1. Abstract. It should be more descriptive. It is difficult to understand the main purpose of this manuscript and what the authors are reporting.

●**Abstract**

BACKGROUND

Adult neuronal ceroid lipofuscinosis (ANCL) can be caused by compound heterozygous recessive mutations in *CLN6*. The main clinical features of the disease are neurodegeneration, progressive motor dysfunction, seizures, cognitive decline, ataxia, vision loss and premature death.

CASE SUMMARY

A 37-year-old female presented to our clinic with a 3-year history of limb weakness and gradually experiencing unstable walking.The patient was diagnosed with CLN6 type ANCL after the identification of mutations in the *CLN6* gene.The patient was treated with antiepileptic drugs. The patient is under ongoing follow-up. Unfortunately, the patient's condition hasdeteriorated, and she is currently unable to care for herself.

CONCLUSION

There is presently no effective treatment for ANCL.However, early diagnosisand symptomatic treatment are possible.

2. Please answer in the introduction, "Why should this case be reported?"

●The mutation sites are novel and contribute to the knowledge of mutations causing neuronal ceroid lipofuscinosis. Although there is no curative treatment for neuronal ceroid lipofuscinosis, early diagnosis and symptomatic treatment are possible.

3. Could the authors be more specific, "recurrent epileptic seizures, involuntary movement of limbs." What was the type of seizure? What were the abnormal movements?

●Three years prior to admission, the patient began to develop limb weakness and walking instability without obvious inducement. She reported an episode in which she became clouded in mind and fell to the ground, which was

accompanied by limb stiffness, convulsions and upturned eyelid. The symptoms were alleviated after 3-5 min, and she did not experience dizziness, headache, nausea or vomiting. Atonic-clonic seizure was diagnosed.

4. Please provide a brain MRI showing brain atrophy. It would be interesting to add figures of the EEG.

● **Figure 2Brainmagnetic resonance imaging showed that the patient had obvious brain atrophy.**

Figure 3Electroencephalogram findings. The basic rhythmic activity on electroencephalogramwas the mid-potential 8-9c/s alpha wave and poor amplitude adjustment.Both sides were approximately equivalent.The visual response existed, andincreased fast waves in both hemispheres were recordedwithout an obvious spike and slow wave.

5. Could the authors provide a table with similar reports? E.g. variables: symptoms at presentation, age

●**Figure 1History of present illness.**

6. It would be interesting to provide a table with near loci mutation and their symptoms. Trying to explain the mutation based on the clinical manifestation.

● 1. The mutation and symptoms of the disease are described in the ***Discussion*** section.2. Adult neuronal ceroid lipofuscinosis (ANCL) can be caused by compound heterozygous recessive mutations in *CLN6*. The main clinical features of the disease are neurodegeneration, progressive motor dysfunction, seizures, cognitive decline, ataxia, vision loss and premature death.

The complex analysis reported by Xueqiang Wang, et. al., is interesting in the relevant field of adult metabolic abnormalities of genetic origin; however it should be clearly stated in the title that this finding was made in a single patient. Thus, this report could be labeled as a "Case Report". Also the fact that the patient is Chinese does not seems relevant for studies of similar cases from different origin; I think that the words "in China" are confusing and could be deleted.

● 1. Rare adult neuronal ceroid lipofuscinosis associated with CLN6 gene mutations: A case report and review of the literature