GUIDELINES

Management of hypertension: summary of NICE guidance

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This is one of a series of *BMJ* summaries of new guidelines based on the best available evidence; they highlight important recommendations for clinical practice, especially where uncertainty or controversy exists. Further information about the guidance, a list of members of the guideline development group, and the supporting evidence statements are in the full version on bmj.com.

Hypertension is one of the most important preventable causes of death worldwide and one of the commonest conditions treated in primary care in the United Kingdom, where it affects more than a quarter of all adults and over half of those over the age of 65 years.[1] This article summarises the most recent recommendations from the National Institute for Health and Clinical Excellence (NICE) on the management of hypertension,[2] which updates the 2004 and 2006 clinical guidelines.[3-5]

Recommendations

NICE recommendations are based on systematic reviews of best available evidence and explicit consideration of cost effectiveness. When minimal evidence is available, recommendations are based on the Guideline Development Group's experience and opinion of what constitutes good practice. Evidence levels for the recommendations are in the full version of this article on bmj.com.

Diagnosing hypertension

- If blood pressure measured in the clinic is 140/90 mm Hg or higher:
  - -Take a second measurement during the consultation
  - -If the second measurement is substantially different from the first, take a third measurement
  - -Record the lower of the last two measurements as the clinic blood pressure. (Updated recommendation)
- If the clinic blood pressure is 140/90 mm Hg or higher, use ambulatory blood pressure monitoring to confirm the diagnosis of hypertension. This strategy will improve the accuracy of the diagnosis compared with current practice[6] and was also shown to be cost effective—indeed, cost saving—for the NHS. (Updated recommendation)
- When using ambulatory blood pressure monitoring to confirm a diagnosis of hypertension, ensure that at least two measurements an hour are taken during the person's usual waking hours (for example, between 0800 and 2200). Use the average value of at least 14 measurements taken during the person's usual waking hours to confirm a diagnosis of hypertension. (New recommendation)
- If a person cannot tolerate ambulatory blood pressure monitoring, home blood pressure monitoring is a suitable alternative to confirm the diagnosis. (New recommendation)
- When using home blood pressure monitoring to confirm a diagnosis of hypertension:
  - -For each blood pressure recording, take two consecutive measurements, at least one minute apart and with the person seated, and
  - -Record blood pressure twice daily, ideally in the morning and evening, and
  - -Continue recording blood pressure for at least four days, ideally for seven days, and
  - -Discard the measurements taken on the first day and use the average value of all the remaining measurements to confirm a diagnosis of hypertension. (New recommendation)
- While waiting for a confirmed diagnosis of hypertension, investigate target organ damage (such as left ventricular hypertrophy, chronic kidney disease, and hypertensive retinopathy) and formally assess cardiovascular risk. (New recommendation)
- Use risk equations to assess cardiovascular risk—for example, the Framingham risk calculator[7] (as used in the Joint British Societies' risk charts available in the *British National Formulary* and available from http://bnf.org/bnf/bnf/61/204016.htm) and QRISK2 (available from http://qrisk.org/).[8 9]

Thresholds for intervention

- If the person has severe hypertension (clinic blood pressure ≥180/110 mm Hg), consider starting antihypertensive drug treatment immediately, without waiting for the results of ambulatory or home blood pressure monitoring. (New recommendation)
- Offer lifestyle advice to people with hypertension at initial diagnosis and then periodically thereafter.
- Offer antihypertensive drug treatment to people aged under 80 years with stage 1 hypertension (that is, an average ambulatory or home blood pressure of ≥135/85 mm Hg and <150/95 mm Hg; a clinic blood pressure of ≥140/90 mm Hg and <160/100 mm Hg) and who have one or more of the following:
  - -Target organ damage
  - -Established cardiovascular disease
  - -Renal disease
  - -Diabetes
  - -A 10 year cardiovascular risk equivalent to ≥20%. (Updated recommendation)

