

CPD study pack

February 2021;265(1845)

The study pack contains this month's CPD exercise plus all relevant articles:

- Reducing cardiovascular risk pivotal in diabetic kidney disease
- Tight blood pressure control key in polycystic kidney disease
- Holistic approach needed to tackle chronic primary pain

Fillable PDF templates*

- CPD exercise is a fillable PDF

CPD prompt and framework templates

- Journal article ● Case review ● Audit

* Adobe reader app needed for ipad etc
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Practitioner CPD exercise

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Show impact

- Collection of data
- Practice audit to show improved care
- Case studies
- Implement a protocol
- Prescribing review
- Educational meetings
- Significant event audit
- Practice meeting
- Implement a new service
- Take on new role
- Significant event audits
- Change with feedback from patients and/or colleagues

Further options for sessional GPs

- Self audit of own practice:
- Record-keeping
- Referrals for investigations
- Prospective condition based reviews
- Random case analysis with colleagues
- Review of telephone triage outcomes
- Update drugs in emergency bag

CPD Framework

Online all articles are associated with CPD prompts/frameworks to written reflection:

Adobe fillable PDFs

- Article review framework
- Case review framework
- Audit framework

Word documents

- Reflection on a journal article
- Stages of reflection on a situation
- Team reflection

DIABETIC KIDNEY DISEASE

1 Which of the statements are true?

A Early diabetic kidney disease is asymptomatic

True ☐ False ☐

B Individuals with diabetic kidney disease have a reduction in life expectancy of around 6 years

True ☐ False ☐

C Diabetic kidney disease typically occurs in association with diabetic retinopathy and neuropathy

True ☐ False ☐

D The rise in urinary albumin excretion frequently precedes any fall in eGFR

True ☐ False ☐

E A renal biopsy is rarely necessary to confirm diabetic kidney disease

True ☐ False ☐

2 A List four modifiable risk factors for diabetic kidney disease.

B List four non-modifiable risk factors for diabetic kidney disease.

3 All patients with diabetic kidney disease should have at least annual measurement of which parameters?

4 List any changes to your clinical practice that you may make having read this article.

POLYCYSTIC KIDNEY DISEASE (PKD)

5 Which of the statements are true?

A The liver is the most common site for extrarenal cysts

True ☐ False ☐

B The size and number of renal cysts will continue to increase with time

True ☐ False ☐

C Individuals with PKD may present with hypertension before a decline in eGFR is noted

True ☐ False ☐

D The PKD2 mutation is associated with a more aggressive phenotype

True ☐ False ☐

6 Match the statistic to the statement.

A The percentage of cases of PKD caused by the PKD1 mutation

B The percentage of cases of PKD caused by the PKD2 mutation

C The proportion of patients with PKD who have no known family history

D The prevalence of intracranial berry aneurysms in PKD patients over the age of 60

E The percentage of PKD patients affected by liver cysts

1 15%

2 20%

3 25%

4 35%

5 80%

7 Intracranial haemorrhage caused by rupture of an intracranial berry aneurysm is a life-threatening complication of PKD. It should be considered in any patient presenting with which features?

Time credits =

Impact credits =

8 List any changes to your clinical practice that you may make having read this article.

- B** Chronic primary pain represents chronic pain as a disease in itself
True ☐ False ☐
- C** A secondary chronic pain can coexist with a primary chronic pain
True ☐ False ☐
- D** Chronic primary pain can be a precursor to chronic secondary pain
True ☐ False ☐
- E** Chronic primary pain may not always improve despite best medical care
True ☐ False ☐

10 A How is chronic primary pain defined?

B What are the five categories of chronic primary pain?

11 Complete the statement.

A comprehensive

and

history should always be obtained to elucidate any potential

or possible

The combination of

history and

or

examination points towards a diagnosis of chronic primary pain.

Assessment must take into consideration any

or

12 List any changes to your clinical practice that you may make having read this article.

CHRONIC PRIMARY PAIN

9 Which of the statements are true?

A Chronic pain affects around a quarter of the UK population
True ☐ False ☐

Answers

persists for longer than three months and is not better accounted for by another diagnosis of chronic pain

10 B Chronic widespread pain; complex regional pain syndrome; chronic primary headache or orofacial pain; chronic visceral pain; chronic primary musculoskeletal pain

11 pain; medical; underlying cause; red flags. suggestive; normal; negative. disability; emotional; distress

5 A True **B** True **C** True **D** False (PKD mutation)

6 A 5 **B** 1 **C** 3 **D** 2 **E** 5

7 Thunderclap headache; new focal neurology; or unexplained syncope

10 A Pain in one or more anatomical regions that

1 A True **B** False (16 years) **C** True **D** True **E** True

2 A Poor glycaemic control; hypertension; smoking; hyperlipidaemia

2 B Genetic susceptibility; duration of diabetes; increasing age; male sex

3 Blood pressure; renal function [serum creatinine, eGFR]; urinary ACR

Reducing cardiovascular risk pivotal in diabetic kidney disease

Mkandawire MJ, Maxwell AP. Reducing cardiovascular risk pivotal in diabetic kidney disease.
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Reducing cardiovascular risk pivotal in diabetic kidney disease

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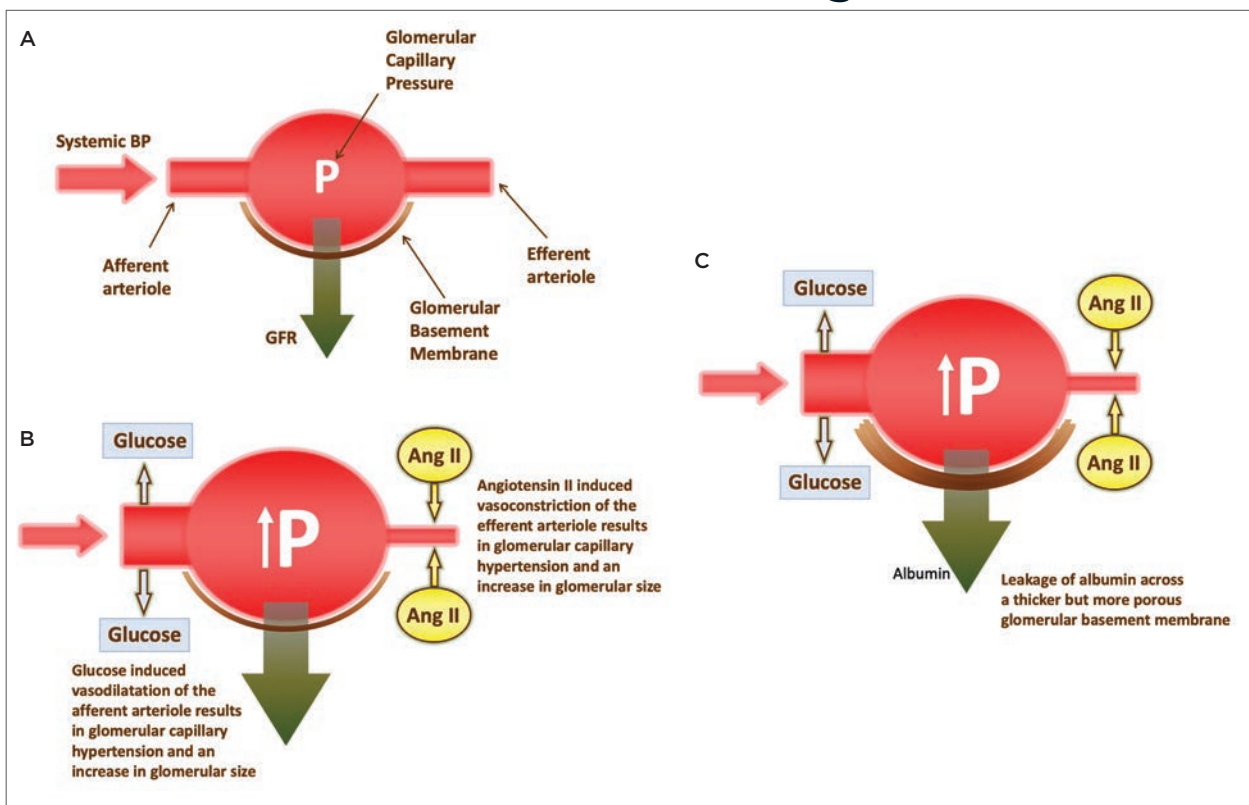
FIGURE 1

Glomerular haemodynamic changes in diabetic kidney disease

A Normal glomerular capillary pressure and glomerular filtration

B Glomerular capillary hypertension, glomerular hypertrophy and hyperfiltration associated with glucose induced vasodilatation of the afferent arteriole and angiotensin II mediated efferent arteriolar vasoconstriction

C Development of microalbuminuria associated with thickened but leaky glomerular basement membrane



How is diabetic kidney disease diagnosed?

