Dear editor,
Thank you for your reply and time.
Below are our responses to the esteemed reviewers.
Also, we have rewrote few sentences with a better English.
We believe our revised manuscript is an improved and better version.

Specific Comments to Authors:
1. The mechanism of methanol toxicity mentioned in the submission is the accumulation of formic acid in body fluid will lead to metabolic acidosis, inhibition of mitochondrial cytochrome C oxidase and depletion of adenosine triphosphate (ATP). EPO has anti-apoptotic and angiogenic activities, anti-inflammatory and antioxidant effects, as well as potential neuroprotective and neurogenic effects on ischemia. The author's explanation of the mechanism of EPO treating "methanol toxicity" is probably not clear enough. Personally, the author mentioned in the article that "supportive treatment including electrolyte imbalance correction and hemodialysis" is the real treatment method because they are used to deal with accumulation of formic acid in body fluid which lead to metabolic acidosis. EPO treatment is only an auxiliary method. The author needs to explain it.

Thank you. The treatment of methanol toxicity involves correcting metabolic acidosis with sodium bicarbonate and inhibiting further metabolism with either fomepizole or ethanol. Fomepizole is preferred but very expensive and unavailable in most third world countries. In addition, fomepizole offers no benefits after hemodialysis or after visual damage has occurred. Also, high-dose systemic steroids are commonly used to protect or improve vision in methanol-induced optic neuropathy, but no randomized trials support its benefit. Hence the need for alternative visual protective treatments.

For severe cases, the indications for hemodialysis are: significant metabolic acidosis (pH < 7.25-7.30), visual abnormalities, deterioration of vital signs despite intensive supportive care, electrolyte imbalance unresponsive to conventional therapy, or a serum methanol concentration 415.6 mmol/L (50 mg/dL). However, many cases of visual disturbances or deterioration occur days or weeks after the toxicity and the hemodialysis. Also, some patients are not candidates for hemodialysis, based on the criteria mentioned above, and experience visual deterioration days after methanol toxicity.

Li J, Feng ZJ, Liu L, Ma YJ. Acute methanol poisoning with bilateral diffuse cerebral hemorrhage: A case report. World J Clin Cases 2022; 10(19): 6571-6579 [PMID: 35979299 DOI: 10.12998/wjcc.v10.i19.6571]

As stated in our manuscript, the investigation on the neuroprotective and neuroregenerative properties of EPO is a current active area of research. Several studies reported visual acuity improvement when EPO was used in patients with optic nerve disorders. The main goal of EPO is to protect, maintain or help restore the deteriorated vision days and weeks after the dialysis.

Modarres M, Falavarjani KG, Nazari H, Sanjari MS, Aghamohammadi F, Homaii M and Samiy N. Intravitreal erythropoietin injection for the treatment of non-arteritic anterior ischaemic optic neuropathy. Br J Ophthalmol 2011; 95: 992-5 [PMID: 21131378 DOI: 10.1136/bjo.2010.191627]
Kashkouli MB, Pakdel F, Sanjari MS, Haghighi A, Nojomi M, Homaee MH and Heirati A. Erythropoietin: a novel treatment for traumatic optic neuropathy-a pilot study. Graefes Arch Clin Exp Ophthalmol 2011; 249: 731-6 [PMID: 20890611 DOI: 10.1007/s00417-010-1534-3]
Borhani-Haghighi A, Ghodsi M, Razeghinejad MR, Mardani S, Mardani M, Nikseresht AR, Safari A and Bagheri MH. Erythropoietin for acute multiple sclerosis in patients with optic neuritis as a first demyelination event. Neurosciences (Riyadh) 2012; 17: 151-155

Also, many studies showed that EPO is involved in neurogenesis and can prevent neuronal degeneration such as in retinal ganglion cell (RGC) and axons. However, further research is needed to delineate the exact mechanism of EPO,

In conclusion, hemodialysis is an absolute indication in severe cases of methanol toxicity to prevent death, coma, cerebral hemorrhage, and blindness. The EPO as the reviewer stated is an auxiliary treatment to prevent vision loss, maintain the visual acuity, or even improve the vision weeks or months after the ingestion.
The text in the discussion was fixed to explain this concept.

Shingo T, Sorokan ST, Shimazaki T and Weiss S. Erythropoietin regulates the in vitro and in vivo production of neuronal progenitors by mammalian forebrain neural stem cells. J Neurosci 2001; 21: 9733-43 [PMID: 11739582 DOI: 10.1523/JNEUROSCI.21-24-09733.2001]
Tan H, Kang X, Zhong Y, Shen X, Cheng Y, Jiao Q and Deng L. Erythropoietin upregulates growth associated protein-43 expression and promotes retinal ganglion cell axonal regeneration in vivo after optic nerve crush. Neural Regen Res 2012; 7: 295-301 [PMID: 25806072 DOI: 10.3969/j.issn.1673-5374.2012.04.010]
Yamasaki M, Mishima HK, Yamashita H, Kashiwagi K, Murata K, Minamoto A and Inaba T. Neuroprotective effects of erythropoietin on glutamate and nitric oxide toxicity in primary cultured retinal ganglion cells. Brain Res 2005; 1050: 15-26 [PMID: 15979589 DOI: 10.1016/j.brainres.2005.05.037]

2. The authors may cite some references from F6 Publishing system.

After searching the F6 Publishing system, we have found 2 interesting articles and cited them:

https://www.wjgnet.com/2307-8960/full/v10/i19/6571.htm
https://www.wjgnet.com/2220-3141/full/v9/i3/54.htm

Reviewer #2:
Scientific Quality: Grade A (Excellent)
Language Quality: Grade A (Priority publishing)
Conclusion: Accept (General priority)
Specific Comments to Authors: good

Thank you for your time.