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Commentary Open Access

Hypertension-A Challenge to Modern Medicine

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Commentary

Cardiovascular disease is one of the leading causes of death in adults and one of the main reasons for morbidity. Hypertension is one of the most important modifiable risk factors of cardiovascular disease and in India; it is the leading non communicable disease and estimated to be attributable for nearly 10 percent of all deaths [1]. Adult hypertension prevalence has risen dramatically over the past three decades from 5 per cent to between 20-40 percent in urban areas and 12-17 per cent in rural areas [2,3].

Hypertension is classified as either primary (essential) hypertension or secondary hypertension; about 90-95% of cases are categorized as "primary hypertension" which means high blood pressure with no obvious underlying medical cause. Coined essential hypertension nearly 100 years ago, primary hypertension is typically a genetic disease, with susceptibility increasing based on one's environment. The remaining 5-10% of cases (secondary hypertension) is caused by other conditions that affect the kidneys, arteries, heart or endocrine system. Secondary hypertension, however, is second to primary hypertension and occurs when the patient has no family history of hypertension, with no obvious reasons for a diagnosis.

Benign high blood pressure is essential hypertension running for a considerably long period of time and being asymptomatic. The term benign distinguishes this type of high blood pressure from the more aggressive and rapidly developing accelerated hypertension which is also known as malignant hypertension. Malignant high blood pressure is more acute and severe rapidly reaching its end stage which may be stroke, heart attack or heart failure if left unattended. The term malignant hypertension was used by pathologists who noted that these patients have acute target organ injury associated with fibrinoid necrosis of the wall of an artery.

On the other hand benign high blood pressure is slow moving and less damaging compared to accelerated hypertension. It often remains dormant for years in unaware individuals. It is in reality more common amongst many populations than malignant hypertension. This form refers to a patient who was diagnosed with primary hypertension that was controlled at one point for a period of time, and now the patient's BP is elevated despite no changes in medication or lifestyle. The patient experiences progressive increases in BP that can top out at extremely high levels of 240 mmHg/120 mmHg in a relatively short period of time.

Even moderate elevation of arterial blood pressure is associated with a shortened life expectancy. Dietary and lifestyle changes can improve blood pressure control and decrease the risk of associated health complications, although drug treatment is often necessary in people for whom lifestyle changes prove ineffective or insufficient.

There seems to be a strong familial and genetic predisposition in the pathogenesis of essential hypertension, whereas a number of modifiable predisposing factors are also involved. More than 50 genes have been examined in association studies with hypertension, and the number is constantly growing. One of these genes is the angiotensinogen (AGT) gene. It was seen that increasing the number of AGT increases the blood pressure and hence this may cause hypertension. In single variant tests, it has been shown that SNPs were enriched for variants associated with adiposity, type 2 diabetes, coronary heart disease and kidney function in previously published Genome Wide Association Study, providing evidence that genetic loci related to blood pressure contribute to cardiovascular outcomes. The genetic factors contribute to approximately 30-70% of disease susceptibility. The proportion of hypertensive patients with affected siblings was studied in 6000 Caucasian patients, showing a recurrence risk of ~3.5 for hypertension [4].

The main determining factors for essential hypertension include, age and sex (For adults BP is lower in women comparable to men, however in later life blood pressures are higher in women),weight gain, salt and alcohol intake, smoking and lack of physical activity.

Hypertension is a major risk factor for the development of cardiovascular disease. Its impact for stroke and end stage renal disease (ESRD) is the greatest. Hypertensives when compared to normotensives develop twice as much as coronary heart disease (CHD), 4 times as much congestive heart failure (CHF) and seven times as much stroke.

One of the most important prognostic factors in hypertension is electrocardiographic or echocardiographic left ventricular hypertrophy, with the later already present in as many as 25% patients with newly diagnosed hypertension. In a multi-centric study with hypertensive patients with no prior history of cardiovascular/renal disease LVH was accompanied by a threefold increase in cardiovascular events.

Because of the firmly established facts on LVH, antihypertensive therapy has been directed to reduce left ventricular mass. But despite impressive data it has to be seen whether intensive antihypertensive therapy can completely normalize excessive risks of cardiovascular/renal disease than in untreated patients.

A number of new classes of antihypertensive drugs have become available in the recent years which appear to hold therapeutic potential for better management of hypertension. Losartan, an angiotensin II receptor antagonist, does not produce cough which is classically seen with ACE inhibitors. Fenoldopam, a dopamine D1-receptor agonist, has a rapid and short duration of action and is ideally suited by intravenous infusion for quick control of BP in hypertensive emergencies. Kentaserin, a serotonin (5-HT2A) receptor antagonist, has a long duration of action and can be given once daily. It has the

added benefit of having antiplatelet effect. Monatepil, a dual alpha-receptor and calcium channel blocker, has potent antihypertensive effect, lowers serum cholesterol and also has anti-atherosclerotic effect. Dual ACE and endopeptidase inhibitor, such as alatriopril, has a "broad spectrum" antihypertensive effect and may be effective in majority of hypertensive patients. Many other classes of antihypertensive drugs are still in the investigative stage, and their therapeutic potentials and safety need to be ascertained in long-term controlled clinical trials.

The problem of hypertension can't be solved by medical treatment alone. Active educational efforts should be directed to the community about creating awareness about hypertension and its complications. The community needs to be educated of the benefits of leading physically active life, maintaining ideal body weight, stopping smoking, reducing alcohol intake, stress management.

Dietary approach to stop hypertension trial (DASH) - a multi-centre controlled-feeding study of dietary patterns to lower blood pressure emphasized fruits, vegetables, low fat dairy products, diet low in saturated fats has been tried recently to curb the effects of hypertension.

Future looks good for patients' with hypertension. With advent of newer drugs and also rise in patients' awareness; it looks like we can finally get a hold of this modern day pandemic.

References

1. Patel V, Chatterji S, Chisholm D, Ebrahim S, Gopalakrishna G, et al. (2011) Chronic diseases and injuries in India. Lancet 377: 413-428.
2. Gupta R (2004) Trends in hypertension epidemiology in India. J Hum Hypertens 18: 73-78.
3. Reddy KS, Shah B, Varghese C, Ramadoss A (2005) Responding to the threat of chronic diseases in India. Lancet 366: 1744-1749.
4. Brown MJ (1996) The causes of essential hypertension. Br J Clin Pharmacol 42: 21-27.