Risperidone-induced pretibial edema: A case report

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Dear Editor,

Risperidone that combines potent D2 and 5-HT2 receptor antagonism is a widely prescribed atypical antipsychotic agent which is effective in especially schizophrenia and bipolar disorder. The most common side effects are known as extrapyramidal symptoms, dizziness, nausea and sedation (1). However, there have been recent reports of possible edema as a side effect (2,3). In this article, we present a case of pretibial edema that develops risperidone after added to the current treatment and recovers after it has been discontinued.

A 63-year-old man with a 16-year history of bipolar disorder and on a maintenance dosage of trifluperazine (2 mg/d) and valproic acid (1000 mg/d) was admitted to our psychiatric clinic because of restlessness. His diagnosis is bipolar disorder in remission. His laboratory results, chest radiograph, ECG and electroencephalography were all normal. We replaced the trifluperazine 2 mg/d with risperidone 2 mg/d. By the end of 3-day period, although his complaints improved, a 3+ pretibial edema has developed in both of the patient's tibia without pain. Repeated his complete blood count, kidney function tests, liver function tests, thyroid function tests, protein, electrolytes and sedimentation, there were no explanation for the edema. The nephrology, endocrinology and cardiology consultations remained normal. We suggested his edema might have been due to risperidone using. We discontinued risperidone and the edema completely reduced within 5 days. We introduced aripiprazole (5 mg/d) and his edema did not recur. According to Naranjo algorithm, probable of adverse reaction due to risperidone-induced pretibial edema: 5-8 = Probable adverse effects were identified (4).

Our patient had no previous physical illness, including cardiac, renal disease and hypertension and developed pretibial edema when he was administered risperidone. After discontinuation of the risperidone, edema reduced. We have yet to find another alternative explanation that more clearly describes the development of edema in our patient. We think that the edema is associated with risperidone due to the temporal relationship between drug intake and edema arrival and regression of edema after drug discontinuation.

In the literature there are some reports that edema may develop related to use of various psychotropic drugs (5, 6). It is known that it can develop due to antipsychotics, especially olanzapine (5,7). However, a limited number of cases of edema developed with risperidone, a potent antipsychotic, have been reported (2,3). In addition, although the etiology of patient’s pretibial edema remains unclear, some mechanisms may be suggested to explain the possible relationship between pretibial edema and risperidone use. Firstly, it may be considered as an allergic reaction to risperidone or its non-therapeutic components in the drug (1). Secondly, we may suggest drug interactions. Because our patient has been taking valproic acid as well as riperidone. Therefore, the complex interaction between risperidone and other psychotropic drugs may have contributed to pretibial edema in our case (1,4). Another possible mechanism is that effect of risperidone on renal and peripheral vessels has been proposed in risperidone-related edema (3,8). Since an open mechanism of risperidone-induced pretibial edema is not known, further studies is needed to determine dose dependence, risk factors, potential mechanisms and the appropriate treatment modality for this condition.

Risperidone is a potent antipsychotic that is frequently prescribed in clinical practice. Clinicians' attention to possible side effects of riperidone will reduce unwanted side effects in patients at risk. However, controlled studies are needed to clarify the relationship between risperidone and edema.
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