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Wilson's Disease: Current Therapies, Its Controversies, and Potential New Therapeutics

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ABSTRACT

Wilson's Disease is a rare genetic disorder with the prevalence of 1 in every 30,000 people, due to the mutation of the ATP7B gene responsible for copper metabolism. The mutation causes copper accumulation in the body, especially in the liver and brain, which leads to hepatic, neurological, psychological symptoms. These symptoms, if not treated properly, may lead to death after several years. Several treatments including low copper diet, zinc salts treatment, chelating agents (penicillamine, trientine, ammonium tetrathiomolybdate), and liver transplant are currently available. Severe neurological deterioration and other side effects need new, more efficient, and safer therapeutics. Several new therapeutic agents including 4-phenylbutyrate, curcumin, chelating polymeric beads, and long term metabolic correction has been tested in *vitro* and *in vivo*. These new therapeutics may be a potential new treatment with less side effect and greater efficacy for Wilson's disease patients.

Keywords: 4-PBA, 4-phenylbutyrate, curcumin, microbeads, Wilson's disease

ABSTRAK

Penyakit Wilson adalah kelainan genetik langka, dengan prevalensinya 1 setiap 30.000 orang, disebabkan mutasi gen ATP7B yang bertanggung jawab untuk metabolisme tembaga. Mutasi menyebabkan akumulasi tembaga, terutama di hati dan otak, menyebabkan beberapa manifestasi klinis, neurologis, psikologis. Jika tidak diobati, dapat menyebabkan kematian setelah beberapa tahun. Beberapa cara pengobatan termasuk diet rendah tembaga, pengobatan garam seng, agen *chelating* (penicillamine, trientine, ammonium tetrathiomolybdate), dan transplantasi hati. Namun, adanya keterbatasan, gangguan neurologis parah, dan efek samping lain membutuhkan cara baru yang lebih efisien dan lebih aman. Beberapa terapi baru yang lebih potensial dengan efek samping lebih kecil, termasuk 4-phenylbutyrate, kurkumin, manik-manik polimer khelat, dan koreksi metabolik jangka panjang telah diuji secara *in vitro* dan *in vivo*. Dias Rima Sutiono, Giardani Syafitri Sudiro. Penyakit Wilson: Terapi Masa Kini, Kontroversi, dan Terapi Potensial

Kata kunci: 4-PBA, 4-Phenylbutyrate, curcumin, manik-manik mikro, penyakit Wilson

INTRODUCTION

Wilson's disease (WD) is a genetic disorder caused by the mutation of the ATP7B gene on chromosome 13.¹ This mutation causes an autosomal recessive disorder in copper (Cu) metabolism due to the dysfunction of Cu translocase in ATP7B protein highly expressed in kidney, liver, and placenta.^{1,2} ATP7B encodes a transmembrane protein ATPase responsible for transporting Cu to either biliary excretion or ceruloplasmin production.^{1,3} The protein itself will stay in the trans-golgi network (TGN) in normal Cu condition to help in the production of ceruloplasmin that is essential for iron metabolism. ATP7B will move

towards the bile canaliculi during excess Cu concentration to help the dispose of Cu to the bile onto the feces.² Its dysfunction leads to the accumulation of Cu, especially in the liver and brain, and leads to Cu toxicity of the organs.^{1,2} There have been more than 650 determined mutations of the ATP7B gene with sixty percent of them classified as missense mutations.⁴

Worldwide, WD is estimated to have the prevalence of 1 in every 30,000 people⁵ with the ratio of mutation carriers of about 1 in 90 people.⁶ The most common mutations of ATP7B gene are R778L in Southeast Asia,

H1069Q/G in Europe and North America, and the combination of both in Indian population.^{1,2} The phenotype of the disease may change according to the difference in mutational properties. Early detection and treatment are vital for the survival and even may lead to the prevention of neurological symptoms in patients with hepatic manifestation.⁶ Untreated cases may lead to death within 2-5 years subsequent to the onset of neurological symptoms.⁴

CLINICAL MANIFESTATIONS

The clinical manifestation of WD may appear at any age with the majority between 5 to 35

Controversies In Therapeutics

Michael Seilmaier



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