Hypertension : an insight

Ch Karunakar[1], K Spandana[2], Ravinder Reddy K[3]

[1]Assistant Professor, [2]Postgraduate student, [3] Professor, Department of General Medicine, Prathima Institute of Medical Sciences, Nagunur, Karimnagar, Telangana, India.

Address for correspondence: Dr K Spandana, Postgraduate student, Department of General Medicine, Prathima Institute of Medical Sciences, Nagunur, Karimnagar, Telangana, India.

email id - kspandana219@gmail.com

The history of hypertension goes back a long way. In ancient Chinese and Indian Ayurvedic medicine, the quality of an individual's pulse felt by gentle palpation was a window into the condition of the cardiovascular system. What was called "hard pulse" possibly would qualify for the modern term of hypertension.

Traditionally the discovery of hypertension is attributed to Richard Bright (1836) who found an association between renal disease and left ventricular hypertrophy (LVH). He speculated that LVH was a consequence of high BP. For nearly 50 years after Bright, all hypertension was thought to be caused by renal disease. Frederick Akbar Mahomed (1849–1884), an Irish-Indian physician working at Guy's hospital in London, first reported elevation of blood pressure in a person without evidence of kidney disease (later came to be known as "essential hypertension). The name 'hypertensive vascular disease' was introduced by Janeway. Sir Clifford Allbutt described many features of essential hypertension and also the concept of hypertension as a generalized circulatory disease which he referred to as 'hyperpiesia"The term essential hypertension ('EssentielleHypertonie') was coined by Eberhard Frank in 1911

The basis of measurement of blood pressure was established by the pioneer works of Hales in 1733.The first practical clinical instrument for measuring blood pressure was a water-filled sphygmomanometer developed by Von Basch and Von Recklinghausen in the 1880s. Air filled cuff-based sphygmomanometer was developed by Scipione Riva-Rocci in 1896 which enabled the measurement of systolic blood pressure by palpating the radial pulse. Nikolai Sergeyevich Kortokoff describedauscultatory sounds in 1905, which allowed estimation of both diastolic and systolic BP.

Volhard and Fahr (1914) used clinical and pathological criteria to differentiate essential hypertension from renovascular hypertension. Volhard also described malignant hypertension – a rapidly progressing lethal type of high BP which can occur as a complication in all forms of hypertension. Later, many types of secondary hypertension have been described, apart from those related to renal disease

Treatment of Hypertension

Historically, acupuncture, venesection and bleeding by leeches were the sole means of treating what was called 'hard pulse disease'. Venesection was recommended by the Yellow Emperor of China, Cornelius Celsus, Galen, and even Hippocrates. A low salt diet had been suggested as early as 1922 by Allen and Sherrill for BP reduction. The rice diet of Kempner(which included a daily intake of 2000 Calories and consisted of rice, fruit and juices that limited sodium intake to 150mg, protein to 20g, and fat to 5g) became popularized in 1940s.

Sodium thiocyanate was the first chemical substance to be used in the treatment of hypertension,byTreupel and Edinger in 1900 but became unpopular due to its toxic side effects.

Some rather radical methods of reducing BP in patients with accelerated or malignant hypertension included injection of pyrogens (typhoid bacilli), radical surgical methods such as sympathectomy and adrenalectomy. First sympathectomy was done by the surgeon Fritz Bruening in 1923. Chemical sympathectomy with tetraethyl ammonium chloride, hexamethonium(ganglion-blockers) was introduced later. Dr. Edward D. Fries used the antimalarialPentaquine in 1947 to reverse the signs (headache and hematuria) of malignant hypertension in patient who was turned down for sympathectomy as a poor surgical risk. In the 1940s, impressive hypotensive efficacy had been noted with the herb veratrumviride. Lower doses of veratrum was used along with low sodium diet for lowering BP but there was a narrow therapeutic range.

Despite the increasing evidence for the association of cardiovascular diseases and mortality with blood pressure, many members of the medical community were skeptical about the imperative to lower blood pressure. Even eminent cardiologists and expert physicians considered elevated blood pressure to be necessary for adequate perfusion of vital organs until late 1950s.

The natural history of untreated hypertension is illustrated by the case of President Franklin Delano Roosevelt who had a BP reading of 162/98mm Hg in 1937 at the age of 57, (but did not receive therapy because of the then medical opinion and knowledge) progressed to target organ failure and death from hemorrhagic stroke in 1945.

In 1950, Dr. Tinsley R. Harrison published the first edition of his Principles of Internal Medicine which advocated that "treatment of hypertension should be based on symptoms of coronary difficulties.Those with chest pain or other overt signs of disease should have their hypertension treated; others should not.[1]"

Three years after Roosevelt's death, the pivotal National Heart Act was signed into law by President Truman. The Act created the path for the study of heart diseases and resulted in several studies including the Framingham Heart study. Despite evidence presented in the late 1950s on the clinical benefits of treating malignant hypertension, including electrocardiographic changes showing reversal of left ventricular hypertrophy, a group of physicians who have been referred to as the "New York Nihilists" refused to accept that treatment was helpful. "Benefit achieved in the management of malignant hypertension," they stated, "may represent the 'natural history of the disease.'"