What are the management approaches?

How should patients be monitored?



DIABETIC KIDNEY DISEASE IS THE MOST COMMON CAUSE OF CHRONIC KIDNEY DISEASE (CKD) AND THE

main cause of end-stage renal disease worldwide.¹

Clinical features of diabetic kidney disease can develop in up to 40% of individuals with diabetes.² The rise in diabetic kidney disease prevalence is predominantly in individuals with type 2 diabetes mellitus as it is more common than type 1 diabetes and the risk factors for CKD are clustered in older adults with type 2 diabetes.²

Diabetes is associated with a reduction in life expectancy of around six years³ but the combination of diabetes and cardiovascular disease (CVD) is associated with a reduction of around 12 years.³ Individuals with diabetic kidney disease have an even

greater reduction in life expectancy of around 16 years⁴ primarily because diabetic kidney disease is associated with a very high risk of CVD and premature mortality from cardiovascular events.^{2,3}

RISK FACTORS

There are genetic and environmental risk factors for diabetic kidney disease. Modifiable risk factors for diabetic kidney disease, and CVD, include poor glycaemic control, hypertension, smoking and hyperlipidaemia.⁵ Non-modifiable risk factors for diabetic kidney disease include genetic susceptibility, duration of diabetes, increasing age and male sex.⁵

Optimal blood glucose control coupled with effective blood pressure lowering can substantially reduce the risk of development and progression of

diabetic kidney disease.^{6,7} Diabetic kidney disease is a microvascular diabetic complication and typically occurs in association with diabetic retinopathy and neuropathy.^{5,6,7}

MECHANISMS

The clustering of diabetic microvascular and macrovascular complications is linked to common pathological mechanisms. These include formation of advanced glycation end products, increased oxidative stress, endothelial dysfunction, growth factor pathway disruption and accelerated atherosclerosis.⁸

Diabetic kidney disease is driven by abnormal renal haemodynamics that result in elevated intraglomerular capillary pressure (see figure 1, above). Individuals with diabetes can have a combination of afferent arteriole

SYMPOSIUM RENAL MEDICINE

DIABETIC KIDNEY DISEASE

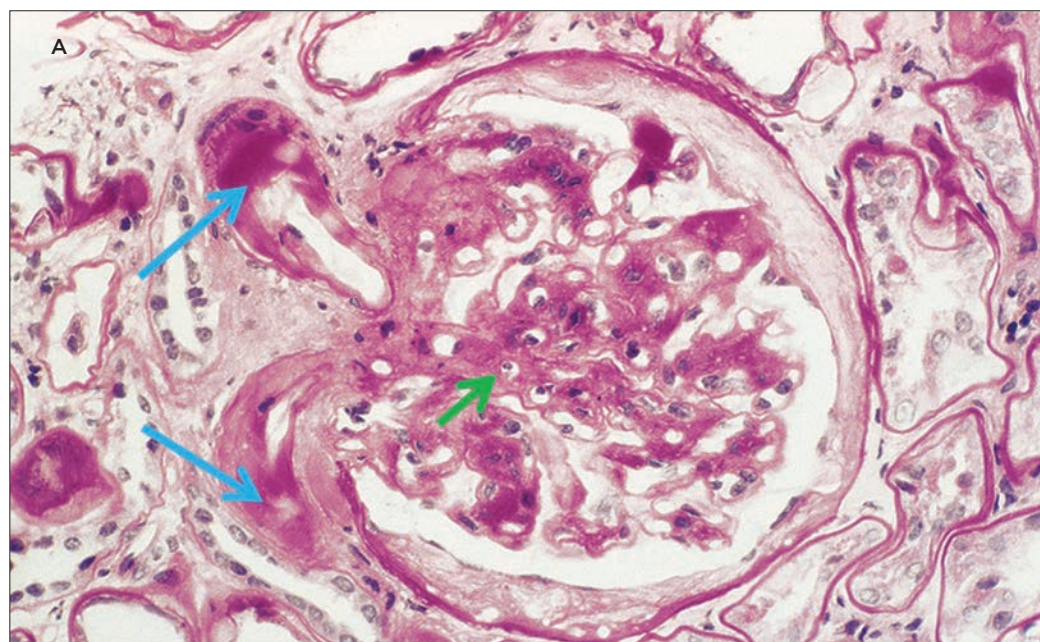
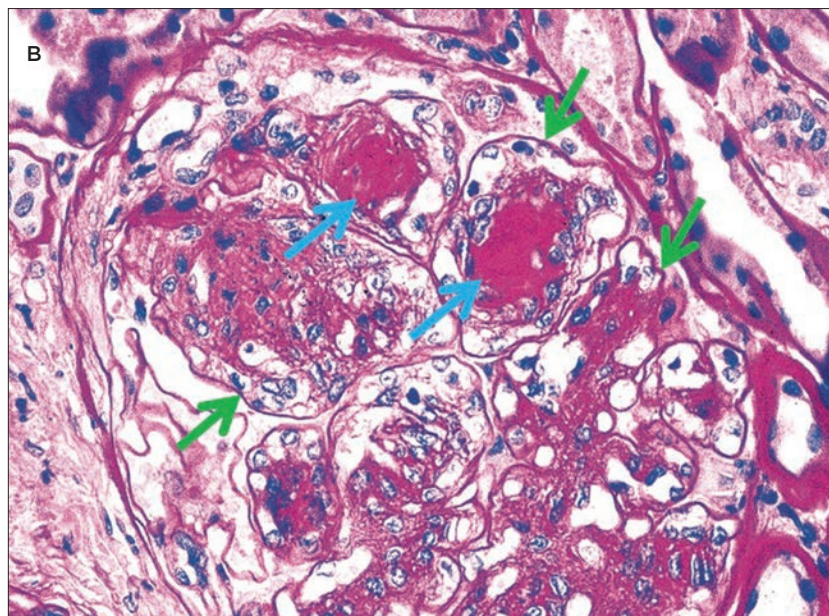


FIGURE 2
Glomerular histological changes in diabetic kidney disease

A Both afferent and efferent arterioles are extensively hyalinised or thickened due ultimately to the haemodynamic and toxic effects of the diabetic milieu (blue arrows). The supporting tissue or mesangium of the glomerulus is thickened (green arrow)

B The classic nodules seen in advanced diabetic kidney disease are well demonstrated (blue arrows). There is thickening of the glomerular basement membrane (green arrows) due to sustained hyperglycaemia and chronic glomerular capillary hypertension



dilatation (secondary to high glucose levels) and efferent arteriole vasoconstriction (secondary to high angiotensin II levels) that cause a rise in intraglomerular capillary pressure (see figure 1, p11).^{2,8} Other changes include glomerular basement membrane thickening and loss of podocytes (cells that are integral to the glomerular filtration barrier).² These pathological changes result in loss of glomerular barrier function detected clinically by albuminuria (see figure 1, p11) and eventually by a fall in glomerular filtration rate (GFR).

The afferent arteriole dilatation (of the glomerulus) can be reversed by SGLT2

inhibitors (SGLT2i). The efferent arteriole vasoconstriction can be modified by blockade of the renin-angiotensin-aldosterone system (RAAS).^{2,8}

DETECTION

Early diabetic kidney disease is asymptomatic and can be detected by persistently high urinary albumin (protein) excretion or sustained reduction in estimated glomerular filtration rate (eGFR) below 60 ml/min/1.73m².² The rise in urinary albumin excretion frequently precedes any fall in eGFR, therefore serum creatinine can be normal initially. Urinary albumin excretion is measured by the urinary

albumin: creatinine ratio (ACR) and the risk of diabetic kidney disease is directly related to the absolute amount of albumin excretion.

