The Practitioner

CPD study pack

December 2018;262(1821)
The study pack contains this month's CPD exercise plus all relevant articles:

- Diagnosis and treatment of Sjögren's syndrome
- Managing low back pain in primary care
- Improving outcomes for kidney transplantation

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Practitioner CPD exercise

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Show impact
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- 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 for 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

SJÖGREN'S SYNDROME

1 Which of the statements are true?

A Sjögren's syndrome often presents in patients in their 60s
True ☐ **False** ☐

B Younger patients tend to present with systemic disease
True ☐ **False** ☐

C Arthralgia may be the dominant clinical feature at presentation
True ☐ **False** ☐

D Older patients are more likely to have classical sicca symptoms
True ☐ **False** ☐

E Azathioprine and hydroxychloroquine should be discontinued during pregnancy
True ☐ **False** ☐

2 Match the statistic to the statement.

A The proportion of patients who do not have severe systemic disease

B The percentage of cases who test positive for rheumatoid factor

C The proportion of patients who are female

D The percentage of patients with Raynaud's phenomenon

1 80%
2 85%
3 90%
4 95%

3 Diagnosis of Sjögren's syndrome should be made by a rheumatologist. What investigations may be carried out in rheumatology to confirm diagnosis?

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

LOW BACK PAIN

5 Complete the statement:

Low back pain is a

condition. It can originate in the vertebrae,

, ligaments,

joints or

muscles.

6 Use of the STarT Back risk assessment tool enables patients with nonspecific low back pain to be graded into two broad categories. What are these categories?

7 Which of the statements are true?

A Herpes zoster may cause low back pain
True ☐ **False** ☐

B Mechanical low back pain usually presents in patients aged over 60
True ☐ **False** ☐

C A positive straight leg raise test (direct and crossed) can indicate nerve root irritation
True ☐ **False** ☐

D Low back pain may be referred to the hips and legs
True ☐ **False** ☐

E Mechanical low back pain varies with activity and time
True ☐ **False** ☐

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

KIDNEY TRANSPLANTATION

9 Which of the statements are true?

A Patients in their 70s are not eligible for a transplant
True ☐ **False** ☐

B All living kidney donors are offered lifelong follow-up
True ☐ **False** ☐

C Transplantation from a living donor is associated with better graft and patient survival than that from a deceased donor
True ☐ **False** ☐

D A living kidney donor does not have to be genetically related to the recipient
True ☐ **False** ☐

10 Complete the statement.
After living kidney donation, the donor will have a substantially higher serum

and substantially lower

The latter does not represent

kidney disease.

The change in renal function simply reflects reduced

11 List three treatment approaches that will improve survival in patients with end-stage renal disease.

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

Answers

SJÖGREN'S SYNDROME
1 A False (40s and 50s) **B** True **C** True **D** True **E** False

2 A 2 **B** 3 **C** 3 **D** 1

3 Scintigraphy/sialography; labial gland biopsy

LOW BACK PAIN
5 Heterogeneous; discs; facet; paravertebral

6 Patients who will respond to simple analgesics and exercise advice; those who are at higher risk of poor recovery, and require more intensive physiotherapy and psychology input

7 A True **B** False (20-55) **C** True **D** True **E** True

KIDNEY TRANSPLANTATION
9 A False **B** True **C** True **D** True

10 Creatinine; eGFR; chronic; nephron mass

11 A transplant compared with dialysis; a living donor as opposed to a deceased donor transplant; transplantation before requiring dialysis

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Diagnosis and treatment of Sjögren's syndrome

Brown GA, Negoescu A. Diagnosis and treatment of Sjögren's syndrome.
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Diagnosis and treatment of Sjögren's syndrome

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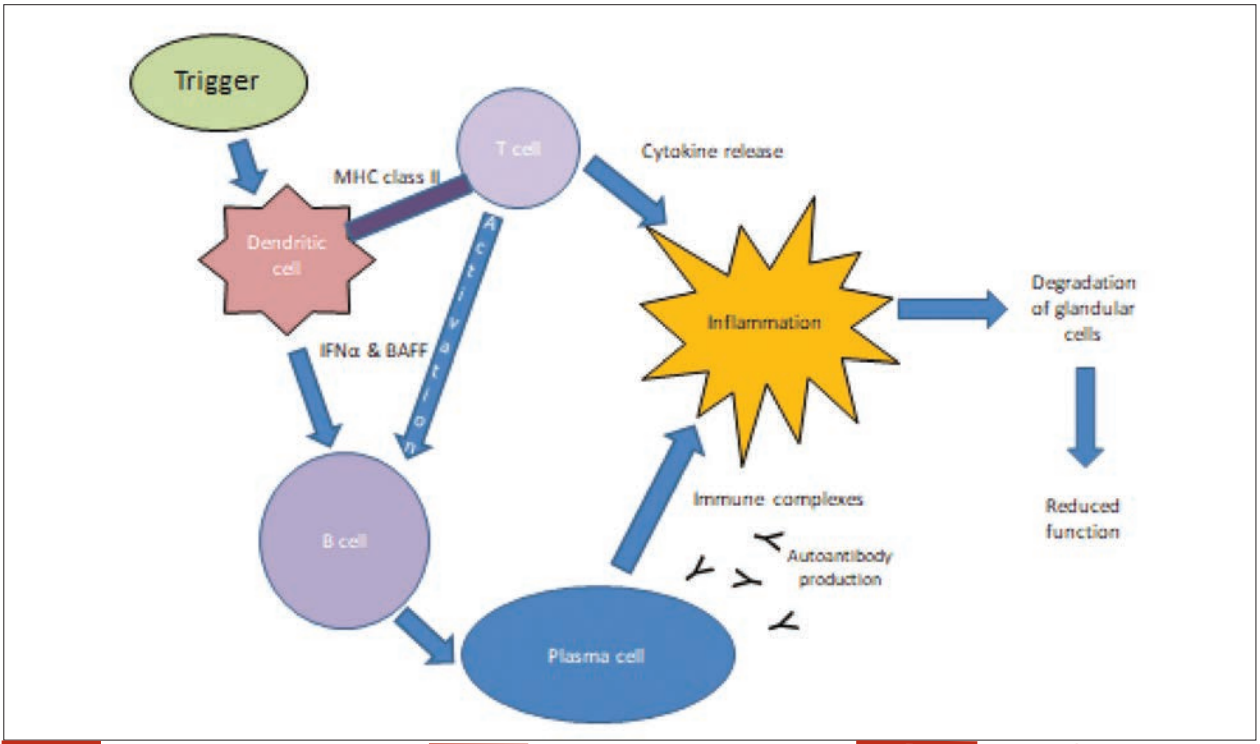

FIGURE 1
Pathophysiology of primary Sjögren's syndrome resulting in exocrine gland destruction

How do patients present in primary care?