Step 1	**A** (for patients aged <55 years) or **C*** (for patients aged ≥55 years and all black people of African or Caribbean descent)
Step 2	**A + C***
Step 3	**A + C + D**
Step 4	**Resistant hypertension** **A + C + D + further diuretic†** (or α blocker or β blocker if further diuretic treatment is not tolerated or is contraindicated or ineffective) Consider seeking specialist advice

Key
A = Angiotensin converting enzyme inhibitor or angiotensin II receptor blocker
C = Calcium channel blocker
D = Thiazide-like diuretic
* Calcium channel blocker preferred, but consider thiazide-like diuretics in people with oedema or high risk of heart failure
† Consider low dose spironolactone or higher doses of thiazide-like diuretic

Drug treatment of hypertension

- Offer antihypertensive drug treatment to people of any age with stage 2 hypertension (an average ambulatory or home blood pressure of ≥150/95 mm Hg; a clinic blood pressure ≥160/100 mm Hg) irrespective of the presence of target organ damage, cardiovascular disease, renal disease, or the 10 year risk of cardiovascular disease. (Updated recommendation)
- For people aged under 40 years with stage 1 hypertension and no evidence of target organ damage, cardiovascular disease, renal disease or diabetes, consider seeking specialist evaluation for secondary causes of hypertension and a more detailed assessment of potential target organ damage. This is because 10 year cardiovascular risk assessments can underestimate the lifetime risk of cardiovascular events in these younger people. (Updated recommendation)

Blood pressure medication

The figure outlines an algorithm showing the four steps in the drug treatment of hypertension.

- If blood pressure is not controlled by the treatment offered at each step, review medication to ensure that the treatment is at optimal or best tolerated doses before moving to the next step. (Updated recommendation)
- For people aged 80 years and over, offer the same antihypertensive drug treatment as for people aged 55-80 years, taking into account any comorbidities. (Updated recommendation)

Step 1

- For people aged under 55 years, offer an angiotensin converting enzyme (ACE) inhibitor or a low cost angiotensin II receptor blocker (ARB). If an ACE inhibitor is prescribed and is not tolerated (for example, because of cough), offer an ARB. (Updated recommendation)
- Do not combine an ACE inhibitor with an ARB to treat hypertension. This is not the most rational combination to reduce blood pressure and may result in more adverse events without any additional clinical benefit.[10] (Updated recommendation)
- For people aged over 55 years and black people of African or Caribbean family origin of any age, offer a calcium channel blocker. If this is not suitable—for example, because of oedema or intolerance—or if there is evidence of heart failure or a high risk of heart failure, offer a thiazide-like diuretic. (Updated recommendation)
- If diuretic treatment is to be started or changed, offer a thiazide-like diuretic, such as chlortalidone (12.5-25.0 mg once daily) or indapamide (1.5 mg modified release once daily or 2.5 mg once daily), in preference to a conventional thiazide diuretic such as bendroflumethiazide or hydrochlorothiazide. (Updated recommendation)
- For people who are already taking bendroflumethiazide or hydrochlorothiazide and whose blood pressure is stable and well controlled, continue treatment with the bendroflumethiazide or hydrochlorothiazide. (Updated recommendation)

Step 2

- Offer a calcium channel blocker in combination with either an ACE inhibitor or an ARB. (Updated recommendation)
- If a calcium channel blocker is not suitable for step 2 treatment—for example, because of oedema or intolerance—or if there is evidence of heart failure or a high risk of heart failure, offer a thiazide-like diuretic. (Updated recommendation)

Step 3

- If treatment with three drugs is needed, offer an ACE inhibitor or ARB, combined with a calcium channel blocker and a thiazide-like diuretic. (Updated recommendation)

Step 4 (Resistant hypertension)

