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Spinal Epidural Lipomatosis: A Review of Pathogenesis, Characteristics, Clinical Presentation, and Management

Keonhee Kim, MD[1,2], Joseph Mendelis, MD[3,4], and Woojin Cho, MD, PhD[3,4]

Abstract

Study Design: Narrative review of available literature.

Objective: To summarize current trends in pathogenesis and management of spinal epidural lipomatosis (SEL) and suggest areas where more research would be of benefit.

Methods: The available literature relevant to SEL was reviewed. PubMed, Medline, OVID, EMBASE, Cochrane, and Google Scholar were used to review the literature. Institutional review board approval is not applicable for this study.

Results: This article clearly summarizes current trends in the pathogenesis and management of SEL.

Conclusions: Possible etiologies of SEL include exogenous steroid use, endogenous steroid hormonal disease, obesity, surgery induced, and idiopathic disease. Comorbidities such as acquired immunodeficiency syndrome and Scheuermann's disease have also been implicated in the pathogenesis of SEL. Steroid-induced SEL seems to have a proclivity for the thoracic region of the spine and has a higher incidence of paraplegia when compared with other forms. Several treatment modalities exist for SEL and are dictated by the underlying cause of the disorder. These include weight reduction, cessation of steroid medications, treatment of underlying endocrine abnormalities, and surgical decompression. Conservative treatments generally aim to decrease the thickness of adipose tissue in the epidural space, but the majority of patients tend to undergo surgical decompression to relieve neurologic symptoms. Surgical decompression provides a statistically significant reduction in symptoms, but postoperative mortality is high, influenced primarily by the patient's preoperative comorbidities. Physicians should consider the underlying cause of SEL in a given patient before pursuing specific treatment modalities, but alarm symptoms, such as the development of acute cauda equina syndrome, should likely be treated with urgent surgical decompression.

Keywords

spinal epidural lipomatosis, pathogenesis, steroid-induced spinal epidural lipomatosis, hormone-induced spinal epidural lipomatosis, idiopathic spinal epidural lipomatosis, obesity

Introduction

Spinal epidural lipomatosis (SEL) is a relatively rare but well-known condition characterized by the overgrowth of epidural adipose tissue within the spinal canal (Figure 1). It is simply accumulation of fat in the canal even though symptomatic SEL should be treated. While SEL can be asymptomatic, patients often present with symptoms related to nerve or spinal cord compression. Steroids represent an important cause of SEL, with the first case of steroid-induced SEL after renal transplantation being reported by Lee et al in 1975.[1] Several disease states are currently thought to be involved in the pathogenesis of SEL—these include long periods of exogenous steroid use, exposure to endogenous steroid resulting from endocrine abnormalities, obesity, postsurgical changes, and idiopathic disease.[2] There is disagreement in the literature regarding the

[1] Yonsei University, Seoul, Republic of Korea
[2] Severance Hospital, Seoul, Republic of Korea
[3] Albert Einstein College of Medicine, Bronx, NY, USA
[4] Montefiore Medical Center, Bronx, NY, USA

Corresponding Author:
Woojin Cho, Montefiore Medical Center, Montefiore Green Medical Arts Pavilion, 3400 Bainbridge Ave, 6th Floor, Bronx, NY 10467-2404, USA.
Email: wcho@montefiore.org