PRIMARY SJÖGREN'S SYNDROME IS A CHRONIC SYSTEMIC IMMUNE-MEDIATED condition of unknown aetiology characterised by focal lymphocytic infiltration of exocrine (mainly salivary and lacrimal) glands. It affects 0.1-4.6% of the general population in Europe and 90% of cases are female.[1]

MECHANISMS

The pathophysiology of primary Sjögren's syndrome remains unknown and is likely to be the result of environmental factors triggering inflammation in individuals with a genetic predisposition.

It is accepted that T-cells, B-cells and dendritic cells infiltrate exocrine glands, interfering with their function and causing destruction through cell-mediated and cytokine-activated pathways, leading to chronic inflammation as identified on tissue biopsy, see figure 1, above.

How should diagnosis be confirmed?

PRESENTATION

Sjögren's syndrome often presents in the fifth or sixth decade. However, it can be seen in younger people who tend to present with systemic disease and are less likely to have classical sicca symptoms i.e. dryness of the mouth, eyes or vagina.[2,3]

Although patients generally present with sicca symptoms, other, systemic, symptoms may include arthralgia, fatigue, Raynaud's phenomenon, sensorimotor neuropathy and a dry cough. Peripheral neuropathy can be sensory or sensorimotor, with sensorimotor involvement seen in patients with higher B-cell activity. Cranial neuropathy is rare. The frequency of these symptoms is shown in table 1, left.

Patients may present with arthralgia or a frank inflammatory arthritis as the dominant clinical feature, although on direct questioning many of these patients will report longstanding sicca symptoms.

In patients who develop an inflammatory arthritis, this tends to be »

Table 1

Common symptoms and signs in primary Sjögren's syndrome[4]

Symptoms and signs	Frequency
Raynaud's phenomenon	80%
Fatigue	75%
Dry skin/pruritus	50%
Arthralgia	33-50%
Glandular swelling	25-66%
Dry cough	40%
Sensorimotor neuropathy	< 25%
Purpura rash (hypergammaglobulinaemic)	9%

What are the management approaches?

polyarticular and symmetrical, typically affecting the wrists and small joints of the hands. It is intermittent in nature and usually not deforming or erosive.

Practical tip 1: *Consider primary Sjögren's syndrome as a differential to rheumatoid arthritis in patients presenting with symmetrical inflammatory arthralgia/arthritis affecting the wrists and small joints of the hands.*

Sjögren's syndrome mimics

While Sjögren's syndrome is an important condition to consider in patients presenting with sicca symptoms, a variety of other conditions, drugs and dehydration may contribute to, or cause, mucosal dryness.

Other causes of sicca symptoms include: head and neck radiotherapy (salivary flow can be reduced after the first dose of radiotherapy and this may persist for up to three months post completion), sarcoidosis, acute anxiety, IgG4 disease, hepatitis C, HIV. The latter two are also associated with salivary gland hypertrophy. Chronic ocular conditions (such as blepharitis and conjunctivitis) are a common cause of dry eyes.

The most common drugs which may induce sicca symptoms are highlighted in table 2, below.

Practical tip 2: *Commonly prescribed drugs such as opioids, antidepressants, beta-blockers, proton pump inhibitors, antihistamines and muscarinic antagonists may induce sicca symptoms.*

ASSESSMENT

Because of its complex presentation and heterogeneity, diagnosis may be delayed by 3-11 years.[3]

The presence of any four of the six criteria shown in table 3, p13, is indicative of primary Sjögren's syndrome, as long as either item IV (histopathology) or item VI (serology) is positive. The presence of any three of the four objective criteria (items III, IV, V, VI) is also indicative of the condition.

The following need to be excluded before the classification criteria can be applied:

- Post head and neck radiotherapy
- Hepatitis C infection
- HIV
- Pre-existing lymphoma
- Sarcoidosis
- Graft versus host disease
- Use of anticholinergic drugs (within a time shorter than four times the half-life of the drug)

In patients with a potentially associated disease, for instance, an established diagnosis of rheumatoid arthritis (RA) or another well defined connective tissue disease such as systemic lupus erythematosus (SLE), the presence of item I or item II plus any two from among items III, IV, and V may be considered indicative of secondary Sjögren's syndrome.

Examination findings during a GP consultation may include the following:

- **Eyes:** Dilatation of the conjunctival vessels, corneal lesions, and blepharitis
- **Mouth:** May look dry and a wooden tongue depressor may stick to the tongue. Local infections including oral candidiasis and dental caries are not uncommon
- **Submandibular glands:** May be enlarged but more obvious is bilateral enlargement of the parotid glands. Because of the intermittent nature of glandular swelling, this may be absent at the time of examination
- **Features of other autoimmune disorders** such as RA, SLE, scleroderma and primary biliary cirrhosis

Patients with suspected primary Sjögren's syndrome should be tested for antinuclear antigen (ANA) to look for the presence of anti-Ro/La antibodies which are present in two-thirds of cases. Rheumatoid factor may be present in up to 90% of cases. High levels of IgG are commonly seen in patients with primary Sjögren's syndrome.