- If clinic blood pressure remains higher than 140/90 mm Hg after treatment with the optimal or best tolerated doses of the drug combination mentioned in step 3 (an ACE inhibitor or an ARB combined with a calcium channel blocker and a diuretic), regard this as resistant hypertension, and consider adding a fourth antihypertensive drug and/or seeking expert advice. (Updated recommendation)
- For treatment of resistant hypertension:
  - -Consider further diuretic treatment with low dose spironolactone (25 mg once daily) if the blood potassium concentration is 4.5 mmol/L or lower. Use particular caution in people with a reduced estimated glomerular filtration rate because they have an increased risk of hyperkalaemia
  - -Consider higher dose thiazide-like diuretic treatment if the blood potassium concentration is higher than 4.5 mmol/L. (Updated recommendation)

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- Management of stable angina (*BMJ* 2011;343:d4147)
- Management of hip fracture in adults (*BMJ* 2011;342:d3304)
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- If further diuretic treatment for resistant hypertension at step 4 is not tolerated or is contraindicated or ineffective, consider an α blocker or β blocker. (Updated recommendation)

If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of four drugs, seek expert advice if not yet obtained. (Updated recommendation)

Monitoring blood pressure treatment

- Use clinic blood pressure measurements to monitor the response to antihypertensive treatment with lifestyle modifications or drugs. (Updated recommendation)

For people identified as having a "white coat effect"—that is, a discrepancy of more than 20/10 mm Hg between clinic and average daytime ambulatory blood pressure or average home blood pressure measurements at the time of diagnosis—consider ambulatory or home blood pressure monitoring as an adjunct to clinic blood pressure measurements to monitor the response to antihypertensive treatment with lifestyle modification or drugs. (Updated recommendation)

Blood pressure targets

- Aim for a target clinic blood pressure below 140/90 mm Hg in people aged under 80 years with treated hypertension. (Updated recommendation)
- Aim for a target clinic blood pressure below 150/90 mm Hg in people aged 80 years and over with treated hypertension. (Updated recommendation)

Overcoming barriers

The recommendation that ambulatory blood pressure rather than clinic blood pressure measurements should be used to confirm the diagnosis of hypertension will have a profound impact on patient care by reducing the number who are incorrectly labelled as hypertensive and thus inappropriately prescribed antihypertensive treatment. Currently, only some primary care practices have access to ambulatory blood pressure monitoring devices, with the rest having to access them through referral to secondary care. Sufficient numbers of validated ambulatory devices (refer to www.bhsoc.org/blood_pressure_list.stm for a list of clinically validated monitors) will need to be procured and adequately maintained. Staff will need to be trained in their use and how to interpret data generated in the reports. The implementation of ambulatory blood pressure monitoring should be determined locally, reflect what is best and most convenient for patients, and not necessarily be based on current models of service configuration. The Guideline Development Group anticipates that practices and consortiums will devise various strategies that do not involve specialist referral to expand provision, and that procurement costs will fall as demand increases.

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RATIONAL TESTING

Investigating mixed hyperlipidaemia

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This series of occasional articles provides an update on the best use of key diagnostic tests in the initial investigation of common or important clinical presentations. The series advisers are Steve Atkin, professor, head of department of academic endocrinology, diabetes, and metabolism, Hull York Medical School; and Eric Kilpatrick, honorary professor, department of clinical biochemistry, Hull Royal Infirmary, Hull York Medical School. To suggest a topic for this series, please email us at practice@bmj.com

Mixed hyperlipidaemia is often associated with metabolic syndrome, non-alcoholic fatty liver disease, and risk of developing type 2 diabetes

A 50 year old white man visited his general practitioner after the recent death of his brother from a myocardial infarction at the age of 63. Apart from a single gout attack, his medical history was unremarkable. He was taking no medications, denied excess alcohol intake, and was a former smoker (of 20 cigarettes a day), having stopped about seven years ago. He was centrally obese with a body mass index of 34 (weight (kg)/height (m)^2) and a waist circumference of 107 cm and height 176 cm. His blood pressure was 144/94 mm Hg. His fasting lipid profile was total cholesterol 6.6 mmol/L, triglycerides 5.1 mmol/L, high density lipoprotein cholesterol 1.1 mmol/L; calculation of low density lipoprotein cholesterol was not possible owing to high triglyceride levels. His fasting glucose was 6.3 mmol/L.

