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Analytical Methods of Cilnidipine and Its Combinations

Nelson Cox*

Department of Pharmaceutical Analysis, Professor in chemistry, Ahram Canadian University, Egypt

Letter

Hypertension is a condition in which blood pressure is elevated to an extent where benefit is obtained from blood pressure lowering. The risk of complications is proportional to the level that blood pressure raises. Calcium channel blockers are a class of compounds used in the treatment of hypertension. The dihydropyridine (DHP) group, a subclass of the calcium channel blocker works almost exclusively on L-type calcium channels in the peripheral arterioles and reduce blood pressure by reducing total peripheral resistant. Long acting DHP is preferred because they are more convenient for patients and avoid the large fluctuations in plasma drug concentration which are associated with side effects. Amlodipine is the most distinct DHP and the most popular. The drug was patented in the year 1986 and its commercial sale began by 1990. The current article provides a state of art about the analytical and bio analytical techniques available for the quantification of drug as a single entity and in combined pharmaceutical formulations between 1989 and 2019 [1].

Hypertension is one of the most significant observed public health challenges contributing to cardiac disease and death globally. Hence, to lower the risk for cardiovascular disease, it is admissible to control blood pressure strictly. Combining two or more antihypertensive agents with different action mechanisms can be used reliably to achieve the aforementioned condition. This combination therapy has proved beneficial in preventing major cardiovascular diseases, reducing the risk for adverse effects, and maximizing drug compliance.

Calcium channel blockers (CCB) are first-line drugs in the treatment of hypertension. However, CCB alone was insufficient in lowering blood pressure. Hence, CCBs have been widely co-administered to treat hypertension. These drugs act by inhibiting calcium (Ca)-channels in the myocardium and vascular smooth muscle cells, which lowers the myocardial contractions, decrease pulse conduction, and causes vasodilation. Thus, they are found to be effective in the treatment of essential hypertension. Furthermore, among the three main classes of CCBs, 1,4-dihydropyridines (DHP) have contributed to a widely used hypotensive drug class [2].

Among various 1,4-dihydropyridine CCBs, Cilnidipine (CLD) shows unique action on sympathetic N-type Ca-channels, besides acting on L-type Ca-channels, as with most Ca-channel antagonists. Their action is performed through vasodilatation, decreased heart rate, and increased renal blood flow. CLD has opted as CCB of choice in hypertensive patients with diabetes, chronic kidney disease, and patients developing edema. It is a novel 4th generation CCB. It is found to dilate both efferent and afferent arterioles resulting in decrease in pressure in the capillary bed. Hence, the accumulated fluid of tissues flows back to veins, thus skipping pedal edema incidence. It shows a slow onset but long-lasting hypotensive effect by inhibiting sympathetic neurotransmission and norepinephrine release. It shows excellent selectivity for vascular smooth muscle. CLD has also emerged as a good candidate for combination therapy.

CLD is chemically described as 2-Methoxyethyl (2E)-3-Phenyl-2-propen-1-yl-2,6-dimethyl-4-(3-nitrophenyl)-1,4-dihydro-3,5-pyridinedicarbox¬ylate. The development of CLD can be credited jointly to Fuji Viscera Pharmaceutical Company, Japan, and Ajinomoto, Japan which was approved in 1995. Countries like China, Japan, Korea, India, and several countries in the European Union have approved this drug. CLD is a light yellow-coloured crystalline powder. It is insoluble in water. The molecular weight of CLD is 492.528 g/mol, and the molecular formula is C27H28N2O7. Absorption of CLD is rapid; maximum peak concentration is achieved in 2 hours. Distribution in kidneys, liver, plasma and other tissues is high. Even after repeated oral administration, accumulation of CLD is not observed. It has a large volume of distribution. It shows low bioavailability due to low aqueous solubility and high permeability. Microsomal enzymes highly metabolize it with a dehydrogenation process in both the liver and kidney. Elimination is 20% through the urine and 80% through feces. The half-life of the hypotensive effect for CLD is about 20.4 min. It shows cardioprotective, renoprotective, and neuroprotective effects. Administration of CLD has been shown to decrease blood pressure safely and effectively, without excessive blood pressure reduction or tachycardia [3].

Methods of analysis

Various analytical methods for the determination of CLD are presented here as compendial and reported methods. Compendial methods are official methods.

Compendial method

Japanese Pharmacopeia and Indian Pharmacopeia approved CLD in 2018 in 2016, respectively. The identification method for CLD includes Ultraviolet (UV)/ Visible (Vis)-spectrophotometry and infrared spectrophotometry. In the Japanese Pharmacopeia, chromatographic separation was achieved on stainless steel column with dimensions 25 cm x 4.6 mm and particle size of 5 µm, and the mobile phase reported is a mixture of sodium acetate buffer and methanol. The detection wavelength is 240 nm using a UV detector. The column temperature mentioned is 25°C.

The Indian Pharmacopeia also mentioned chromatographic separation using Phenomenex-Prodigy ODS 3V column of dimension 25 cm x 4.6 mm and particle size of 5 µm. The mobile phase reported is a mixture of acetonitrile (ACN): 0.01M sodium acetate buffer (70:30% v/v). The flow rate is reported to be 1.0 mL/min. The injection volume is 20 µL, and the detection wavelength was 240 nm using a UV detector [4].

***Corresponding author:** Nelson Cox, Department of Pharmaceutical Analysis, Professor in chemistry, Ahram Canadian University, Egypt. E-mail: coxn9595@gmail.com

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Reported methods of analysis

The rapid progress of science and technology has led to the development of numerous newly synthetic drugs prompting the development of analytical methods for determining these drugs in the manufacturing phase of the pharmaceutical formulations and their determination in the human body. Thus, the analysis of pharmaceuticals has gained progressive importance in the overall drug development process. This study aimed to comprehensively review the literature and collect the evidence concerning the analysis of CLD and its combinations of dosage forms. Data was assembled by search on Google Scholar, Pubmed, and Elsevier's Science Direct. The keywords included "estimation of cilnidipine", "analytical method development of cilnidipine," "pharmaceutical preparations of cilnidipine," "cilnidipine in biological fluids." Figure 2 provides chronological reported methods for estimation of CLD. The results show that CLD can be estimated by spectrophotometry, High- performance liquid chromatography (HPLC), Liquid chromatography-mass spectroscopy (LC-MS), High- performance thin layer chromatography (HPTLC), voltammetry, capillary electrophoresis, and spectrofluorimetry, either in the form of raw materials or pharmaceutical preparations. Different analytical methods for estimating CLD as reported in the literature are comparatively provided.This review provides a complete insight for the analysis of CLD, alone or in combination in pharmaceutical preparations or biological fluids. Figure 4 provides comparative data of estimation methods for CLD alone and in combination with other drugs [5,6].

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