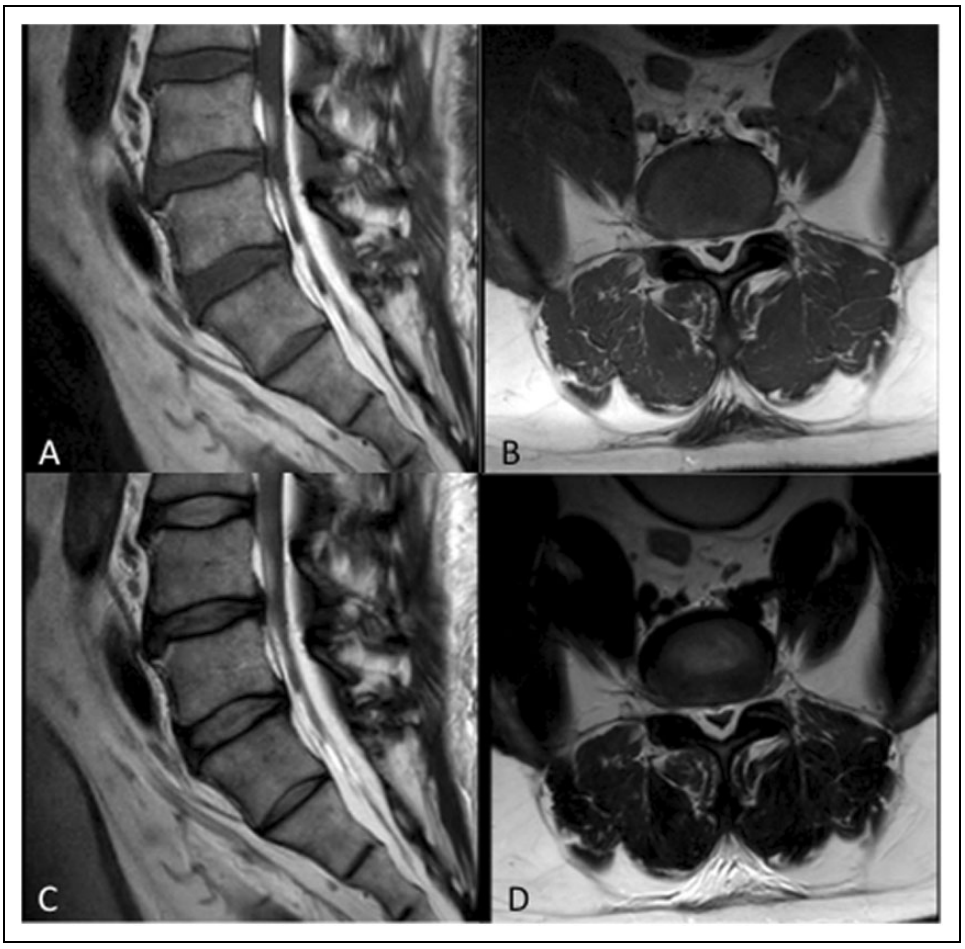

Figure 1. Sagittal and axial T1 (A, B) and T2 (C, D) weighted magnetic resonance images demonstrating epidural lipomatosis at L5-S1 level with crowding of the thecae sac.

definition of idiopathic disease, with some authors using *idiopathic* to indicate SEL of an unknown cause, while others using it to describe SEL associated with obesity or another unknown cause. Our group defines idiopathic SEL as disease in nonobese patients with no known underlying disorder. The aim of this article is to provide an overview of the available literature on SEL and summarize current trends in pathogenesis and management.

Materials and Methods

An electronic database search was conducted using PubMed, Medline, OVID, EMBASE, Cochrane, and Google Scholar. Publications written in English, published from 1967 to 2018, were selected for this review. The following key words were used to retrieve the articles relevant to the topic: spinal, epidural, lipomatosis, idiopathic, characteristics, etiology, management, steroid, and obesity.

Etiology

SEL can be classified into 5 main categories according to pathogenesis: exogenous steroid use, endogenous steroid hormonal disease, obesity, surgery induced, and idiopathic. In 2005, Fogel et al reported the proportion of each category as follows: the exogenous steroid group represents 55.3% of cases, endogenous steroid hormonal disease represents 3.2% of cases, obesity-associated disease represents 24.5% of cases, and 17% of cases are thought to be idiopathic.[3]

Exogenous Steroid Use

In 2008, Al-Khawaja et al analyzed a total of 111 patients with SEL and found that more than 50% of cases were due to exogenous steroid use, many treated with long-term glucocorticoids.[4] Exogenous steroid use is generally accepted as the most common cause of SEL, and it is regarded as the most significant risk factor for developing SEL.[3,4] Some evidence suggests that steroids may enlarge preexisting epidural adipose tissue.[5] There are many conditions requiring steroid administration as first-line treatment including organ transplantation, Crohn's disease, and nephritic syndrome, and cases of SEL have been observed after steroid treatment in these conditions.[3] Diseases such as ulcerative colitis require long-term use of

glucocorticoids and have been shown to increase the likelihood of developing SEL.[6] The number of steroid injections administered to a patient has shown a strong positive correlation with the incidence of SEL,[7] but a recent review attempting to determine the mean cumulative dose of prednisone prior to the onset of SEL failed to reach statistical significance.[8]