Practical tip 3: *Testing for ANA should be performed in all patients with suspected inflammatory arthritis, alongside rheumatoid factor.*

REFERRAL

Referral to a rheumatologist for confirmation of diagnosis (which may involve scintigraphy/sialography, labial gland biopsy) is recommended.

Referral should ultimately be guided by the level of clinical suspicion. Specialist referral should be considered in patients with sicca symptoms who test positive for ANA. However, if a patient is ANA negative but has sicca symptoms, any of the signs and symptoms listed in table 1, p11, and/or suggestive examination findings, then we would also advise referral to a rheumatologist.

Long-term follow-up by a specialist is also required. The general consensus is that patients should be seen every six months by a rheumatologist for those with stable disease, and specific disease activity scores should be incorporated as standard practice.

TREATMENT

Current treatment strategies include a combination of symptom control and immunosuppression, and multidisciplinary involvement. Management of sicca symptoms includes lifestyle changes in combination with topical lubricants.

Patients with dry eyes should be advised not to wear contact lenses, avoid air conditioning where possible and use simple lubricant drops that do not contain preservatives. Liposomal sprays can reduce evaporative tear loss, and lacrimal gland inflammation can be managed by applying damp steamed eye pads. Oral pilocarpine can be

Table 2

Common drugs known to induce sicca symptoms

Class	Drug
Anticholinergic	Antidepressants e.g. amitriptyline Anxiolytics e.g. diazepam Antipsychotics e.g. clozapine
Muscarinic antagonists	Tamsulosin hydrochloride Ipratropium hydrochloride
Antihistamines	Cetirizine hydrochloride, loratadine
Opioids	Morphine, codeine, tramadol
Antihypertensives	Beta-blockers ACE inhibitors
Proton pump inhibitors	Omeprazole

Table 3

Classification criteria for Sjögren's syndrome (sensitivity 97.4%, specificity 89.4%)[5]

Criteria	Evidence	Assessment
I Ocular symptoms	At least one of: **a** Persistent troublesome dry eyes for > 3 months **b** Recurrent sensation of sand or gravel in the eyes **c** Using tear substitutes > 3 times a day	Suitable for assessment in primary care
II Oral symptoms	At least one of: **a** Daily feeling of dry mouth for > 3 months **b** Recurrent or persistent swelling of salivary glands as an adult **c** Frequently drinking liquids to aid in swallowing dry food	Suitable for assessment in primary care
III Ocular signs	Positive Schirmer test or high Rose Bengal score	Performed in secondary care or by community optometrist
IV Histopathology	Focal lymphocytic sialadenitis in minor salivary glands	Salivary gland biopsy in secondary care required
V Objective evidence of salivary gland involvement	Abnormal salivary flow, parotid sialography, salivary scintigraphy	Performed in secondary care
VI Autoantibodies	Antibodies to Ro/SSA or La/SSB antigens, or both	Suitable for assessment in primary care by ordering generic ANA

considered in those with significant symptoms. Ophthalmology input is advisable in severe disease and in patients with inflammatory eye disease, who may require steroid-containing eye drops or ciclosporin.

Dryness of the oral cavity can be managed by reducing sugar and acid intake using pastilles and salivary spray replacement. Dental health is important and patients should see a dentist on a regular basis as they have a higher risk of dental cavities. Toothpaste with a high fluoride content is recommended in addition to intermittent chlorhexidine mouthwash, and regular oral hygiene.

Salivary gland enlargement may occur during acute flares or through duct obstruction from stones or strictures. A short course of oral or intramuscular glucocorticoids often helps and there is anecdotal evidence that local massage may help reduce the swelling.

Management of fatigue as recommended by the NICE guideline includes good sleep hygiene, relaxation techniques, graded exercise programmes and cognitive behaviour therapy.[6]

Systemic features are seen in around 70% of patients[2] and commonly affect joints, lung, skin and peripheral nerves.

Glucocorticoids may be used in systemic flares, or at a low dose to improve sicca symptoms and salivary flow. Hydroxychloroquine may improve joint pain, fatigue, Raynaud's and sicca symptoms, while methotrexate is used in patients with predominant joint involvement (inflammatory arthritis). Stronger disease modifying agents such as azathioprine, methotrexate, cyclophosphamide or mycofenolate mofetil are reserved for serious complications and organ-threatening disease (cytopaenias, lung disease, and neuropathy).

Clinical trials with biologics in Sjögren's syndrome have been disappointing to date, when compared with other rheumatic conditions such as RA.

POTENTIAL COMPLICATIONS

Severe systemic disease occurs in about 15% of patients. This may include interstitial lung disease, myositis, idiopathic thrombocytopaenic purpura, autoimmune liver disease, and renal involvement.[7]

Other autoimmune diseases may be present in association with primary Sjögren's syndrome, including thyroid disease, primary biliary cirrhosis and coeliac disease.

B-cell lymphoma is a serious complication and affects 5-10% of patients with primary Sjögren's syndrome.[8] The risk of developing lymphoma has previously been shown to be 7 to 19 times higher compared with the general population.[9] Predictors for development of lymphoma include: persistent salivary gland enlargement, lymphadenopathy, Raynaud's phenomenon, anti-Ro/La positivity, rheumatoid factor positivity, and low serum C4 levels.[10]

Practical tip 4: *Have a low threshold for assessing and investigating primary Sjögren's syndrome patients presenting with constitutional symptoms.*

Pregnancy poses many challenges and is associated with a higher complication rate in primary Sjögren's syndrome than in the general population.

Obstetric complications include preterm delivery, lower birthweight, and pulmonary hypertension. Anti-Ro antibodies in particular may cross the placenta and induce congenital heart block (occurring in < 2% of pregnancies) and neonatal lupus. The former can be detected by fetal ultrasound, and often »

key points

SELECTED BY

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Primary Sjögren's syndrome is a chronic systemic immune-mediated condition of unknown aetiology characterised by focal lymphocytic infiltration of exocrine (mainly salivary and lacrimal) glands. It affects 0.1-4.6% of the European population and 90% of cases are female. Although it usually presents in the fifth or sixth decade, it can be seen in younger people who tend to present with systemic disease and are less likely to have classical sicca symptoms.