Microalbuminuria, defined as a urinary ACR of 3-30 mg/mmol on two occasions three months apart (see table 1, p13), is the earliest clinically detectable indicator of diabetic kidney disease.

Patients with diabetic kidney disease are diagnosed based on elevated urinary albumin excretion and/or reduced renal function. A renal biopsy (see figure 2, left) is rarely necessary to confirm diabetic kidney disease. Individuals with type 2 diabetes should have annual urinary ACR measurements from the time of diabetes diagnosis⁹ while annual ACR measurements should begin from the age of 12 in those with type 1 diabetes.¹⁰

REFERRAL

NICE has published chronic kidney disease (CKD) guidelines⁹ but has not produced separate guidance for diabetic kidney disease. Risk for cardiovascular events and/or progression to end-stage renal disease can be stratified by eGFR (G) and albuminuria (A) (see table 1, p13).

NICE guidelines suggest that patients with known or suspected CKD should be referred to a nephrologist in the following situations:

- eGFR < 30 ml/min/1.73 m² (i.e. CKD stages 4 and 5)
- ACR ≥ 70 mg/mmol despite maximum tolerated angiotensin converting enzyme inhibitor (ACEi) or angiotensin II receptor blocker (ARB) therapy
- Sustained decrease in GFR ≥ 25%, and a change in GFR category or sustained decrease in GFR ≥ 15 ml/min/1.73 m² within 12 months
- ACR ≥ 30 mg/mmol (ACR category A3), together with haematuria
- Uncontrolled hypertension on four antihypertensive drugs at therapeutic dose
- Suspicion of alternative CKD diagnosis (family history, kidney structural problem or renal stone disease)
- Suspected renal artery stenosis

NICE recommendations for frequency of clinical follow-up are based on CKD stage and amount of proteinuria.⁹

In 2020, Kidney Disease: Improving Global Outcomes (KDIGO) guidelines were published for diabetic kidney disease incorporating the recent evidence for benefits of SGLT2i in reducing cardiovascular and renal risks.¹¹ Multiple clinical trials of different SGLT2i

Table 1

Increasing risk for adverse outcomes (cardiovascular events and death) with decrease in eGFR and/or increase in urine albumin: creatinine ratio (ACR). Adapted from NICE CG182⁹

eGFR ml/min/1.73m ²	Albuminuria ACR	Albuminuria ACR	Albuminuria ACR
	A1 <3 mg/mmol	A2 3-30 mg/mmol	A3 >30 mg/mmol
G1 > 90	No CKD	G1 A2	G1 A3
G2 60-89	No CKD	G2 A2	G2 A3
G3a 45-59	G3a A1	G3a A2	G3a A3
G3b 30-44	G3b A1	G3b A2	G3b A3
G4 15-29	G4 A1	G4 A2	G4 A3
G5 <15	G5 A1	G5A2	G5 A3

Green = lowest risk red = highest risk

(CREDENCE, EMPA-REG, CANVAS, DAPA-HF) have demonstrated that these agents are associated with reduced rates of major adverse cardiovascular events, heart failure hospitalisation, cardiovascular death and composite renal outcomes (stabilisation of GFR and reduction in proteinuria).^{11,12}

MANAGEMENT

Ideally, patients should be empowered to achieve optimal control of blood glucose and understand how to minimise their individual modifiable risk factors for diabetic kidney disease.¹³ Unfortunately, fragmentary care can occur if the patient is attending multiple hospital clinics and receives conflicting advice.

There is good evidence that multifactorial interventions that optimise blood pressure, limit proteinuria and minimise cardiovascular risk can reduce morbidity and premature mortality.⁷ Smoking cessation, aerobic exercise, a healthy diet and weight control are all useful non-pharmacological components of diabetes care.

Excellent glycaemic control can delay onset of microalbuminuria.^{6,13} HbA_{1c} targets are individualised, e.g. < 53 mmol/mol (< 7%), in a patient with a long life expectancy, hypoglycaemia awareness and few comorbidities. In contrast, a more relaxed HbA_{1c} target can be adopted if the patient has end-stage renal disease, macrovascular complications, a short life expectancy and limited hypoglycaemia awareness.¹¹

For individuals with diabetes and albuminuria, a blood pressure target < 130/80 mmHg is advocated by NICE

guidelines.⁹ A reduction of proteinuria to < 1 g/day (ACR < 70 mg/mmol) retards progression of diabetic kidney disease and reduces the number of cardiovascular events.¹⁴

If proteinuria is still ≥ 1 g/day (ACR ≥ 70 mg/mmol) some guidelines have suggested a more stringent target BP of 125/75 mmHg particularly for younger patients and those with type 1 diabetes. A less stringent target systolic blood pressure of < 150 mmHg is appropriate in much older individuals to reduce risks associated with multiple antihypertensive drugs.

RAAS blockade is effective in reducing proteinuria, managing hypertension and reducing cardiovascular risk.

ACEi and ARB are considered equivalent in terms of efficacy.¹⁵ Both ACEi and ARB can lower intraglomerular capillary pressure as they reverse the angiotensin II mediated vasoconstriction of the glomerular efferent arteriole. Unless there are clear contraindications all patients with diabetic kidney disease should be prescribed an ACEi or ARB at the maximum tolerated dose.^{9,11}

KDIGO recommends prescribing SGLT2i for individuals with type 2 diabetes and eGFR ≥ 30 ml/min/1.73m².¹¹ SGLT2i can reduce intraglomerular capillary pressure by increasing the glomerular afferent arteriole tone.¹⁶ SGLT2i act by blocking sodium coupled glucose reabsorption in the proximal tubule resulting in increased sodium delivery to the distal tubule that in turn stimulates tubuloglomerular feedback and an increase in afferent arteriole tone.

SGLT2i are also associated with a modest reduction in weight and blood pressure (related to salt and water losses

associated with reduction in tubular glucose reabsorption).^{11,16} If the patient is at risk of hypovolaemia, consider reducing or stopping diuretics before starting SGLT2i treatment.¹¹ SGLT2i have been well tolerated in clinical trials and can continue to be used when eGFR is < 30 ml/min/1.73m².

SGLT2i are associated with an increased risk of genital fungal infections and a small increased risk of ketoacidosis.^{11,12,16} The effects of SGLT2i are additive to RAAS blockade with ACEi or ARBs.^{11,16}

After commencing a RAAS blocker some patients will experience an initial rise in serum creatinine. Provided this is not > 30% of baseline creatinine and potassium levels remain < 6 mmol/L RAAS blockade can be safely continued. Paradoxically, it has been observed that an initial fall in eGFR after RAAS blockade is predictive of a slower long-term decline in renal function.¹⁷

Treatment with an ACEi (or ARB) should be temporarily stopped during intercurrent illness or episodes of volume depletion (vomiting, diarrhoea, the perioperative period), see box 1, p14. Too great a reduction in intraglomerular pressure can lead to a rapid fall in GFR and acute kidney injury.

Patients with diabetic kidney disease and persisting proteinuria ≥ 1 g/day (ACR ≥ 70 mg/mmol) may require additional antihypertensive agents to achieve targets. Despite this, blood pressure and proteinuria may still be poorly controlled.

It is worth checking concordance with drug therapy as well as ensuring that dietary sodium restriction is maximised.

The effects of RAAS blockade can be blunted if sodium intake is too high.¹⁸ A daily sodium chloride intake of 5 g or

»

key points

SELECTED BY

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Diabetic kidney disease is the most common cause of chronic kidney disease (CKD) and the main cause of end-stage renal disease worldwide. The rise in diabetic kidney disease prevalence is predominantly in individuals with type 2 diabetes mellitus. Diabetic kidney disease is associated with a very high risk of cardiovascular disease (CVD) and premature mortality from cardiovascular events. Modifiable risk factors for diabetic kidney disease (and CVD) include poor glycaemic control, hypertension, smoking and hyperlipidaemia. Diabetic kidney disease is a microvascular diabetic complication and typically occurs in association with diabetic retinopathy and neuropathy.