Lynch et al reported a rare case of acute onset of bilateral lower limb weakness after glucocorticoid treatment for ulcerative colitis, implying that a patient's sensitivity to steroid may also play a role in the development of SEL in addition to treatment duration, medication formulation, and cumulative dose.[5,9] The glucocorticoid receptor is observed in adipose tissue.[10] Tok et al reported a case of symptomatic SEL following a single epidural injection of exogenous steroid in a diabetic patient.[11] Other steroid hormones can loosely interact with the glucocorticoid receptor, and treatment with testosterone and other anabolic steroids have been associated with cases of SEL in the lumbar region without the use of glucocorticoids.[5]

Endogenous Steroid Hormonal Disease

Exposure to endogenously produced steroids also has a known association with SEL. Several cases of SEL associated with hormonal diseases such as hypothyroidism, Cushing syndrome, carcinoid tumor, and pituitary prolactinoma have been described in the literature.[2,12,13] Overproduction of endogenous steroid is thought to play a similar role as the administration of exogenous steroid in the pathogenesis of SEL. Just as excess steroid causes the accumulation of fat around the neck and trunk region leading to the Cushingoid body habitus, it is thought that excess steroid hormone can lead to the enlargement of adipose tissue in the epidural space, causing nerve compression.[12]

Obesity

Among nonexogenous steroid-related disease, obesity is thought to be the most common cause of SEL.[3] High body mass index (BMI) and obesity are thought to cause a chronic inflammatory condition, which may contribute to the overgrowth of adipose tissue in spinal canal.[3,14-18] Haddad et al suggested that obesity could be a significant driving force in the development of SEL in patents that have never been exposed to exogenous steroid medications.[14] Patients with SEL in the lumbosacral region of the spine tend to have high BMI and type 2 diabetes mellitus.[15,18] Morbidly obese patients occasionally show a higher incidence of SEL without exposure to steroid medications.[16,17] Furthermore, the incidence of SEL is increased in patients with high BMI and triglyceride levels.[7] Fujita et al found that the size of adipose tissue in the epidural region was significantly enlarged in these patients compared with a control group.[18] Obese SEL patients also showed approximately 2.6-fold higher levels of inflammatory cytokines such as tumor necrosis factor-α and interleukin-1β when compared with a control group.[18] This suggests that obesity facilitates chronic inflammation in epidural tissue, which may induce SEL.

The link between obesity and SEL, however, is not without controversy. In 2008, Aliciglu et al analyzed a total of 63 patients with confirmed SEL, and they found that the thickness of epidural adipose tissue did not significantly correlate with BMI or waist circumference.[19] In 2016, Al-Omari et al reported similar results, suggesting that there was no statistical difference between postoperative patients with and without SEL. All patients underwent surgical decompression with or without fusion.[20] Their group analyzed 28 patients (14 patients with SEL and 14 controls with degenerative disk disease without SEL), looking at previously considered as risk factors for SEL: BMI, medical comorbidities, history of steroid injections, and endogenous steroid disease, none of which showed statistical significance. The preoperative duration of symptoms was the only factor that showed significance between the groups—on average double in patients with SEL.[20] It is possible that obese patients with SEL may be a heterogeneous group, explaining the differing results.

Surgery-Induced SEL

Surgical intervention may induce the accumulation of epidural adipose tissue. Choi et al reported a case of SEL after surgical intervention for symptomatic spondylolisthesis. The patient had a BMI of 25.5 and received 2 rounds of epidural steroid injections prior to surgery. The patient underwent an anterior lumbar interbody fusion at the L5/S1 level and laminectomy with herniated disk removal at the L3/L4 level.[21] No abnormal epidural adipose tissue was noted during the procedure. The patient's back and radiating leg pain completely resolved after surgery. Five months later, the patient presented with claudication, back pain, and leg pain, and SEL at L4/5 level was diagnosed on magnetic resonance imaging (MRI).[21]

Choi et al also reported a case of SEL after percutaneous vertebroplasty at T11 and L2 levels. A single dose of steroid was administered directly into the epidural space 1 month after surgery. Five months after the procedure the patient presented with symptomatic SEL.[21] In both cases, the patients had been exposed to exogenous steroid treatment for a short period of time, but the possible effects of surgical intervention on the subsequent development of SEL should not be overlooked.

