychotic referrals to the psychosis unit in the same trust over the same period. All patients were referred by neurologists and psychiatrists for assessment and treatment, the majority from south eastern England. Patients came from a similar range of socioeconomic backgrounds and were mostly white.

All patients referred with a provisional diagnosis of pseudoseizure underwent a comprehensive evaluation by a consultant neuropsychiatrist. All had an interictal sleep EEG and magnetic resonance imaging of the brain. A diagnosis of epilepsy was excluded on the basis of the clinical history, the ictal presentation, and the results of investigations. A diagnosis of pseudoseizure was confirmed by taking into account the atypical features of the "seizure" episode, the course and response to treatment, and any psychological characteristics suggestive of a dissociative disorder. If the clinical diagnosis was in any doubt, VEEM was carried out. One pseudoseizure group thus comprised patients in whom one or more episodes had been captured on VEEM or, unusually, during a routine EEG recording. The other group consisted of patients in whom the clinical features of pseudoseizures were so obvious as to obviate the clinical need for VEEM, or who did not have an episode in spite of prolonged VEEM, or who refused VEEM.

The same psychiatrist reviewed the patients' clinical notes for basic demographic data and any record of a history of asthma or the use of asthma treatments, as reported by the referring physician or by the patient. Data were collected retrospectively, and there had been no more specific enquiry about respiratory illness than would occur during a routine neuropsychiatric consultation. Wherever recorded, the documented age of onset, clinical course, and treatment of the

Table 1 Cross tabulation comparing asthma prevalence in pseudoseizure subgroups, total pseudoseizure group, and psychotic controls

	VEEM confirmed pseudoseizures	Non-confirmed pseudoseizures	Total pseudoseizures	Psychotic controls	Total
Asthma	14 (29.8%)	13 (23.6%)	27 (26.5%)	6 (8.6%)	33
No asthma	33 (70.2%)	42 (76.4%)	75 (73.5%)	64 (91.4%)	139
Total	47 (100%)	55 (100%)	102 (100%)	70 (100%)	172

VEEM, video-encephalographic monitoring.

reported asthma were noted. It is possible that treatments were more likely to have been documented in inpatients, who would have had more extensive consultations. The rater extracted the same data from the clinical summaries of the psychotic control group.

Data were entered into SPSS. The rates of reported asthma calculated for all groups were cross tabulated. The χ^2 statistic and Student's t test were applied where appropriate to compare differences in rates of reported asthma between groups.

RESULTS

We included 102 patients with pseudoseizures, mean (SD) age 34 (11.8) years, range 15 to 67. One patient with both pseudoseizures and epilepsy, but without EEG capture of pseudoseizures, was excluded. The pseudoseizure patients were predominantly female (68 female, 34 male; male to female ratio 1:2), consistent with other studies. The mean duration of pseudoseizures was 8.0 (8.4) years (range 1 to 44). Mean age of onset was 27 (12.1) years and was not significantly skewed by the inclusion of four female patients who also suffered from true epilepsy, in whom the onset of pseudoseizures was rather more difficult to determine. The pseudoseizure subgroups did not differ significantly in age (p = 0.6) or sex distribution (p = 0.6). The psychotic control group (n = 70) was slightly younger (t = 2.1, p = 0.4), with mean age 30.9 (8.9) years, range 17 to 56, and had a reversed sex ratio (46 male, 24 female; male to female ratio 1.9:1).

Cross tabulation and the χ^2 test of reported asthma prevalence between groups yielded the following (table 1). Compared with psychotic controls, significantly more patients in the total pseudoseizure group had a reported history of asthma (Pearson χ^2 = 8.6; p = 0.003). There was no significant difference in prevalence between pseudoseizure subgroups. When each group was compared with the psychotic controls, the significance persisted: non-EEG confirmed pseudoseizures, Pearson's χ^2 = 8.9, p = 0.003; EEG confirmed pseudoseizures, Pearson's χ^2 = 5.4, p = 0.02.

A χ^2 test applying a 3×2 table comparing psychotic and both pseudoseizure subgroups yielded a Pearson's χ^2 of 9.1 (p = 0.01).

DISCUSSION

We demonstrated a prevalence of asthma of 8.6% in our nonanxious psychotic control group, but the method of asthma prevalence estimation may attract criticism.[17] Recent British retrospective studies using case records,[18] self report survey,[19] and prescription rates[21] found asthma prevalence rates of 7%, 6%, and 5%, respectively, consistent with previous prevalence figures.[21 22] Therefore, using a comparable methodology, the asthma prevalence figures for our control group are similar to those for the south east England general population; this preliminary study thus supports the clinical impression of higher rates of asthma in pseudoseizure patients.

Within the limitations of the study, and in the absence of any other obvious physiological mechanism, this finding may be better understood by considering the complex confounding role of hyperventilation. Both pseudoseizures and asthma are associated with hyperventilation, as well as anxiety. In patients with true asthma, both hyperventilation and anxiety have been shown to trigger an acute attack. Pseudoseizures are often comorbid with anxiety, and hyperventilation can induce pseudoseizures. In fact, hyperventilation is often described as part of the initial phase of an attack, and may be accompanied by its secondary somatic experiential effects: paraesthesiae, tetanus, dizziness, and even reported loss of awareness and collapse.