Pathological changes result in loss of glomerular barrier function detected clinically by albuminuria and eventually by a fall in glomerular filtration rate (GFR). Early diabetic kidney disease is asymptomatic. Microalbuminuria, defined as a urinary ACR of 3–30 mg/mmol on two occasions three months apart is the earliest clinically detectable indicator of diabetic kidney disease. Diagnosis is based on elevated urinary albumin excretion and/or reduced renal function. Individuals with type 2 diabetes should have annual urinary ACR measurements from the time of diabetes diagnosis while annual ACR measurements should begin from the age of 12 in those with type 1 diabetes.

There is good evidence that multifactorial interventions that optimise blood pressure (BP), limit proteinuria and minimise cardiovascular risk can reduce morbidity and premature mortality. Excellent glycaemic control can delay onset of microalbuminuria. HbA_{1c} targets are individualised. For patients with diabetes and albuminuria, a BP target < 130/80 mmHg is advocated by NICE.

RAAS blockade is effective in reducing proteinuria, managing hypertension and reducing cardiovascular risk. Angiotensin-converting enzyme inhibitors (ACEi) and angiotensin II receptor blockers (ARBs) are equivalent in terms of efficacy. Unless there are clear contraindications, all patients with diabetic kidney disease should be prescribed an ACEi or ARB at maximum tolerated dose.

The effects of SGLT2i are additive to RAAS blockade with ACEi or ARBs. Treatment with an ACEi or ARB should be temporarily stopped during intercurrent illness or episodes of volume depletion (vomiting, diarrhoea, the perioperative period). Dual RAAS blockade with an ACEi and ARB is not recommended.

Precision of HbA_{1c} measurement decreases in advanced CKD (eGFR < 30 ml/min/1.73m²) and especially in dialysis patients (with shorter red cell survival and unpredictably prolonged half-life of several hypoglycaemic drugs). All patients with diabetic kidney disease should have at least yearly measurements of BP, renal function (serum creatinine; eGFR) and urinary ACR. The NICE guidelines for CKD are useful in deciding when to refer patients with diabetic kidney disease to a nephrologist for joint care.

Box 1

Medicine sick day rules that may reduce the risk of acute kidney injury

When you are unwell with any of the following:

- Vomiting or diarrhoea
- Fevers, sweats or shaking

Then stop the drugs listed below. Restart these drugs when you are well (after 24–48 hours eating and drinking normally)

Medications to stop temporarily

- ACE inhibitors: (drug names ending in 'pril')
- ARBs: (drug names ending in 'sartan')
- NSAIDs: anti-inflammatory painkillers; e.g. ibuprofen, diclofenac, naproxen
- Diuretics: e.g. bendroflumethiazide, furosemide, spironolactone
- Metformin

less is recommended.^{9,11} Great care should be taken if using salt substitutes containing potassium chloride, as individuals with diabetic kidney disease taking RAAS inhibitors may develop dangerous hyperkalaemia.

Addition of a diuretic to aid sodium excretion can be useful particularly for oedematous patients. Dual RAAS blockade with an ACEi and ARB is not recommended because of the risk of hyperkalaemia and acute kidney injury.

As GFR falls, dose reduction is required for some drugs.²⁴ Metformin can be continued at reduced dose when eGFR is < 45 ml/min/1.73m² but is usually discontinued when eGFR < 30 ml/min/1.73m².¹¹ Metformin should be temporarily stopped in the setting of acute illness such as sepsis, volume depletion or acute cardiac events as there is a small but important risk of lactic acidosis.

Glucagon-like peptide receptor agonists (GLP-1 RA) can be helpful in patients with type 2 diabetes and diabetic kidney disease who have not achieved HbA_{1c} targets despite metformin and SGLT2i (or who are unable to use those drugs).¹¹ Several GLP-1 RA drugs do not need dose adjustments for CKD but there is limited data on their use in severe CKD (eGFR < 15 ml/min/1.73m²). GLP-1 RA should not be used in combination with dipeptidyl peptidase-4 (DPP-4) inhibitors.

Insulin, as it is metabolised by the kidney, may accumulate as GFR falls and so dosing requirements may need to be reduced.

Sulphonylureas should be used with caution as even the 'short acting' analogues can have prolonged duration of action in CKD. This increases the risk of hypoglycaemia and potential accidents e.g. while driving.

Thiazolidinediones are associated

with fluid retention and increased cardiovascular risk making other oral hypoglycaemic classes more attractive.

One important practice point to bear in mind is that the accuracy and precision of HbA_{1c} measurement decreases in advanced CKD (eGFR < 30 ml/min/1.73m²) and especially in dialysis patients (with shorter red cell survival and unpredictably prolonged half-life of several hypoglycaemic drugs).¹¹

MONITORING AND FOLLOW-UP

Individuals with early diabetic kidney disease (CKD stages 1–3) will typically have stable or very slowly progressive loss of renal function.

Arguably, the major focus of care for patients with diabetic kidney disease should be on limiting the excess cardiovascular risk. Statin therapy for primary prevention of CVD is recommended for individuals with type 1 diabetes who have had diabetes for at least ten years or who are older than 40 years of age.¹⁹ Most individuals with type 2 diabetes and diabetic kidney disease will have a 10% or greater ten-year risk of CVD and would be suitable for statin therapy.¹⁹

All patients with diabetic kidney disease should have at least yearly measurements of blood pressure, renal function (serum creatinine; eGFR) and urinary ACR.

The NICE guidelines for CKD are helpful in deciding when to refer a patient with diabetic kidney disease to a nephrologist for shared care.⁹ Nephrology clinics can help with longer term monitoring of renal function, patient education and discussion about renal replacement therapy choices for end-stage renal disease (dialysis, renal transplantation, conservative care).^{20,21}

The recent SGLT2i clinical trials provide evidence for important

benefits of these drugs in reducing cardiovascular and renal morbidity and mortality.

Competing interests: None

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Useful information

For healthcare professionals KDIGO guidelines

2020 Clinical practice guideline for diabetes management in CKD
Summary of recommendations and practice points, pp S22-S29
[www.kidney-international.org/article/S0085-2538\(20\)30718-3/fulltext](http://www.kidney-international.org/article/S0085-2538(20)30718-3/fulltext)

NICE guidelines

NG28. Type 2 diabetes in adults: management
www.nice.org.uk/guidance/ng28

CG182. Chronic kidney disease in adults: assessment and management
www.nice.org.uk/guidance/cg182

Edinburgh Renal Unit

Practical guidance for GPs
<https://edren.org/ren>

For patients

Diabetes UK: Kidneys (nephropathy)
www.diabetes.org.uk/guide-to-diabetes/complications/kidneys_nephropathy

Patient Info

Diabetic kidney disease
<https://patient.info/diabetes/diabetes-mellitus-leaflet/diabetic-kidney-disease>

Edinburgh Renal Unit

Diabetic kidney disease
<https://edren.org/ren/handbook/diabetic-kidney-disease>

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Tight blood pressure control key in polycystic kidney disease

