

Lisinopril as a Cause of Psychosis in a Patient with CLL and Dermatomyositis

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Abstract

Background: Lisinopril is the most prescribed medication in the United States likely due to its potent effect on both the cardiovascular and renal system. With increased use and popularity of this medication, it is important to understand the myriad of adverse effects that may associated with lisinopril therapy. By understanding these effects, clinicians can be more adept at addressing them if and when they arise.

Case Presentation: Herein, we report a case of a 62-year-old male with a history of chronic lymphocytic leukemia and dermatomyositis who presented with visual hallucinations after lisinopril use.

Discussion: With lisinopril and other angiotensin-converting enzyme inhibitors being commonly used in all specialties of medicine, it is important to understand the common, as well as rare, adverse effects. Recognition of symptoms such as psychosis will help limit incorrect diagnoses, minimize treatment complexity, and ultimately provide better patient care.

Conclusion: With the increasing use of lisinopril and other ACEIs, a broader adverse effect profile is being elucidated. Lisinopril was the culprit in this patient in inducing psychosis in the form of visual hallucinations. This finding was strengthened by the fact that with ceasing use of lisinopril, his visual symptoms abated.

Keywords: Lisinopril; Psychosis; CLL; Dermatomyositis

Background

From years 2015 - 2016 the prevalence of hypertension was 29%, reaching up to 40% in some populations [1]. To address this immense biomedical burden, in addition to diet and exercise recommendations, lisinopril is often prescribed to manage hypertension. As a potent ACE (angiotensin-converting enzyme) inhibitor, lisinopril is often the first pharmaceutical intervention in such cases [2]. Epidemiologically, we have observed an increase in the rate of hypertension and its risk factors - obesity, stress, poor diet - and as such, the prevalence of patients taking lisinopril has increased, making it the most commonly prescribed medication in the United States [3]. Its adverse effect profile mostly consists of cough however other well recognized side effects include hypotension, hyperkalemia, angioedema, and fetal renal effects.

Over the past few years, case studies have reported occasional psychosis, specifically visual hallucinations, associated with lisinopril and other ACE inhibitors. Indiscriminate of age, the patients who were afflicted with psychosis, ranged from 17 to 102 years old, with most being over 70 years of age. Investigators believe that the ACE inhibitors were likely the cause of these psychoses because with ceasing use,

a decrease in symptoms was reported, often within 7 days. Interestingly, two patients who resumed lisinopril treatment after cessation of symptoms, were again afflicted with psychosis [4].

Several hypotheses exist as to why lisinopril and other ACE inhibitors induce such a psychosis. One theory is that ACE potentially degrades opioid molecules preventing its buildup, and, with ACE inhibitor use, an accumulation of opioid molecules occurs. Additionally, in one study, naloxone was utilized as a successful treatment for ACE inhibitor-induced psychosis. Other theories also exist relating inflammation and ACE [4].

Here we will discuss a case report of a patient who developed visual hallucinations after use of lisinopril.

Case Presentation

Herein, we present the case of a 62-year-old patient with a 6-year history of chronic lymphocytic leukemia who is currently controlled with immunotherapy. He began traditional chemotherapy (FCR) in the first half of 2016, however chemotherapy was eventually deemed unsuccessful later that year. As such, he was prescribed to begin CLL immunotherapy with Ibrutinib. One month after initiating Ibrutinib treatment, the patient had to stop this regimen due to skin rashes. To address these rashes, the patient was placed on 60mg prednisone daily, which resulted in rashes abating. With every attempt to reduce the prednisone, however, the rashes worsened. After 8 months of attempting to discontinue prednisone use, the patient was diagnosed with dermatomyositis (DM). The patient's oncologist suggested he begin with Venetoclax for the CLL, which in turn would also aid the DM. After 3 months on the Venetoclax, however, there was no change in his DM. The patient then requested, from his dermatologist, a medicament to treat his DM. It was advised that he begin IVIG therapy instead of any immune suppressants. As such, he was began with 140g (1.5 g/kg). Four months after starting IVIG, his doctors decided to wean him off the steroids. He had multiple permanent side effects from the steroids. Two years' time since initial presentation, he presented reporting heaviness in his chest and after thorough examination, he was prescribed metoprolol 50 mg BID. He, however, continued to have high blood pressure and so his treatment team suspected this was due to IVIG use, and the volume of fluids the patient received with it. Therefore, the patient's cardiologist recommended he begin with 10 mg lisinopril, to help protect his kidneys as the IVIG therapy would be a lifelong commitment.

The patient began taking low dose lisinopril (10mg) prior to sleep to minimize any adverse effects. Approximately 2 - 5 days after beginning lisinopril treatment, however, the patient began to experience hallucinations, specifically with what he describes as moving shadows in his bedroom. He would awaken several times nightly, having very vivid dreams. The patient reported waking up and seeing various 'monsters' in the shadows floating around the ceiling of his bedroom. These events occurred on most nights, and these hallucinations became incredibly bothersome to the degree that he was holding a pillow up above himself to keep the hallucinatory objects from attacking him.

The patient stated had been on multiple medications for a while and never had a similar issue.

Due to these symptoms, the patient self-elected to stop taking lisinopril immediately. He also consulted with his cardiologist who agreed. On the first night of not taking lisinopril the patient reported to not have any hallucinations, and although over the course of the following week he had a similar event, he reported they were very mild and/or not nearly as vivid or scary. As such, his cardiologist placed him on losartan, and he has not had any more issues since being off the lisinopril. Furthermore, patient was referred to psychiatry for follow up.

Discussion

Throughout the years, several cases have been similarly reported about patients developing psychosis while on an ACEI. These incidences of psychosis occur in some patients who recently began the ACEI therapy, while others have been taking it for years. The patient presented herein was interesting in that he had many medical comorbidities that are important to consider. Moreover, the patient was

on several medications, which could have compounded the effect of the ACEI on the brain. Given the timeline of events and that he had not experienced the visual hallucinations with his other medications, and also that with discontinuing lisinopril his symptoms abated, lisinopril becomes the most likely cause. It is important however to consider how the other medications may have predisposed him to the psychosis.

Additionally, it is also worthwhile to inquire whether the patients who experience such symptoms from ACEI are genetically predisposed. As previously discussed, other reports on such cases have proposed theories including ACEI use decreasing endogenous opioid breakdown, and the role of ACE in inflammation. While the specific cause in this case remains unclear, it would be worthwhile for the next steps in evaluating a possible connection between ACEI and psychosis to include genetics evaluation, as well as the comparison of current medication regimens and comorbidities.

Conclusion

This case demonstrates lisinopril as a cause of visual hallucinations. It is critical to raise awareness since lisinopril and other ACEIs are commonly used. It is critical that physicians recognize these symptoms as a potential medical adverse effect to ACEIs rather than an onset of dementia or other medical condition.

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