With the aim of stimulating further hypotheses, we propose four broad models that could explain the observed association in terms of the evolution of symptoms. According to the first three models, the observed excess of reports is not of true asthma, but instead represents somatisation in the absence of any chest symptoms, a misattribution of non-asthmatic respiratory symptoms, or a conversion syndrome closely mimicking asthma.

The first model is that multiple somatic complaints are not uncommon in patients with conversion disorders, including pseudoseizures. These complaints may arise even in the absence of any precipitating subjective physical sensation, and are unaccompanied by evidence of abnormal breathing or chest signs on investigation.

The second model suggests that in patients who do in fact suffer from episodes of dysfunctional breathing or hyperventilation by virtue of anxiety or another mechanism, these subjective symptoms may be misattributed (and misdiagnosed) as asthma. In the event of physiologically more severe or prolonged hyperventilation a patient may even suffer a critical episode of hypocapnoeic dizziness or collapse. If such a patient was also psychologically prone to dissociation these symptoms could be elaborated into seizure-like attacks. These attacks may be reinforced by secondary gain and recur with variable frequency, perhaps precipitated by the same triggers as the early attacks of respiratory symptoms.

The third model, probably a more remote possibility, is that of an asthmatic conversion syndrome, which may occur singly or along with other conversion disorders. Although not yet described in the psychiatric literature, a group of patients with very specific asthma-like symptoms but negative asthma tests has indeed been identified.[23] Little is known about their psychopathology, but Ringsberg's investigations of small samples of such patients have shown that stress triggers attacks that resemble asthma attacks more than hyperventilation.

Some of our patients with a history of asthma may have "pseudoasthma," and the coexistence of these dissociative forms of epilepsy and asthma may be explained in terms of psychological illness templates. Even "mild" pseudoseizures are alarming events, particularly to relatives and bystanders, while milder asthma attacks may be considered less seriously. This, and the relatively close alliance between the disciplines of psychiatry and neurology, may explain the numerous reports on dissociative seizures, while the respiratory equivalent is extremely rare, even in tertiary referral centres. A dissociative form of asthma would have a more variable severity, and milder attacks may afford sufficient secondary gain to perpetuate the condition without necessitating further referral. The above models overlap somewhat, and the proportion of our sample that could be explained by these models can

Table 2 Similarities shared by asthma and epilepsy

- Most often idiopathic. Predisposition may be attributed to family history or events in childhood, for example trauma, infection, etc. Variable age of onset and a range of biopsychosocial precipitants.
- May present as potentially life threatening and socially alarming paroxysms, precipitated by environmental and emotional factors.
- Often initially diagnosed clinically, with further investigation if refractory to treatment or if complicated.
- Costly long term treatment for control of symptoms, and possibly hospital admission during exacerbations.
- Chronic course, with intermittent clustered attacks, potentially leading to avoidance behaviour and instant, brief, and unpredictable access to a sick-role.
- May remit spontaneously or progress to significant disability in spite of patients' compliance with treatment.

only be determined with certainty by establishing the severity and duration of respiratory symptoms, and by appropriate physical investigations.

The fourth but intuitively less plausible model assumes that our study population does in fact suffer more true asthma than controls.

If it were shown to predate the onset of pseudoseizures, a model of dissociative elaboration—similar to that described in our second model—may apply: a patient with true asthma may experience episodes of hyperventilation, the triggers for and the symptoms of which may be different from a typical attack of bronchoconstriction (and perhaps more anxiety provoking). These subjective symptoms provide a susceptible patient with the substrate for dissociation into other syndromes, such as drop attacks and seizures. This may particularly be the case in people with asthma, where a template for intermittent acute illness behaviour, similar to that of epilepsy, already exists (table 2). Through the same pathway, an undiagnosed asthma patient may present to a neurologist with pseudoseizures. If not investigated, the respiratory symptoms may be thought to represent episodes of anxiety hyperventilation. This may lead to an underestimation of asthma in this dissociative population.

It is desirable, from a clinical as well as from a research viewpoint, that the diagnosis of pseudoseizure should whenever possible be confirmed by VEEM. However, this may seem a counsel of perfection to some clinicians, as facilities for VEEM are not widely available in many parts of the United Kingdom. For this reason we chose to analyse VEEM confirmed and VEEM unconfirmed diagnoses separately as well as together, and showed that the subgroups did not differ with respect to the strength of their apparent association with asthma.

Our study does not lend concrete support to any one of the models outlined above over the others, although overreporting of asthma among the dissociative group seems most likely. A more detailed longitudinal history of the onset and course of both pseudoseizures and asthma will be needed if there is to be further clarification. Until then clinicians presented with a patient suffering from atypical seizures and asthma-like symptoms should consider dissociation and anxiety hyperventilation in their differential diagnosis.

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Competing interests: none declared

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