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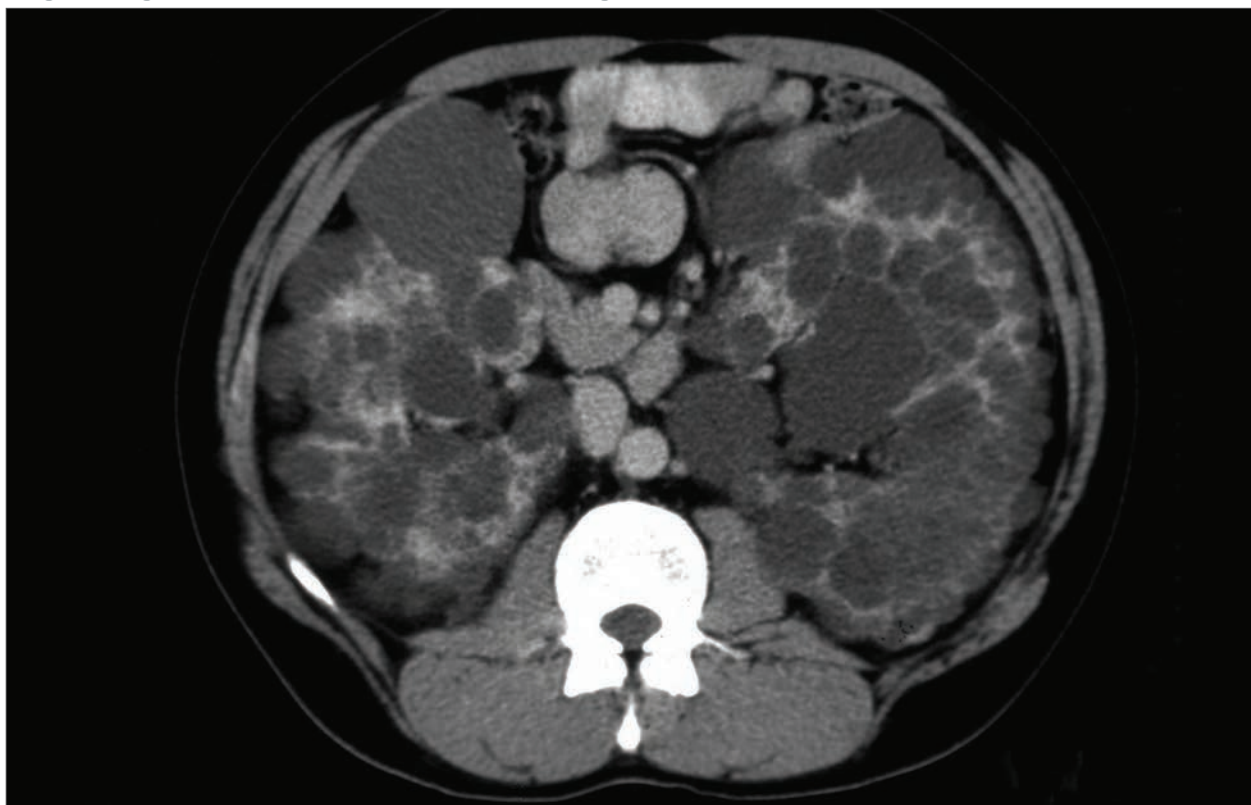


FIGURE 1
CT scan of bilateral renal cysts and enlarged kidneys in polycystic kidney disease

How does polycystic kidney disease present?

What are the potential complications?

What are the management strategies?



POLYCYSTIC KIDNEY DISEASE (PKD) IS THE MOST COMMON CAUSE OF INHERITED KIDNEY DISEASE

worldwide.

A recent meta-analysis estimated prevalence as < 5 per 10,000 population.¹ Despite being a rare disease it accounts for 7% of cases of end-stage renal disease (ESRD) in the UK annually.²

PKD can be asymptomatic and should be considered in any patient with renal cysts noted on abdominal imaging, especially if they have a history of hypertension.

PKD is a genetic disorder with an autosomal dominant mode of inheritance, hence any child of an affected parent has a 50% chance of inheriting the condition.³ Despite this 25% of patients will have no known family history and these cases are

thought to be due to de novo mutations in the causative genes.³

‘Polycystic kidney disease can be asymptomatic and should be considered in any patient with renal cysts noted on abdominal imaging’

PKD is caused by mutations in the genes coding polycystins, proteins involved in the function of cell membranes of renal tubules and other organs.⁴

The vast majority (95%) of cases are caused by one of two gene mutations. Mutation of the PKD1 gene (located on chromosome 16) accounts for around 80%, while mutation of the PKD2 gene (located on chromosome 4) accounts for around 15%.⁵

The PKD1 mutation is associated with a more aggressive phenotype. Median age at onset of ESRD is 54 years for patients with a PKD1 mutation compared with 69 years for patients with a PKD2 mutation.⁶

However, considerable variation is seen, even within families with the same mutation, suggesting that other environmental factors may also influence disease progression.

PRESENTATION

PKD is a multisystem disease characterised by renal and extrarenal »

features (see table 1, below), however in the early stages it can be asymptomatic. It can present in a variety of ways.

Incidental finding

Multiple renal cysts may be seen on radiographic scans performed for alternative indications. The ultrasound criteria for the diagnosis of PKD are summarised in table 2, below, and are dependent upon the number of cysts and their distribution between both kidneys.⁷

Family history

Given the inherited nature of PKD, patients may present requesting investigation for diagnosis prior to the onset of symptoms. A general practice may have several generations of an affected family on its register.

Table 1

Clinical features of polycystic kidney disease

Renal

- Haematuria (visible or non-visible)
- Hypertension
- Loin pain
- Cyst haemorrhage and/or cyst infections
- Renal calculi
- Chronic kidney disease progressing to end-stage renal disease
- Enlarged and palpable kidneys

Extrarenal

- Liver cysts and liver pain
- Cerebral haemorrhage
- Cardiac valve problems (e.g. mitral valve prolapse)
- Seminal vesicle cysts (males)
- Diverticulosis
- Ovarian cysts (females)
- Hepatomegaly

Hypertension

Patients with PKD may present with hypertension before a decline in eGFR is noted.

Chronic kidney disease (CKD)

As PKD progresses there is gradual loss of kidney function, with a predictable decrease in eGFR (falling annually by up to 5 ml/min/1.73m²).⁸ CKD may be identified on routine blood tests performed for other reasons.

The size and number of renal cysts will continue to increase with time leading to localised pressure effects and damage to surrounding normal renal parenchyma.

Risk factors for more rapid progression include younger age at diagnosis, large kidney volume, hypertension, male gender, visible haematuria and inheritance of a PKD1 mutation.

Cyst complications

Cysts and their complications account for many of the symptoms that patients experience. Cyst haemorrhage, rupture or infection can all present with acute loin pain. Infections can be severe and associated with systemic upset and sepsis. Bleeding can occur into cysts and as a result may not be 'visible' but can be sufficiently severe to cause symptomatic anaemia. Visible haematuria can occur in the setting of cyst rupture or haemorrhage if the involved cyst communicates with the urinary tract.

DIAGNOSIS

Both PKD genes are very large and many mutations are described in each gene. This makes routine genetic testing difficult. It is often unnecessary in those with a family history of PKD. Genetic testing may be considered in patients who have no family history, younger relatives of affected individuals who wish to become kidney donors or patients who have features of other renal cystic disorders.

DNA sequencing techniques are rapidly evolving, and it may soon be possible to offer routine DNA sequencing for PKD patients.

At present, diagnosis is primarily made on the basis of ultrasound sonography showing evidence of renal cysts⁷ (see table 2, below) and family history. Computerised tomography (CT), see figure 1, p17, and magnetic resonance imaging (MRI) can also be used to identify smaller cysts as they can achieve much greater resolution than ultrasound. However, these imaging modalities are not routinely used for diagnosis.

'Greater increases in total kidney volume have been associated with faster decline in eGFR'

MRI measurement of the total kidney volume is a novel technique. Greater increases in total kidney volume have been associated with faster decline in eGFR.⁹

COMPLICATIONS

PKD can cause a number of extrarenal complications.

Liver cysts

In addition to renal cysts, cysts can also develop in other organs. The liver is by far the most common site for extrarenal cysts affecting around 80% of PKD patients.

Cysts can cause liver capsular pain, early satiety, nausea and vomiting.¹⁰ They rarely compromise hepatic function but can become infected and may require extended courses of antibiotics.