What is the next investigation?

Mixed hyperlipidaemia is defined as fasting triglyceride >1.7 mmol/L and total cholesterol as >5 mmol/L (as in this case). Diagnosis of mixed hyperlipidaemia is important because of its associations with increased cardiovascular risk, its strong association with risk of diabetes, and its role as an ancillary liver function test. Guidelines from the National Institute for Health and Clinical Excellence recommend the use of risk scores such as Framingham (http://cvrisk.mvm.ed.ac.uk)[1] and QRISK (www.qrisk.org)[2] to assess cardiovascular risk.[3] In patients with this type of lipid profile, clinical investigations also need to determine if the hyperlipidaemia is primary or secondary. Factors in the history particularly relevant to a finding of hyperlipidaemia are a family history of diabetes and any alcohol intake.

Repeat testing is warranted to reduce biological and analytical variation and to exclude transient causes of hypertriglyceridaemia (such as non-fasting sample, alcohol,[4] acute viral infection, or other cause of sepsis[5]). The Friedewald equation often used for calculating low density lipoprotein cholesterol is not valid in non-fasting samples and underestimates the concentration and so is invalid in patients with severe hypertriglyceridaemia (>4.5 mmol/L)

Exclude secondary causes of hyperlipidaemia by bearing in mind the following points, and testing where appropriate.

- Triglyceride concentrations show a curvilinear increase with HbA_{1c} and half of patients with uncontrolled diabetes are hypertriglyceridaemic. Hypertriglyceridaemia (>3 mmol/L) should prompt investigation for diabetes even if fasting glucose concentrations are normal as about 30% of patients will have impaired glucose tolerance and about 10% undiagnosed type 2 diabetes.[6] Patients with conditions likely to cause or be associated with insulin resistance—for example, obesity, polycystic ovary syndrome, or the metabolic syndrome—have an increased frequency of hypertriglyceridaemia. Diagnose diabetes with a fasting glucose or HbA_{1c} test or if necessary a glucose tolerance test.[7]
- Excess alcohol intake. Hypertriglyceridaemia (>10 mmol/L) will develop in 25% of patients with a normal lipid profile who are given 1 g alcohol/kg body weight in a loading test, especially if combined with a meal high in saturated fat.[4] One unit of alcohol is about 8 g. Alcohol excess is best diagnosed by a history but can be associated with raised γ glutamyl transferase, increased levels of carbohydrate deficient transferrin (>2.5%), raised high density lipoprotein cholesterol, and erythrocytic macrocytosis (>99 fL).
- Obstructive hepatic disease and parenchymal hepatic disease. These can be diagnosed by conducting a liver profile (bilirubin, alanine aminotransferase, alkaline phosphatase) and other investigations. If the patient is at increased cardiovascular risk, guidelines recommend not starting statin treatment when alanine aminotransferase levels exceed three times the upper reference limit of normal (about 150 U/L), but there is no cut-off for γ glutamyl transferase.
  - Non-alcoholic fatty liver disease (also known as hepatic steatosis) is often found in patients with the metabolic syndrome and is distinguished by raised γ glutamyl transferase and later raised aspartate aminotransferase and alanine aminotransferase.[8] A ratio of aspartate aminotransferase to alanine aminotransferase of <1.3 is found in 87% of cases of non-alcoholic fatty liver disease. The

LEARNING POINTS

Mixed hyperlipidaemia (defined as fasting triglyceride >1.7 mmol/L and total cholesterol >5 mmol/L) is often associated with lifestyle factors such as excess alcohol or obesity