Idiopathic Disease

Several reported cases of idiopathic SEL can be found in the literature without previously mentioned risk factors or obesity, and meta-analyses estimate that approximately 17% of known SEL cases are idiopathic.[3,20,22-25]

Associated Diseases and Their Treatment

A patient's medical comorbidities seem to play a role in the development of SEL. Human-immunodeficiency virus (HIV)

positive patients may have a higher incidence of SEL compared to the general population. Schürmann et al reported a case of SEL associated with highly active antiretroviral therapy (HAART).[26] The report describes a patient diagnosed with acquired immunodeficiency syndrome in 1992, and subsequently treated with HAART. The patient was noted to be myelopathic 11 years after initiation of treatment and was then diagnosed with SEL.[26] The authors concluded that SEL could be a manifestation of HAART-associated lipidodystrophy. In addition to HAART, HIV-positive patients also commonly receive steroids medications during their course of treatment. Exogenous steroid use is considered as a major risk factor for SEL, and protease inhibitor therapy, which is known to cause lipidodystrophy in some patients, possibly facilitates or aggravates SEL.[27-29] This implies that protease inhibitor therapy may be an additional risk factor for SEL. Physicians should consider SEL in the differential diagnosis for HIV-positive patients on HAART with neurologic symptoms, and MRI imaging should be obtained if indicated. The effect of protease inhibitors must be further studied in order to understand their relationship with overgrowth of adipose tissue in the spinal canal.

Abul-Kasim et al reported a correlation between Scheuermann's disease and SEL.[30] A total of 87 individuals were included in the study: 29 with known Scheuermann's disease and 58 controls. MRI was used to define the thickness of epidural fat in these patients. The authors defined SEL as epidural fat maximum (EF_{max}) greater than 0.6 mm and epidural fat ratio (EFR_{max}) greater than 0.51 on MRI regardless of the presence of neurologic symptoms. The thickness of epidural fat in the seventh thoracic vertebral region was significantly thicker in Scheuermann's patients compared with the control group, and while patients with Scheuermann's disease tend to have high BMI, the incidence of SEL in patients with Scheuermann's disease was 14-fold higher, independent of patient BMI.[30,31] The authors advocate for routine screening for SEL in Scheuermann's patients with MRI prior to surgery to avoid impending neurological injury. However, the study population may not represent true SEL patients, as they were included regardless of neurologic symptoms. The study did show a strong correlation between the thickness of epidural adipose tissue and Scheuermann's disease, but further research should be conducted to determine if symptomatic SEL correlates with Scheuermann's disease.

There have been reports of SEL diagnosed in prostate cancer patients who have received androgen deprivation therapy a part of their treatment regimen. This suggests that use of an androgen antagonist agent may be associated with the development of SEL. Tulloch et al reported that exposure to bicalutamide and goserlin 3 for several months was associated with evidence of SEL on follow-up MRI, not present on MRI obtained before the initiation of the androgen antagonist.[32] Mattei et al reported on another patient treated with enzalutamide that was found to have SEL on a posttreatment MRI.[33] It is possible that androgen antagonist agents may be an emerging cause of SEL, but more evidence is needed to better support this hypothesis.

Table 1. Involvement of Disease[a,b].

Characteristics	Thoracic Involvement (%)	Lumbosacral Involvement (%)	Both (%)
Exogenous steroid use	55.8	32.7	11.5
Endogenous steroid hormonal disease	33.3	~0	66.6
Obesity	30.4	69.6	~0
Idiopathic	37.5	50	12.5

[a]Data from Fogel et al.[3]
[b]The group after using exogenous steroid showed more thoracic level involvement than lumbosacral level: 55.8% versus 32.7%. Almost all of endogenous steroid group showed thoracic-level involvement. However, obesity showed more lumbosacral involvement than thoracic level: 69.6% versus 30.4%. Idiopathic groups also showed very similar pattern as the obesity group: 50% versus 37.5%.