Although patients generally present with sicca symptoms i.e. dryness of the mouth, eyes or vagina, other symptoms may include arthralgia, fatigue, Raynaud's phenomenon, sensorimotor neuropathy or a dry cough. In patients who develop an inflammatory arthritis, this tends to be polyarticular and symmetrical, typically affecting the wrists and small joints of the hands.

While Sjögren's syndrome is an important condition to consider in people presenting with sicca symptoms, a variety of other conditions may contribute to, or cause, mucosal dryness. These include head and neck radiotherapy, sarcoidosis, acute anxiety, IgG4 disease, hepatitis C and HIV. Drugs which may induce sicca symptoms include: opioids, antidepressants, beta-blockers, proton pump inhibitors, antihistamines and muscarinic antagonists. Chronic ocular conditions such as blepharitis and conjunctivitis are a common cause of dry eyes.

On examination the eyes may show dilatation of the conjunctival vessels, corneal lesions, and blepharitis. The mouth may look dry and a wooden tongue depressor may stick to the tongue. Patients are more prone to oral candidiasis and dental caries. There may be bilateral submandibular or, more obviously, parotid gland enlargement. There may be features of other autoimmune disorders such as rheumatoid arthritis, SLE, scleroderma and primary biliary cirrhosis. Patients with suspected primary Sjögren's syndrome should be tested for antinuclear antigen to look for the presence of anti-Ro/La antibodies.

Patients should be referred to a rheumatologist to confirm diagnosis, and this may involve scintigraphy/sialography and/or labial gland biopsy. Treatment comprises symptom control and immunosuppression, and multidisciplinary involvement. Glucocorticoids may be used in systemic flares, or at a low dose to improve sicca symptoms and salivary flow. Hydroxychloroquine may improve joint pain, fatigue, Raynaud's and sicca symptoms, while methotrexate is used in patients with inflammatory arthritis.

Severe systemic disease occurs in about 15% of cases. This may include interstitial lung disease, myositis, idiopathic thrombocytopaenic purpura, autoimmune liver disease, and renal involvement. B-cell lymphoma is a serious complication which affects 5-10% of patients. Pregnancy is associated with an increased risk of complications including preterm delivery, low birthweight, and pulmonary hypertension. Anti-Ro antibodies may cross the placenta and induce congenital heart block and neonatal lupus. anti-Ro positive pregnant women benefit from a joint rheumatology-obstetrics clinic or a multidisciplinary approach.

Neonatal lupus manifests as a transient rash, usually developing during the first few weeks of life and resolving after several weeks to months.

Practical tip 5: *Women of reproductive age who are anti-Ro positive must be counselled about the risk of obstetric complications prior to conception, whenever possible.*

Flares of primary Sjögren's syndrome may also occur during pregnancy. It is advisable that patients have good disease control prior to conception, and that the background medication is continued throughout the pregnancy, if compatible, to minimise the risk of flare. Hydroxychloroquine and azathioprine are both compatible with pregnancy. There is also evidence that low-dose aspirin initiated in early pregnancy can improve placental implantation and reduce pre-eclampsia and intrauterine growth retardation.[2]

Practical tip 6: *It is important that women are encouraged to continue taking their background medication during pregnancy (hydroxychloroquine and/or azathioprine), to minimise the risk of flare.*

Anxiety and depression may occur in a third of patients with Sjögren's syndrome. Management should involve a combination of identifying and managing triggers, social support, psychological intervention (cognitive behavioural therapy) and pharmacological therapy. Amitriptyline, propranolol and selective serotonin reuptake inhibitors can all exacerbate sicca symptoms, thus their use should be considered on a case by case basis.

CONCLUSION

Primary Sjögren's syndrome is a chronic complex systemic disease, which can sometimes lead to significant morbidity and mortality. Prompt recognition and management leads to better outcomes for patients, and vigilance with respect to potential serious complications may reduce morbidity and mortality for these patients.

Competing interests: None

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Useful information

The British Society for Rheumatology guideline for the management of adults with primary Sjögren's syndrome
https://academic.oup.com/rheumatology/article/56/10/e24/3895127

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Managing low back pain in primary care

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Managing low back pain in primary care