Intracranial aneurysms

Intracranial haemorrhage due to rupture

Table 2

Ultrasound criteria for the diagnosis of polycystic kidney disease in patients with a positive family history, adapted from Pei et al ⁷

Age (years)	PKD1 genotype	Sensitivity	PKD2 genotype	Sensitivity	Unknown genotype	Sensitivity
15-29	3 or more cysts	94.3%	3 or more cysts	69.5%	3 or more cysts	81.7%
30-39	3 or more cysts	96.6%	3 or more cysts	94.9%	3 or more cysts	95.5%
40-59	2 or more cysts in each kidney	92.6%	2 or more cysts in each kidney	88.8%	2 or more cysts in each kidney	90%
60 +	4 or more cysts in each kidney	100%	4 or more cysts in each kidney	100%	4 or more cysts in each kidney	100%

key points

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Polycystic kidney disease (PKD) is the most common cause of inherited kidney disease worldwide. It has a prevalence of < 5 per 10,000 population and accounts for 7% of cases of end-stage renal disease (ESRD) in the UK. PKD is a genetic disorder with an autosomal dominant mode of inheritance; hence any child of an affected parent has a 50% chance of inheriting the condition. Despite this 25% of patients will have no known family history and these cases are thought to be due to de novo mutations in the causative genes. Routine genetic testing is often unnecessary in those with a family history of PKD. Diagnosis is primarily made on the basis of a positive family history plus an age-specific minimum number of cysts on ultrasonography.

PKD can present asymptotically and should be considered in any patient with renal cysts noted on abdominal imaging, especially if there is a history of hypertension. Patients with PKD may present with hypertension before a decline in eGFR is noted. As PKD progresses there is gradual loss of kidney function, with a predictable decrease in eGFR.

Cyst haemorrhage, rupture or infection can all present with acute loin pain. Infections can be severe and associated with systemic upset and sepsis. Bleeding can occur into cysts and as a result may not be 'visible' but can be sufficiently severe to cause symptomatic anaemia. Visible haematuria can occur in the setting of cyst rupture or haemorrhage if the involved cyst communicates with the urinary tract.

PKD can cause a number of extrarenal complications. Liver cysts affect around 80% of PKD patients and can cause liver capsular pain, early satiety, nausea and vomiting. Intracranial haemorrhage due to rupture of an intracranial berry aneurysm is a potentially life-threatening complication of PKD and should be considered in any patient presenting with thunderclap headache, new focal neurology or unexplained syncope. Mitral valve prolapse is common and other valvular and aortic abnormalities can occur.

Angiotensin converting enzyme inhibitors or angiotensin receptor blockers are the first choice antihypertensive agents in PKD. The target clinic blood pressure should be < 130/80 mmHg. Lipid-soluble antibiotics, such as ciprofloxacin, are effective for cyst infections as they achieve better cyst penetration than non-lipid soluble agents. Tolvaptan, a vasopressin receptor antagonist, can be used in some PKD patients and can slow cyst development and the rate of decline in renal function.

The median age at onset of ESRD is 54 years for patients with a PKD1 mutation compared with 69 years for patients with a PKD2 mutation. Planning for renal replacement therapy (kidney transplant or dialysis) should start well in advance of the onset of ESRD.

of an intracranial berry aneurysm is a potentially life-threatening complication of PKD and should be considered in any patient presenting with thunderclap headache, new focal neurology or unexplained syncope. The prevalence of aneurysms increases with age and they may be present in as many as 20% of patients over 60 years.¹¹

Despite their prevalence there is no consensus on screening for intracranial haemorrhage in PKD patients. Screening is considered on an individual patient basis particularly if there are risk factors such as a strong family history of subarachnoid haemorrhage and/or neurological symptoms.

Cardiovascular abnormalities

Cardiac valvular abnormalities are frequently associated with PKD. Mitral valve prolapse is the most common abnormality although other valvular and aortic abnormalities can occur.

Fertility and pregnancy

Female fertility is not impaired unless patients have advanced CKD. Male fertility may be impaired if there are cysts in the seminal vesicles or prostate. Pregnancy can be complicated by higher rates of hypertension, pre-eclampsia, preterm birth and progression of CKD so pre-pregnancy counselling is recommended. Counselling should include discussion about the risk of transmission of PKD to offspring.

Psychological impact

PKD is a chronic, progressive disease that impacts on patients' mental health as well as their physical health.¹² A UK study found that 22% of PKD patients had evidence of depressive symptoms.¹³ Patients are living with a disorder that they may have seen other family members suffer from. They may fear progressive CKD and the risk of needing dialysis or transplantation in the future.

Patients often live with chronic pain caused by their renal and liver cysts and analgesic options may be limited in those with more advanced CKD.

MANAGEMENT

Hypertension

Tight blood pressure control is one of the cornerstones of PKD management. Hypertension frequently occurs prior to any reduction in eGFR in PKD. Adopting a healthy lifestyle (weight loss, low salt diet, exercise and reduction in alcohol intake) should be advised. However, pharmacological treatment will usually be required at some point.

Angiotensin converting enzyme inhibitors (ACEi) or angiotensin receptor blockers (ARB) are the first choice antihypertensive agents in PKD. Hypertension in PKD is partly mediated by excess renin and angiotensin II. Patients should be commenced on either an ACEi or ARB and it should be gradually titrated up to the maximum tolerated dose.¹⁴

'Hypertension often occurs prior to any reduction in eGFR in polycystic kidney disease'

Treatment should be aimed at achieving a clinic blood pressure < 130/80 mmHg. Calcium channel blockers can be added as a second-line agent.

ADH secretion appears to stimulate more rapid cyst growth, increase in kidney volume and faster decline in eGFR in PKD. Although thiazide or thiazide-like diuretics are normally used as third-line antihypertensive therapy in other patients with hypertension they should be avoided in PKD. Thiazides (and other diuretics) cause volume depletion and stimulate excess ADH secretion. In contrast, PKD patients are advised to maintain a high fluid intake to suppress ADH as much as possible.

Cyst infections

Cyst infections can be difficult to distinguish from simple cyst rupture or haemorrhage as they can all cause loin pain. However, cyst infection is associated with persistent pain, systemic symptoms and signs (e.g. pyrexia) or even septic shock.

Infections can be difficult to eradicate as cysts are 'walled off' from the renal parenchyma and do not communicate with the urinary tract. Lipid-soluble antibiotics, such as ciprofloxacin, are effective treatments for cyst infections as they achieve better cyst penetration than non-lipid soluble agents.¹⁵

Disease specific agents

As discussed earlier, ADH (vasopressin) stimulates accelerated cyst growth and faster decline in renal function. Tolvaptan specifically blocks the vasopressin (V2) receptor in the kidney. In a large randomised controlled trial, tolvaptan reduced the rate of increase in kidney volume and also reduced the rate of loss of renal function.⁹ Due to its »

SYMPOSIUM RENAL MEDICINE

POLYCYSTIC KIDNEY DISEASE

mechanism of action, the most common side effects noted were thirst and polyuria.

Tolvaptan therapy can also affect liver function, therefore monthly monitoring with liver function tests is required for the first 18 months of treatment. NICE published guidance on tolvaptan in 2015 and approved its use in patients with PKD who have CKD stage 2 or 3 (eGFR > 30 ml/min/1.73m²) and evidence of rapidly progressive renal disease.¹⁶

Surgical intervention

Surgery is rarely required in PKD. Occasionally, very large cysts may be derooft if they are causing chronic pain although it can often be difficult to identify one causative cyst among a mass of others.

In a small number of cases, kidney size may be so massive that there is no space left in the abdomen for a transplant. Nephrectomy can be considered in this situation although this can result in patients with poor baseline renal function needing to start dialysis. For this reason, if nephrectomy is required, it is usually deferred until the patient has commenced dialysis.

End-stage renal disease

Many patients will eventually progress to ESRD. Planning for renal replacement therapy (kidney transplant or dialysis) starts well in advance of this occurring. Ideally, if the patient has a suitable live donor available then they can be transplanted before requiring dialysis (preemptive transplantation).

However, if they have no suitable live donor then a period of dialysis therapy may be required while awaiting a deceased donor transplant. Kidney transplantation (live donor and deceased donor) is associated with excellent outcomes in PKD.¹⁷

CONCLUSION

PKD is the most common inherited cause of ESRD. It is associated with multisystem complications as well as progressive CKD. Management includes tight blood pressure control, prompt treatment of complications such as cyst infections and, in some cases, use of tolvaptan to slow progression of CKD.