Mixed hyperlipidaemia is a feature of the metabolic syndrome and associated with non-alcoholic fatty liver disease and risk of developing type 2 diabetes

It may also be a marker of endocrine disease (such as polycystic ovary syndrome) or hepatic disease

It is associated with increase in cardiovascular risk not fully captured by commonly used risk calculators

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- Diarrhoea after broad spectrum antimicrobials (*BMJ* 2011;342:d3798)
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grade of impairment and risk of non-alcoholic steatohepatitis can be staged by using the ratio of aspartate aminotransferase to platelet count. This is calculated as follows: ((aspartate aminotransferase/upper level of normal)/platelets (10^9)) × 100. A value <0.42 indicates little fibrosis and >1.2 indicates established fibrosis.

-Viral hepatitis is more often associated with hypertriglyceridaemia in patients infected with hepatitis C virus rather than with hepatitis B virus.

- Severe renal impairment. Nearly all (90%) patients with chronic kidney disease grade 4 will have hypertriglyceridaemia.[9] Conduct initial screening with a creatinine test, using the result to calculate the estimated glomerular filtration rate (using the MDRD (calculated modified diet in renal disease) calculator), and testing for urine protein with a dipstick. If the estimated glomerular filtration rate is <45 mL/min/1.73 m^2 then a ratio of spot urine albumin to creatinine should be calculated to fully stage renal impairment.
- Thyroid dysfunction. Classically this presents with hypercholesterolaemia, although a few cases show a mixed profile.
- Other endocrine causes. Hypertriglyceridaemia is associated with Cushing's syndrome, growth hormone or anterior pituitary deficiency, and polycystic ovary syndrome; these require specialist investigation and management.
- Drug induced hypercholesterolaemia is rare except when systemic corticosteroids or other immunosuppressants (such as ciclosporin) are used and in special groups such as patients with HIV taking older antiretroviral drugs (such as indinavir, stavudine).

Given the strong association between an increased atherogenic index (ratio of triglycerides to high density lipoprotein cholesterol) and future type 2 diabetes,[10] patients should be assessed for the presence of the metabolic syndrome, on the basis of three of the five factors listed below (waist circumference plus two others):

- Waist circumference >95 cm for white people, >88 cm for Asians
- Triglycerides >2.3 mmol/L
- High density lipoprotein cholesterol <1.0 mmol/L (men) or <1.2 mmol/L (women)
- Systolic blood pressure >130 mm Hg
- Fasting glucose >5.5 mmol/L.

Outcome

In our patient an oral glucose tolerance test showed a baseline glucose concentration of 6.4 mmol/L and 120 minute glucose concentration of 10.0 mmol/L, confirming the presence of impaired glucose tolerance. His liver function tests showed aspartate aminotransferase 35 U/L (<55 U/L (upper level of normal)), alanine aminotransferase 45 U/L (<55 U/L), alkaline phosphatase 62 U/L (<130 U/L), γ glutamyl transferase 65 U/L (<78 U/L), with a bilirubin of 10 µmol/L (<21 µmol/L). An ultrasound scan confirmed the presence of fatty liver (hepatic steatosis) but found no other disease. Creatinine and thyroid function tests were normal.

The ratio of total cholesterol to high density lipoprotein cholesterol forms part of the standard data needed to calculate a patient's risk of cardiovascular disease using either the Framingham or QRISK algorithms.[11] These algorithms underestimate risk in patients with hypertriglyceridaemia.[12] Lipid lowering drug treatment is indicated if cardiovascular risk >20% over 10 years. Our patient's Framingham (http://cvrisk.mvm.ed.ac.uk)[1] and QRISK (www.qrisk.org)[2] risk scores were respectively 10.5% and 6.9% over 10 years (relative risk 1.2; heart age 52 years), indicating a likely need for future but not current drug treatment. The final diagnosis was that he had features of the metabolic syndrome including probable non-alcoholic fatty liver disease and had about a 40% likelihood of developing diabetes within four years.[13]

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