Characteristics

Several studies show that males are more likely to be diagnosed with SEL than females.[2,4,34-37] Different subtypes of SEL also seem to preferentially effect different regions of the spine. Fogel et al reported that 55.8% of exogenous steroid use–related SEL involves the thoracic spine while 32.7% involves lumbosacral level and 11.5% affects both (Table 1).[3] The majority (66.6%) of endogenous steroid hormonal disease–associated SEL shows concomitant involvement of the thoracic and lumbosacral regions.[3]

Al-Khawaja et al defined steroid-related SEL to include both exogenous steroid use and endogenous steroid hormonal disease, and they showed that 73% of those patients have disease involvement at the thoracic level.[4] Interestingly, they showed relatively higher rates of paraplegia on presentation compared with non–steroid-related SEL: 25% versus 5%.[4] The thoracic cord can be easily compressed by a relatively small mass, which could influence the presentation and recovery rate. In steroid-related SEL, Al-Khawaja et al reported a 15% to 20% rate of full recovery when the thoracic level was involved. This was significantly lower than lumbar level involvement, which showed a 60% full recovery rate after surgical intervention and conservative management.[4] Lumbosacral involvement was observed in 69.6% of obesity-induced SEL and 50% of patients with idiopathic SEL without obesity.[3] Al-Khawaja et al reported 65% of non–steroid-related SEL, which included obese and idiopathic SEL, involved the lumbar level.[4] Idiopathic SEL in the Korean population showed a very similar distribution when compared with studies conducted in Western countries.[37]

Racial differences may become more prevalent when discussing the pathogenesis of SEL. In the Korean population, it has been reported that up to 68.8% of SEL cases are idiopathic.[37] Fogel et al reported 17% for the same SEL category

in Western countries.[3] This difference suggests that genetic variation may play a role in the pathogenesis of SEL.

Clinical Presentation

Patients with symptomatic SEL can present with radiculopathy, myelopathy, claudication, cauda equina syndrome (CES), or paraplegia. These symptoms are likely caused by compression from excess adipose tissue in the epidural space, and the exact presentation depends on the location and degree of compression. Steroid-induced SEL (both exogenous and endogenous) has been reported to cause spinal cord compression in SEL patients, including acute-onset paraplegia.[38-41] Most cases occurred following an acute thoracic compression fracture due to osteoporosis in the setting of long-term exposure to steroids, and required urgent surgical decompression.[38-41] In light of these findings, it may be prudent to monitor bone density in patients being treated for SEL, as compression fractures in the setting of excess epidural fat may have significantly increased morbidity.

Patients with SEL can also present with CES. Bowel and bladder issues are the most common presenting symptom in addition to classical SEL symptoms such as back pain and leg weakness.[42] Depending on onset and severity, SEL patients with CES can be treated with urgent surgical decompression or managed conservatively. In mild, slowly progressive SEL with CES, patients have been successfully managed with cessation of steroids and weight reduction,[43-46] but in acute-onset CES with severe symptoms, immediate operative management is considered the first-line treatment.[42,47]

Management

As previously mentioned, SEL can be managed conservatively or with surgical decompression. Around 90% of cases end up being managed surgically, but in patients with secondary disease in the lumbar region, this number appears to be closer to 65%.[4] This suggests that location of the lesion should be considered when planning treatment. There may also be a correlation between severity in clinical presentation and time to recovery.[8] The underlying etiology of SEL should also guide treatment decisions.

Conservative Treatment

If exogenous steroid use is the underlying cause, then reducing or discontinuing the offending medication can gradually reduce neurologic symptoms, but patients may still require surgical decompression if they fail conservative measures.[5,48-50]

Weight reduction decreases BMI, manages obesity, and can eventually treat SEL, if SEL is caused by obesity. Kniprath et al reported that weight reduction decreases the amount of epidural fat; they saw noticeable clinical improvement and drastic adipose tissue reduction at the L5-S1 region on MRI after significant weight loss over a 2-year period.[51] Robertson et al published an article recommending 15 kg of weight reduction for SEL patients to control symptoms.[52] Weight reduction could be the primary conservative treatment for obesity-induced SEL, and regardless of treatment types, weight reduction seems to have a beneficial effect on clinical symptoms.[4]

For a patient with SEL, high BMI, and no history of exogenous steroid treatment, a full endocrinologic workup is required to rule out endogenous steroid hormonal disease before considering obesity as a cause.[5] Overproduction of endogenous steroid can induce high BMI and SEL, which mimics obesity-induced SEL. The goal of treatment for endogenous steroid hormonal disease–induced SEL is managing the underlying disease. After resection of endocrine tumor lesions, patients with SEL had a reduction in epidural fat on MRI and reported relief of neurologic symptoms.[12,53]