Competing interests: None

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Useful information

For patients

Polycystic Kidney Disease charity
www.pkdccharity.co.uk/

Patient.co.uk

www.patient.co.uk/health/Polycystic-Kidney-Disease.htm

NIDDK

www.niddk.nih.gov/health-information/kidney-disease/polycystic-kidney-disease/what-is-pkd

Edinburgh Renal Unit

<https://edren.org/ren/edren-info/polycystic-kidney-disease-pkd>

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Holistic approach needed to tackle chronic primary pain

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Holistic approach needed to tackle chronic primary pain

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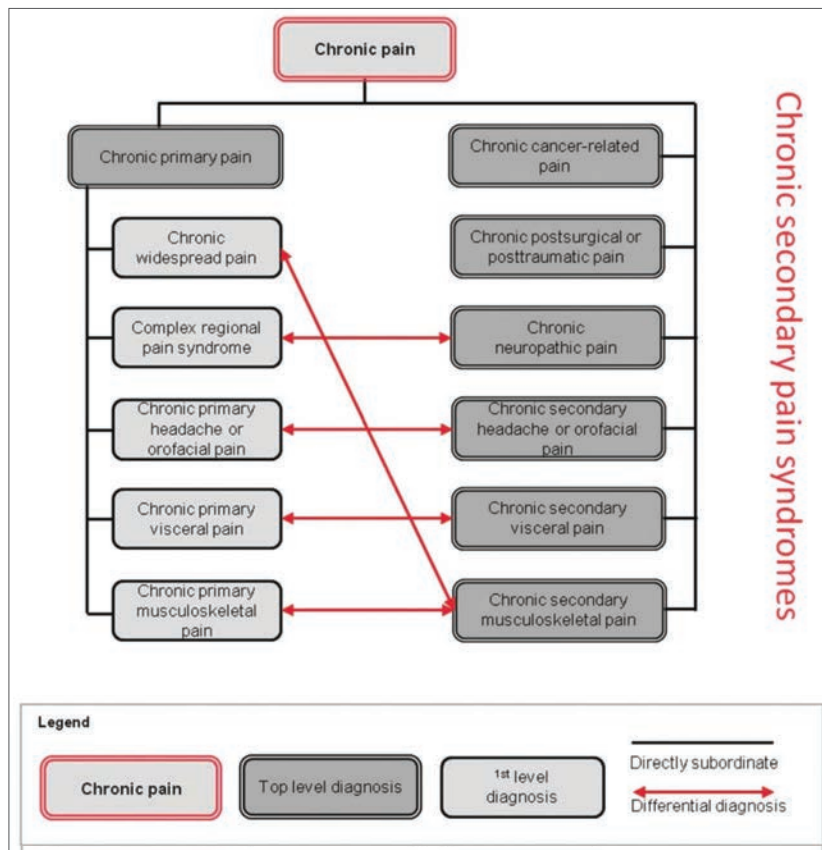
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FIGURE 1

IASP classification of chronic pain.⁵ Chronic primary pain syndromes can be considered as a disease, whereas secondary pain syndromes initially manifest as a symptom of another diagnosis. For example, chronic neuropathic pain resulting from diabetic neuropathy



CHRONIC PAIN IS A LEADING SOURCE OF SUFFERING AND DISABILITY. IT IS DEFINED

as pain that lasts longer than three months and has been estimated to affect between one third and one half of the UK population.²

NEW DIAGNOSTIC CLASSIFICATION

In May 2019, the World Health Organization (WHO) adopted the International Classification of Diseases 11th Revision (ICD-11). ICD-11 is the first version of the coding manual to include a classification system for chronic pain.³

Prior to this, the lack of appropriate diagnostic codes rendered accurate epidemiological investigation of chronic pain difficult, potentially impeding health policy decisions such as financing access to multimodal pain management. ICD-11 was developed by a working group from the International Association for the Study of Pain (IASP) and is based

on current scientific evidence and the biopsychosocial model.⁴

Chronic primary pain represents chronic pain as a disease in itself.⁵ It is defined as pain in one or more anatomical regions that persists for longer than three months and is associated with disability or emotional distress not better accounted for by another diagnosis of chronic pain.⁶

The IASP recommended that the diagnostic entities are subdivided into five groups (see figure 1, above):

- Chronic widespread pain (e.g. fibromyalgia)
- Complex regional pain syndrome
- Chronic primary headache (e.g. chronic migraine) or orofacial pain
- Chronic primary visceral pain (e.g. irritable bowel)
- Chronic primary musculoskeletal pain (e.g. chronic primary low back pain).⁴

Another six subgroups of pain were also identified and can be characterised as secondary to an underlying disease.⁴

It is hoped that the development of a

How is chronic pain classified?

How should patients be assessed?

Which patients should be referred?

more robust coding system will lead to greater recognition of chronic pain as a public health problem, and ultimately improve access to adequate pain treatment for patients worldwide.

ASSESSMENT

The assessment of chronic primary pain, and pain in general, involves a comprehensive biopsychosocial approach.

Chronic primary pain is a diagnosis that requires thorough medical assessment before being confidently made. Patients may be labelled as having chronic primary pain while ongoing assessment is being carried out. They may ultimately be classified as having a chronic secondary pain syndrome as illustrated in figure 1, above. It is possible for a secondary chronic pain to coexist with a primary chronic pain. For example, a patient may be under investigation for fibromyalgia because their proven osteoarthritis or rheumatoid arthritis does not fully

explain their widespread pain. This allows coding for, and thus recording of, the workload created by pain patients in primary care, and epidemiological data to be gathered which will inform future service provision.

INDIVIDUAL ASSESSMENT

Central to the assessment of a patient presenting with chronic pain should be an attempt to get to know the patient as an individual. Active participation and shared decision making fosters a collaborative relationship which can be vital in managing what is ultimately a chronic condition.

‘There is evidence that exercise both reduces pain and improves quality of life’

A comprehensive pain and medical history should always be obtained to elucidate any potential underlying cause or possible red flags that may be present. The combination of suggestive history and normal or negative examination points towards a diagnosis of chronic primary pain.

To fulfil the second half of the definition, assessment must take into consideration any disability or emotional distress. It is important to ask the patient how the pain affects their life, and how their life may affect the pain. This may include their lifestyle and day to day activities, including work and sleep disturbance.

Attention should also be paid to both physical and emotional wellbeing, social interactions and relationships. Identifying any disability and emotional distress in the context of negative history, examination and investigation allows the diagnosis of chronic primary pain to be made. Investigations are guided by the individual presentation, and appropriateness deemed by the assessing clinician.

It is important to get an idea of not only the patient’s understanding (and acceptance) of their condition, but also that of their family, carers and significant others. It is important to discuss their ideas concerning what causes the pain, what might happen in the future and outcomes of any treatment in terms of pain control and quality of life. This will often lead to the healthcare professional acknowledging the fact that pain may not improve, despite best medical care.

Ultimately a care plan should be developed where the possible benefits, risks and uncertainties of all management options are discussed. It is especially important to be sensitive when communicating normal or negative test results in order to avoid invalidating the patient’s experience of pain.

Most importantly, the patient should be ready to engage actively in supported self-management.

MANAGEMENT

NICE draft guidance on chronic pain, published in 2020, has made several recommendations for the management of chronic primary pain.⁷ NICE suggests using these recommendations alongside their existing guidelines for headache, low back pain and irritable bowel.^{8,9,10}

The draft NICE guidance has resulted in much discussion within the pain community.^{11,12} The Faculty of Pain Medicine has raised concerns surrounding the confusing terminology in the document and the possible overemphasis of evidence for acupuncture in the management of chronic primary pain. The confusion surrounding terminology relates to the failure to distinguish clearly between chronic pain and chronic primary pain. Even the title of the draft guidance references chronic pain as the subject, while the majority of the document relates to the management of chronic primary pain.¹¹

Crucially, it does not include the flowchart (see figure 1, p23) which more clearly illustrates that chronic primary pain can be used as part of a differential diagnosis while ongoing assessment is being carried out. It is imperative that all patients with chronic primary pain are thoroughly assessed to avoid the risk of misdiagnosis.