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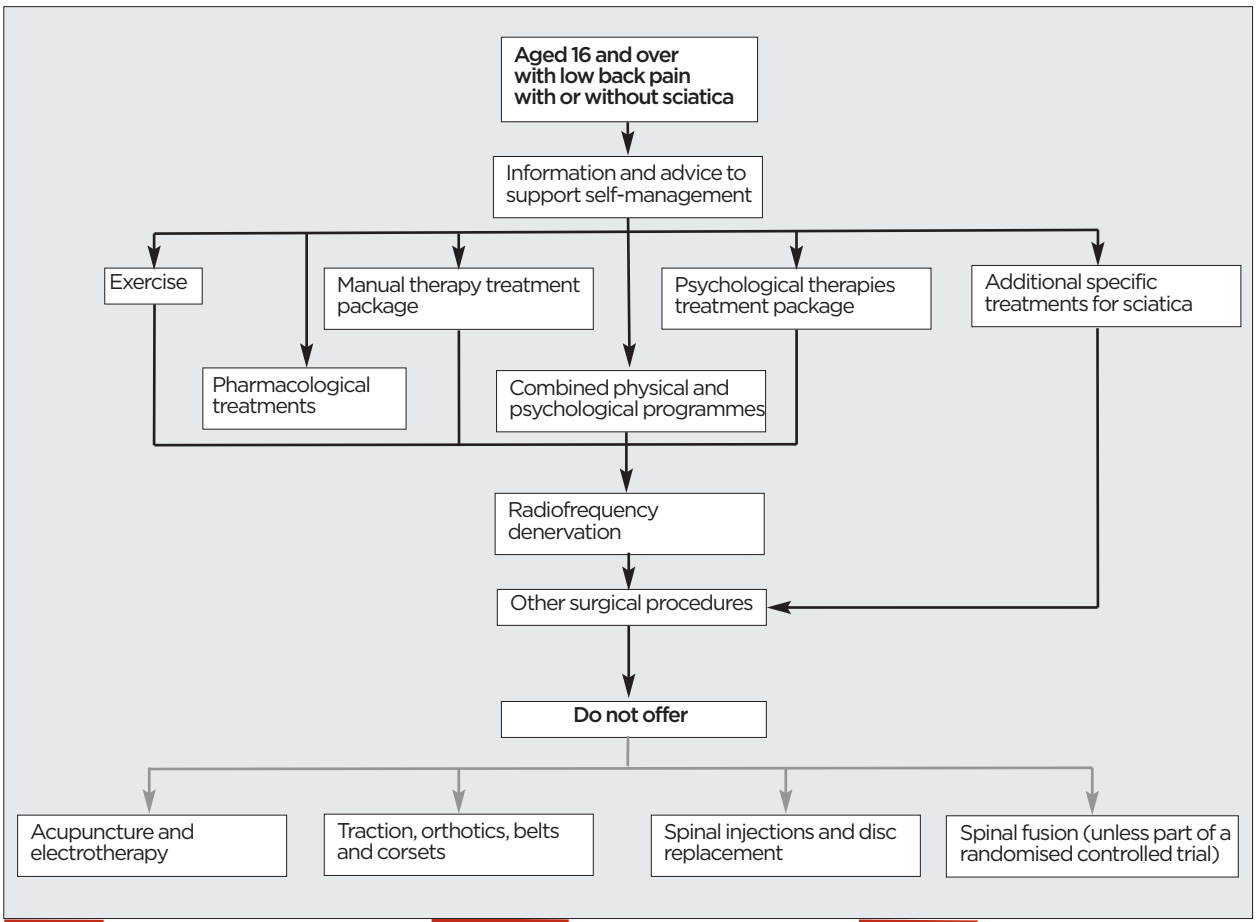

FIGURE 1
Managing low back pain and sciatica (adapted from the NICE pathway[7])

How should patients be assessed?

NONSPECIFIC LOW BACK PAIN IS DEFINED AS TENSION, SORENESS AND/OR STIFFNESS IN THE BACK below the ribs and above the inferior gluteal skin crease, for which it is not possible to identify a specific cause.[1]

Low back pain may be referred out to the hips and legs. This referred leg pain should not be confused with neuropathic radicular pain (sciatica). The latter has a neuropathic quality (sharp, burning, tingling etc) and usually radiates distal to the knee and can be accompanied by sensory, motor and reflex deficits. Sciatica should be managed as neuropathic pain.[2]

Chronic low back pain is a common problem. Lifetime adult prevalence rates

Which patients should be referred?

vary from 50% to 80%, and point prevalence rates from 15% to 30%.[3] Around a quarter of adults say they have experienced back pain in the past month, with one in 40 reporting disabling back or neck pain.[1] Each year around 7% of patients consult their GP with back pain.

A GP with a list of 1,750 patients will typically see at least one patient with low back pain every day.[3] In 80-90% of cases of low back pain presenting in primary care the pain is thought to be mechanical in origin. Low back pain is a heterogeneous condition and can originate in the vertebrae, discs, ligaments, facet joints, or paravertebral muscles. In 65-70% of primary care patients with low back pain there is no

What are the management strategies?

known pathoanatomical cause. Their pain is described as nonspecific and is postulated to arise from muscle strain or ligamentous injury. A further 15-20% of patients have mechanical low back pain with an identifiable cause such as degenerative disc or joint disease. Table 1, p16, lists the common causes of low back pain.

ASSESSMENT

GPs should obtain a focused history and carry out a physical examination.

Mechanical back pain usually presents in patients aged between 20 and 55. The pain is in the lumbosacral region, buttocks and thighs. It is described in nociceptive terms such as dull, aching, »

pressure, tight etc rather than the neuropathic terms used to describe sciatica. The pain varies with activity and time. The patient normally feels well otherwise. Patients presenting with low back pain secondary to underlying serious pathology (< 1%) may have a history of significant traumatic injury, fever, chills, malaise, night sweats, unexplained weight loss, cancer, intravenous drug use, immunosuppression, organ transplantation or corticosteroid use.

A comprehensive general physical examination and focused neurological examination should be carried out. This should include assessment of gait, range of spinal motion and degree if any at which the patient feels pain. Palpate for local spinal and paraspinal tenderness, muscles can be tense and tender, but it is unusual for bone to be tender.

Check for passive and active movement of the lower limbs to assess for normal range of motion and muscle strength. A positive straight leg raise test (direct and crossed) can indicate nerve root irritation. Motor and sensory nerve deficits, and reflex deficits (knee L3/4, ankle L5/S1) should be excluded. In particular, it is important to exclude perineal numbness, or bowel and bladder disturbance, which might indicate cauda equina syndrome.

'It is important to ask about, and examine for, red flags to exclude severe pathology'

Severity of baseline pain (0 to 10 on the visual analogue scale); functional deficits in physical activity; sleep impairment; and any disturbance in psychological function such as depression or anxiety should be assessed. Several questionnaires are available, such as the Brief Pain Inventory (four questions on pain severity, seven questions on impairment of daily activities), or the Hospital Anxiety and Depression Scale (HADS).

Clinicians should not routinely obtain imaging or other diagnostic tests in patients with nonspecific low back pain.[4,5] It is important to ask about, and examine for, red flags to exclude severe pathology (see below).

Yellow flags are psychosocial factors that can predict when a patient may have a poor prognosis for resolution and return to normal function. The use of a risk stratification tool such as STarT Back (see box 1, below) at first point of contact can help identify those patients who require more intensive physiotherapy and a psychology based approach.[6] STarT Back is a prognostic questionnaire that assesses the risk of continuing pain and disability.