Attempts to treat hypertension, with the few drugs available at the time, caused more misery and earlier demise for the patients than leaving them untreated. The search continued for alternatives to sympathectomy. Phenoxbenzamine (sympathetic nerve blocker), hexamethonium (ganglion blocker) and guanethidine (peripheral adrenergic blocker) were discovered. Reserpine from rauwolfia serpentine, initially used as a sedative, was also shown to lower BP through a central sympatholytic action but was poorly tolerated. Hydralazine, a vasodilator, was also in use as an antihypertensive agent by the late 1950s. The biggest step came with the introduction of the first orally effective diuretic Chlorthalidone, an important breakthrough in the management of hypertension, by Freis, Wilson and Parish in 1957.The early 1960s also witnessed the next significant step in antihypertensive agents' discovery. Propranolol, the first ß-blocker was introduced by Prichard and Gillam, followed later by agents with increased cardio selectivity. Despite the progress in research and treatment, there were still skeptics. ACE inhibitors and Calcium channel blockers were introduced in the early 1980s. Angiotensin II receptor blockers (Losartan) were introduced in 1993 and Renin inhibitors (Aliskiren) in 2000.

The Veterans Administration Medical centers (dedicated to the care of war veterans) started VA Co-operative research program, designed and supervised by Dr. Edward Freis. The design of this study became the prototype for future clinical trials and the results formed the basis for early recommendations for antihypertensive therapy [2].

The next milestone in the story of hypertension is publication of the first report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC) in 1977. JNC panel was appointed by National Heart, Lung, and Blood Institute (NHLBI). JNC I emphasized on treating the diastolic blood pressure; there were no recommendations for staging of systolic hypertension. Since then, with the availability of more data from large clinical trials, the recommendations of the JNC reports have become increasingly aggressive and specific shifting more to the treatment of systolic pressures.Subsequent updates were published in 3- to 6-year intervals.JNC-7 was published in 2003 which introduced the term 'prehypertension'(BP of 120-139/80-89 mm Hg) and blood pressure goal <140/90mm Hg(<130/80 mmHg for diabetic and CKD patients). At the time of JNC 1, there were fewer than 30 drugs; at the time of JNC 7, there were more than 100.The availability of effective and safe medications has made it possible to reduce BP to goal levels[3].

The long-awaited eighth report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 8, 2013)is not the official hypertension guideline replacing JNC 7. JNC-8 guidelines were not based on a systematic review of the data, and there was not a thorough analysis of the adverse effect and harms of drugs used for hypertension. JNC 8 is the first contemporary hypertension guideline to apply only RCTs to its recommendations. The JNC-8 hypertension guidelines are not endorsed by the NHLBI, the American Heart Association, the American College of Cardiology, nor any other authoritative body. JNC-8 recommended patients older than 60 years be treated to a systolic blood pressure of less than 150 mmHg, which has generated considerable controversy and caution.The striking findings of the Systolic Blood Pressure Intervention Trial (SPRINT) have received considerable attention because of the demonstration that intensive therapy to a target systolic

blood pressure below 120 mmHg decreases cardiovascular mortality and morbidity more than less intensive treatment to a target systolic blood pressure below 140 mmHg, but this approach is not fully generalizable because the trial excluded patients younger than 50 years and those with diabetes and prior stroke[4].

The latest AHA/ACC High Blood Pressure guidelines,2017 eliminate the category of prehypertension, categorizing patients as having either Elevated (120-129 and less than 80) or Stage I hypertension (130-139 or 80-89) or Stage 2 hypertension(>140/90 mm Hg) and the recommendation of threshold for treatment with antihypertensive agents is consistent with JNC-7.

New drug classes, eg, inhibitors of vasopeptidases, aldosterone synthase and soluble epoxide hydrolase, agonists of natriuretic peptide A and vasoactive intestinal peptide receptor 2, and a novel mineralocorticoid receptor antagonist are in phase II/III of development, while inhibitors of aminopeptidase A, dopamine ß-hydroxylase, and the intestinal Na+/H+ exchanger 3, agonists of components of the angiotensinconverting enzyme 2/angiotensin(1–7)/Mas receptor axis and vaccines directed toward angiotensin II and its type 1 receptor are in phase I or preclinical development. The two main interventional approaches, transcatheter renal denervation and baroreflex activation therapy, are used in clinical practice for severe treatment resistant hypertension in some countries. Novel interventional approaches in early development include carotid body ablation and arteriovenous fistula placement. Importantly, none of these novel drug or device treatments has been shown to prevent cardiovascular disease outcomes or death in hypertensive patients[5].

Like fire, we now know that hypertension is best stopped early and aggressively or, better yet, prevented.The Modern management of hypertension represents a major success story in preventive medicine.

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