Giving patients a final diagnosis of chronic primary pain, without adequate investigation, could result in the mismanagement of specific pain syndromes. This diagnostic term is new and so is not included in the established pain research literature. Consequently, much of the treatment evidence base is excluded with the conclusion that few treatments for this condition are useful. This risks the blanket withdrawal of effective treatments, which are already established.

In terms of non-pharmacological interventions, the principal recommendation is to encourage patients to exercise either in the form of a supervised group programme or by carrying on with their usual exercise.

There is evidence that exercise both

reduces pain and improves quality of life.¹³ Most of this evidence is derived from studies involving fibromyalgia and chronic neck pain and is consistent with the European League Against Rheumatism recommendations for the management of fibromyalgia.¹⁴

Other draft recommendations include consideration of psychological interventions such as cognitive behaviour therapy (CBT) or acceptance and commitment therapy (ACT). ACT has been found to improve quality of life and sleep, and reduce pain and psychological distress.¹⁵ Evidence for CBT pertains to improvement in quality of life only, but there is not enough evidence to support a preference for either CBT or ACT.⁷

Acupuncture delivered in a community setting has also been proposed. Acupuncture has been found to reduce pain and improve quality of life in the short term (3 months) when compared with usual care or sham intervention.¹⁶

‘Chronic primary pain can be a precursor to chronic secondary pain’

Non-pharmacological measures which NICE could not find evidence to recommend include, transcutaneous electrical nerve stimulation (TENS) or inferential therapy, ultrasound, biofeedback or manual therapy.

NICE has made research recommendations for mindfulness and psychotherapy where there appears to be a signal of benefit, but a substantial enough evidence base has yet to be established.

Pharmacological options also have very limited recommendations in the draft NICE guidance. It specifies that off-label prescribing of antidepressants should be considered; specifically, either duloxetine, fluoxetine, paroxetine, citalopram, sertraline or amitriptyline. Evidence indicates that they can lead to improvements in quality of life, pain and psychological distress when compared with placebo.⁷

The draft NICE guidance recommends avoiding opioids, benzodiazepines, NSAIDs, gabapentinoids (unless in a clinical trial or for complex regional pain syndrome), local anaesthetics by any route, corticosteroids, paracetamol, ketamine and antipsychotics.

The recommendation regarding gabapentinoids, specifically pregabalin,

key points

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Chronic pain is defined as pain that lasts longer than three months. It has been estimated to affect between one third and one half of the UK population. A working group from the International Association for the Study of Pain has developed a new classification system for chronic pain. A fundamental distinction is made between chronic primary pain and chronic secondary pain.

Chronic primary pain is defined as pain in one or more anatomical regions that persists for longer than three months, is associated with disability or emotional distress, not better accounted for by another diagnosis of chronic pain. Chronic primary pain is subdivided into five categories: chronic widespread pain (e.g. fibromyalgia); complex regional pain syndromes; chronic primary headache (e.g. chronic migraine) or orofacial pain; chronic primary visceral pain (e.g. irritable bowel syndrome); and chronic primary musculoskeletal pain (e.g. chronic primary low back pain).

Chronic secondary pain is pain secondary to an underlying disease. There are six categories: chronic cancer-related pain; chronic postsurgical or post-traumatic pain; chronic neuropathic pain; chronic secondary headache or orofacial pain; chronic secondary visceral pain; and chronic secondary musculoskeletal pain.

Chronic primary pain is a diagnosis that requires thorough medical assessment before being confidently made. Patients may be labelled as having chronic primary pain while ongoing assessment is being carried out. They may ultimately be classified as having a chronic secondary pain syndrome. It is possible for a secondary chronic pain to coexist with a primary chronic pain. For example, a patient may be under investigation for fibromyalgia because their proven osteoarthritis or rheumatoid arthritis does not fully explain their widespread pain.

Assessment of chronic pain should include a comprehensive pain history, medical history and examination to identify any potential underlying cause or possible red flags. It is important to ask about effects on lifestyle and day to day activities, including work and sleep disturbance; physical and emotional wellbeing; social interactions and relationships. The presence of disability and emotional distress in the context of negative history, examination and investigations allows the diagnosis of chronic primary pain to be made.

Referral to the pain management service should be considered when pain or function have not improved following appropriate primary care management, there is significant distress and where specialist assessment may be useful.

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contradicts that of the SIGN guideline on chronic pain (SIGN 136) where pregabalin is specifically recommended in the treatment of fibromyalgia.¹⁷ SIGN 136 supports the assertion in the draft NICE guidance for the use of antidepressants in chronic primary pain in the form of fibromyalgia.

The draft guidance also recommends that if a patient with chronic primary pain is already prescribed any of the above listed medications, the risks of continuing should be discussed. If the shared decision is to stop the medication, withdrawal effects must be considered especially with opioids, antidepressants, benzodiazepines and gabapentinoids.

REFERRAL

Referral to the pain management service should be considered when pain or function have not improved following appropriate primary care management, there is significant distress and where specialist assessment may be useful.¹⁷

It has been reported that longer delays between specialist referral and specialist consultation result in poorer health and pain management. This deterioration starts as early as five weeks from referral and there is consensus that a delay of longer than six months is clinically unacceptable.¹⁸ Depending on local arrangements, it may be that this is the only way of accessing specialist pain psychology for the provision of ACT or CBT.

CONCLUSION

It is hoped that the implementation of the new classification system will allow pain to be recognised as a centrally important condition in primary care and allow for the capacity to measure incidence, prevalence and impact of pain locally, nationally and internationally.

It should provide the opportunity to match evidenced based treatment to chronic pain subtypes.

At the time of writing the NICE guideline remains in draft form, and the recommendations have not been finalised for adoption in day to day practice. While some of the preliminary recommendations are controversial it is hoped that the final published guideline will provide GPs with support to emphasise the limited effectiveness of medication in chronic primary pain, and the importance of self-management.

Competing interests

Dr James McGuinness has no competing interests. Dr Mick Serpell is an examiner for the FFPMRCA for the Royal College of Anaesthetists. He has received honoraria from Astellas, Grünenthal, NAPP and Pfizer for speaking at

meetings. His institution has received research support in the past five years from Health Technology Assessment (HTA) and Chief Scientist Office (CSO) sponsored studies and commercial sponsored studies (Pfizer, Grünenthal, Lateral Pharma Pty Ltd)

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Useful information

**For patients
Pain Concern**
<https://painconcern.org.uk>

Pain Association
<https://painassociation.co.uk>

The British Pain Society
www.britishpainsociety.org

Show impact

● Collection of data
● Practice audit to show improved care

● Case studies
● Implement a protocol
● Prescribing review
● Educational meetings
● Significant event audit
● Practice meeting
● Implement a new service
● Take on new role
● Significant event audits
● Change with feedback from patients and/or colleagues

Further options for sessional GPs

● Self audit of own practice:
– Record-keeping
– Referrals for investigations
– Prospective condition based reviews
– Random case analysis with colleagues
– Review of telephone triage outcomes
● Update drugs in emergency bag

Time credits

Impact credits
Quick impact

Later impact

Later
impact=

Total credits

Title and reference

Date

What have you learned?

How will this change your practice?

What research/actions will you take that will have further impact on practice?

Draft of your action plan

www.thepractitioner.co.uk

Title

Reflection on Table 1 results

Reason for the audit (target to improve or implement)

Define criterion (or criteria) to be met

What changes were implemented to improve results?

Set the standard and its basis

Reflection on Table 2 results and review with peers

Plan the audit process and any involvement of peers

Time credits

Impact credits

Total credits

Table 1			
Results of data 1 analysis		Date of data collection:	
Criterion	Number sampled	Achievement (%)	Standard (%)
A			
B			
C			

Table 2					
Results of data 1 and data 2 analyses			Date of data collection:		
Criterion	Number sampled Data 1	Achievement (%) Data 1	Number sampled Data 2	Achievement (%) Data 2	Standard (%)
A					
B					
C					