The assessment will enable patients to be graded into two broad categories.[5]
1 Patients with nonspecific low back pain who will respond to simple analgesics, and exercise advice
2 Patients with nonspecific low back pain who are at higher risk of poor recovery, and require more intensive physiotherapy and psychology input

RED FLAGS

If there are concerns that the patient may have a specific cause for their back pain then appropriate referral or investigation is indicated.

Table 1

Common causes of low back pain[3]

Mechanical (80-90%)
- Unknown cause - usually attributed to muscle strain or ligamentous injury (65-70%)
- Degenerative disc or joint disease
- Vertebral fracture
- Congenital deformity (such as scoliosis, kyphosis, transitional vertebrae)
- Spondylolysis
- Instability

Neurogenic (5-15%)
- Herniated disc
- Spinal stenosis
- Osteophytic nerve root composition
- Annular fissure with chemical irritation of nerve root
- Failed back surgery syndrome (such as arachnoiditis, epidural adhesions, recurrent herniation); may cause mechanical back pain as well
- Infection (such as herpes zoster)

Non-mechanical spinal conditions (1-2%)
- Neoplastic (such as primary or metastatic) disease
- Infection (such as osteomyelitis, discitis, abscess)
- Inflammatory arthritis (such as rheumatoid arthritis and spondyloarthropathies, including ankylosing spondylitis, reactive arthritis, enteropathic arthritis)
- Paget's disease
- Other (such as Scheuermann's disease, Baastrup's disease)

Referred visceral pain (1-2%)
- Gastrointestinal disease (such as inflammatory bowel disease, pancreatitis, diverticulitis)
- Renal disease (such as nephrolithiasis, pyelonephritis)
- Abdominal aortic aneurysm

Other (2-4%)
- Fibromyalgia
- Somatoform disorder (such as somatisation disorder, pain disorder)
- Malingering

Box 1

STarT Back screening tool

For questions 1-8, score 1 for agreement, 0 for disagreement
1 My back pain has spread down my leg(s) at some time in the past two weeks

2 I have had pain in the shoulder or neck at some time in the past two weeks

3 I have only walked short distances because of my back pain

4 In the past two weeks, I have dressed more slowly than usual because of back pain

5 It is not really safe for someone with a condition like mine to be physically active

6 Worrying thoughts have been going through my mind a lot of the time

7 I feel that my back pain is terrible and it is never going to get any better

8 In general I have not enjoyed all the things I used to enjoy

9 Overall, how bothersome has your back pain been in the past two weeks?
Not at all (0), slightly (0), moderately (0), very much (1), extremely (1)

STarT Back scoring: Low risk = total score 0-3; high risk = score 4-5 of questions 5-9 only; the rest are medium risk

key points

SELECTED BY

Dr Phillip Bland
Former GP, Dalton-in-Furness, UK

Chronic low back pain is a common problem. Lifetime adult prevalence rates vary from 50% to 80%, and around a quarter of adults say they have experienced back pain during the past month. One in 40 report disabling back or neck pain. Each year around 7% of patients consult their GP with back pain.

In 65-70% of primary care patients with low back pain there is no known pathoanatomical cause. Their pain is described as nonspecific and is postulated to arise from muscle strain or ligamentous injury. A further 15-20% of patients have mechanical low back pain with an identifiable cause such as degenerative disc or joint disease. The pain may be referred to the hips and legs but this should not be confused with neuropathic radicular pain (sciatica), which has a neuropathic quality, usually radiates distal to the knee and can be accompanied by sensory, motor and reflex deficits.

Clinicians should not routinely obtain imaging or other diagnostic tests in patients with nonspecific low back pain. However, if there are concerns that the patient may have a specific cause for their back pain then appropriate referral or investigation is indicated. Admission or urgent referral to orthopaedics or neurosurgery is required if neural deficit is suspected or severe systemic illness is apparent. Referral to rheumatology or oncology may be appropriate for patients with suspected malignancy, infection, fracture, or an inflammatory disorder such as ankylosing spondylitis.

Providing there is no suspicion of serious underlying pathology NICE recommends that GPs should provide patients with information on low back pain with regard to its expected course, the need to remain active, and effective self-management options. Patients should be encouraged to do exercises such as stretching, strengthening, aerobics, yoga, or tai chi in a group setting. Use of the STarT Back risk assessment tool is recommended to assess the risk of continuing pain and disability.

A combined physical and psychological programme incorporating cognitive behavioural therapy should be considered: in patients with a high risk of poor outcome (as assessed by STarT Back); if after initial treatment recommendations have been tried pain is still significant after three months, or sooner if pain is severe and accompanied by distress or physical impairment. NICE suggests this should preferably be in a group context that takes into account the individual's specific needs and capabilities.

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Urgent referral to orthopaedics or neurosurgery is required if neural deficit is suspected, or severe systemic illness is apparent, when rheumatology or oncology may be appropriate. This includes suspected malignancy, infection, fracture, or an inflammatory disorder such as ankylosing spondylitis (prevalence 0.15%).

Red flags include the following:

- Weight loss
- Past medical history of malignancy
- Fever
- Signs of systemic inflammatory disease
- Anatomical change
- Possible fracture
- History of trauma
- Cauda equina syndrome
- Neurological signs (radiculopathy)

MANAGEMENT

In 2016, NICE published an evidence-based clinical guideline (NG59) on managing low back pain and sciatica.[4]

Providing there is no suspicion of serious underlying pathology NICE recommends that GPs should provide patients with information on low back pain with regard to its expected course, the need to remain active, and effective self-management options.

Patients should be encouraged to do exercises such as stretching, strengthening, aerobics, yoga, or tai chi in a group setting as the main component of non-invasive treatment.

'Manual therapy and psychological approaches should be recommended only alongside an exercise programme'

Manual therapy and psychological approaches should be recommended only alongside an exercise programme.[4] NICE does not recommend acupuncture or electrotherapies such as transcutaneous electrical nerve stimulation (TENS), see figure 1, p15.[7] Acupuncture has not been shown to produce any treatment specific benefits when compared with sham acupuncture. There is little evidence that TENS confers any additional benefits.