Surgical Treatment

Surgery is usually considered when clinical symptoms are acute and severe or conservative treatments have failed.[14,54,55] Patients report gradual recovery after laminectomy and show significant improvement in pain and quality of life.[36,54,56,57] Ferlic et al conducted a retrospective study of prospectively collected data on patient outcomes after surgical treatment of lumbar SEL.[57] These patients underwent decompressive surgery only, most commonly with microscopic laminectomy. They reported that roughly half of patients had a clinically relevant improvement in patient-related outcome scores after laminectomy ($n = 19$) or laminotomy ($n = 3$) and this remained true for up to 2 years. Of note, one patient in their series developed recurrent lipomatosis at decompressed as well as adjacent levels as evidenced on MRI 7 years after the initial surgery. This led them to conclude that it is important to remove as much of the fatty tissue as possible during initial decompression to prevent recurrent disease.

Fessler et al published a series of 5 patients with exogenous steroid-associated SEL, 3 of which were managed surgically. While most patients had significant improvement in neurological symptoms postoperatively, they found a 22% mortality rate within 1 year after surgical decompression, leading them to recommend attempting conservative treatment for patients without significant cord compression.[58] The high mortality rate was not due to a lack of surgical technique. Their general medical and immunological conditions were in the category of high mortality risk group. Fogel et al reported on 3 patients with SEL that were managed surgically with laminectomy and decompression and conducted a review of the available literature.[3] They reported that 52.2% of patients with obesity-associated SEL were treated with laminectomy and debulking with a 66.7% success rate, while 48.8% of patients were treated conservatively with weight loss and had a success rate of 81.8%. This suggests that patients with obesity-associated SEL should probably avoid surgery unless weight loss fails. While the surgical decompression itself is considered low risk, one must keep in mind that the majority of patients with SEL have significant medical comorbidities that will need to be managed in the perioperative period.

Al-Omari et al published an independently reviewed matched cohort analysis that directly compared demographic and operative data on 14 patients who underwent lumbar decompression for SEL with 14 patients who underwent lumbar decompression for degenerative spinal stenosis.[20] They reported no difference in the incidence of complications between the 2 groups. Of note, they mentioned that excess epidural fat, in theory, should act as a protective layer during decompression. Despite this, each group in their study had one patient that suffered a dural tear and spinal fluid leak. Both were primarily repaired and neither had any postoperative sequelae. Each group also had one postoperative wound infection successfully treated with surgical debridement and intravenous antibiotics. In the SEL group, they found no cases of symptomatic recurrence of epidural lipomatosis despite the average BMI at final follow-up remaining statistically unchanged.

It is important to mention that clinical trials have not been conducted to compare outcomes of conservative management and surgical decompression in patients with SEL due to the limited number of cases.[2] However, surgical decompression such as laminectomy with resection of epidural adipose tissue is commonly performed to treat neurologic symptoms associated with SEL. Some of the case reports reviewed for this article described surgical management with laminectomy only, making no specific mention of removal of epidural fat.[18,24,29,36,58] However, the majority of case reports we encountered described surgical decompression and debulking of the excess adipose tissue.[4,11,21,23,28,34,37,38,42,43,47,52,54-56] While most of these authors did not describe the method used to debulk excess epidural fat, some mentioned scraping out the fatty tissue with a hook,[37] removing it with disc punches and curettes,[42] or debulking the excess tissue manually with bipolar cautery.[42]

Overall, surgical decompression and removal of excess fatty tissue is a reasonable option in patients with acute cord compression, CES, or in those who have failed conservative management. One should consider conservative management first-line in other patients.

Conclusion

Spinal epidural lipomatosis is a condition characterized by the accumulation of excess adipose tissue in spinal canal, causing symptoms associated with neurologic compression. It can be categorized into 5 groups according to pathogenesis: exogenous steroid use, endogenous steroid hormonal disease, obesity, surgery induced, and idiopathic. Treatment should be guided by patient-specific factors. Physicians should aim to treat the underlying cause of the disorder, with surgical intervention reserved for patients with acute, severe symptoms or those who fail conservative management. Further research is needed in order to better understand associated conditions and elucidate the specific biochemical pathways involved in this disorder, which may lead to the future development of novel therapies.

Declaration of Conflicting Interests

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