When pain is moderate/severe, then the above approaches should be combined with analgesics such as a short course of NSAIDs, or a weak opioid where an NSAID is ineffective or poorly tolerated. Patients should not be offered paracetamol alone.

Major depression is about four times more common in people with chronic back pain than in the general population.[8] Rate of major depression increases in a linear fashion with greater pain severity.[9] There is evidence suggesting that treatment of low back pain can improve depressive symptoms and vice versa.[10,11]

'There is evidence suggesting that treatment of low back pain can improve depressive symptoms and vice versa'

It is recommended that low back pain and depression be treated as separate and independent entities.[12] Initially, an empirical trial of antidepressants is recommended for at least four weeks. If there is less than a 20% reduction in depressive symptoms, a switch to another antidepressant or the addition of an augmentation agent would be recommended. If there is a 20% or greater reduction of symptoms, the antidepressant should be continued for an additional 4 to 6 weeks until the expected maximal benefits are achieved between 12 and 16 weeks.[12]

REFERRAL

A combined physical and psychological programme incorporating cognitive behavioural therapy should be considered: in patients with a high risk of poor outcome (as assessed by STarT Back); if after initial treatment recommendations have been tried pain is still significant after three months, or sooner if pain is severe and accompanied by distress or physical impairment. NICE suggests this should preferably be in a group context that takes into account the individual's specific needs and capabilities.

Patients referred to a chronic pain clinic will undergo a comprehensive holistic assessment. Their medication regimen will be reviewed and adjusted, if needed, and psychotherapy-based treatments with either individual psychotherapy or group-based pain management programmes may be offered. The pain clinic can also provide radiofrequency medial branch nerve »

denervation for suitable patients with severe localised facet joint low back pain.

Around 20% of people are still symptomatic and 3% remain off work a year after an episode of low back pain.[4] NICE encourages clinicians to consider the risks and benefits of pursuing investigations and continuing treatments where these are of limited benefit. Instead, patients could return to primary care management, with pain clinic support where needed.

Competing interests: Dr Mick Serpell has received honoraria from Astellas, Grünenthal, NAPP and Pfizer for speaking at meetings. Dr Sunny Bhatt has no competing interests

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Useful information

For healthcare professionals
National Back Pain and Radicular Pain Pathway
www.noebackpainprogramme.nhs.uk/wp-content/uploads/2015/05/National-Low-Back-and-Radicular-Pain-Pathway-2017_final.pdf

For patients
The Pain Tool Kit
www.paintoolkit.org

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Improving outcomes for kidney transplantation

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Improving outcomes for kidney transplantation

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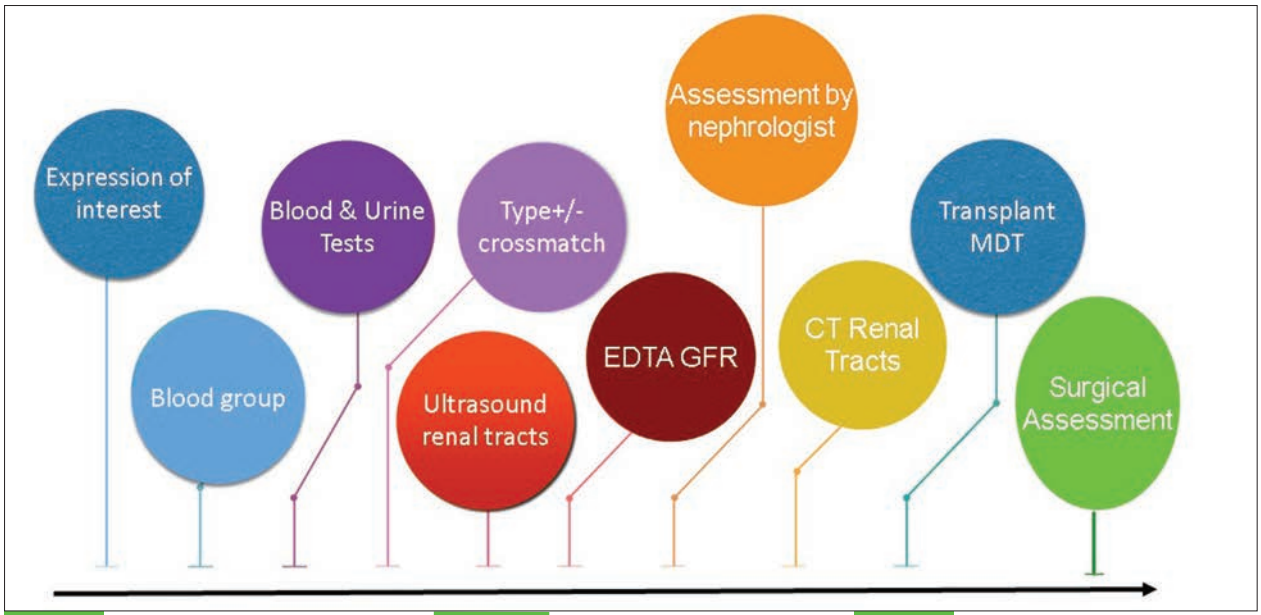

FIGURE 1
Assessment of potential living kidney donors

How should potential donors and recipients be assessed?

KIDNEY TRANSPLANTATION IS THE OPTIMUM FORM OF RENAL REPLACEMENT THERAPY FOR SUITABLE patients with end-stage renal disease (ESRD).[1,2] The benefits compared with maintenance dialysis therapy, in terms of patient survival and quality of life, are universally accepted.

Despite advances in haemodialysis over the past decades, with better molecular clearance, shorter hours, less stringent dietary restriction and the use of recombinant erythropoietin, it remains non-physiological and suboptimal compared with normal renal function.

A successful kidney transplant represents a much closer approximation to true renal replacement with excretion of waste products, regulation of electrolytes and other substances, and synthesis of important hormones; it therefore offers substantial benefits compared with dialysis, see box 1, right. Although it cannot be considered curative, (with the possible exception of transplantation between identical twins), it offers hope of a life expectancy that can approach normal.

What are the risks and possible complications?

SUITABILITY FOR TRANSPLANTATION

Not all patients with ESRD are suitable for transplantation. Assessment centres on the suitability of the patient for:

- General anaesthesia and surgery
- Renal transplant surgery
- Long-term immunosuppression

Table 1, p20, summarises the most common areas that require consideration. The demographic of chronic dialysis patients has changed dramatically in recent decades with increasing age, comorbidity, and prevalence of diabetes. Those with diabetic nephropathy inevitably have a constellation of other complications.

Age per se is not a contraindication to transplantation. There are some patients in good health in their 70s for whom this offers an important advantage in terms of quality of life. In contrast, there are patients with lifelong illness who are unsuitable for transplantation despite being decades younger.

The recent NICE guideline on renal replacement therapy[3] recommends that patients should not be precluded from consideration for transplantation based on BMI alone. However, there are increasing challenges and risks of complications in obese individuals, and few centres will consider transplanting the morbidly obese. In regions without bariatric services, the options for these patients to achieve an 'acceptable' BMI are limited, and some transplant units will accept such patients for transplantation, considering obesity as part of an overall risk assessment on a case by case basis.

If a patient is deemed suitable for a general anaesthetic and surgical procedure, then specific consideration to the requirements for renal transplant »

Box 1

Survival of patients with end-stage renal disease

Patients live longer if they have a:

- Transplant compared with dialysis
- Living donor compared with a deceased donor transplant
- Transplant before requiring dialysis (pre-emptive)

How should donors and recipients be followed up?

Table 1

Assessing suitability for renal transplantation

General anaesthesia/ intermediate surgery	**Comorbidity**		**Frailty**		**Obesity**	
	Cardiovascular disease is common in patients with ESRD		Age per se is not a contraindication Some young/middle-aged patients have had lifelong disease and are unsuitable		Some transplant centres have an absolute cut-off BMI, others do not	
Transplant surgery	**Vascular**		**Urological**		**Space**	
	Vascular calcification	Vascular stenosis	Adequate bladder or alternative drainage		≥ 2 previous transplants	Polycystic kidneys
Immunosuppression	**Malignancy**		**Infection**		**Adherence**	
	Previous malignancy with potential obligatory delay		Chronic infection	Recurrent infections	Psychiatric illness	Previous non adherence

surgery must be considered. Many patients with ESRD, particularly if they have been on dialysis for a long time, have extensive vascular calcification. It may be impossible to clamp the vessels successfully and achieve vascular anastomoses.

In patients with previous transplants or multiple femoral venous catheters for central access, there may be previous vascular disruption limiting the options for implantation of a transplant.

An important minority of patients have renal failure because of urological problems such as obstruction or abnormal bladder function. In such cases it is important to ensure there can be appropriate drainage from a transplanted kidney, or inevitably it will have the same fate as the native kidneys.

For patients who have had two or more transplants already or have very large polycystic kidneys that extend into the pelvis, consideration has to be given to nephrectomy to allow space for transplantation.

Finally, the risks of immunosuppression are evaluated. Antirejection drugs reduce the body's normal immunosurveillance function thereby increasing the risk of malignancy. For those patients with treated malignancy the risk of (potentially aggressive) recurrent disease must be considered. International guidelines advise on obligatory waiting periods following treatment for different types of cancer before solid organ transplantation.

Those with chronic infection e.g. osteomyelitis in dialysis patients with concurrent vascular disease, are also precluded from transplantation until they are infection free. Patients with recurrent infections must have careful consideration of the risk-benefit ratio with transplantation, and all steps to minimise the risk of sepsis taken.

Those who have been non adherent with immunosuppression previously, particularly if it has resulted in the loss of a transplanted organ, must be very carefully assessed before a further transplant can be considered.

The only additional consideration is the risk of recurrent disease in the new kidney. There are a tiny number of cases where the original (typically glomerular) disease returns so rapidly and aggressively that a transplant is not a feasible option.

LIVING VS DECEASED DONATION OUTCOMES

Transplantation from a living donor is associated with better graft and patient survival than that from a deceased donor.[4,5] This persists despite adjustment in analysis for the other factors known to influence outcomes, and is consistently reported in different countries and healthcare settings. In the latest reported outcomes in the UK, five-year patient survival rates following deceased and live kidney donation were 87% and 94% respectively.[6]

'Patients live longer if they have a living donor compared with a deceased donor transplant'

The reasons for the better outcomes associated with living donor transplantation can be categorised into donor and recipient factors.

Donor factors

Injury to a deceased donor kidney can be sustained in the premortal period, at time of death, and in the post-donation stage.

The demography of donors has changed substantially from the early years of transplantation, when the typical donor was young and male. Road safety and drink-driving campaigns have contributed to a great reduction in this cohort of donors, (in the UK in 2017-18 only 3% of deceased donors died because of trauma[7] compared with 25% in 1999,[8] and the average age of donors has risen substantially. In 1992 only 5% of donors were over 65 years old,[8] while 33% in 2017/18 were over 60 years old.[7]

Donors often have preexisting comorbidities such as hypertension or diabetes. Many kidneys now available for transplantation from deceased donors have established chronic damage.

A physiological calamity has occurred in all deceased donors, so inevitably there is a degree of insult to renal function at the time of death. The extent of this is impossible to establish definitively, a substantial proportion of kidneys that are offered for donation are declined as the clinical impression is that there may be irreversible damage. Despite this careful consideration some transplanted kidneys never work.

In comparison to living donor transplants, deceased donor organs have a prolonged period of ischaemia given the obvious inability to organise the transplant in advance. This adds further to the injury to a deceased donor kidney.

Recipient factors

The longer a recipient has been dialysis dependent before transplantation the poorer the outcome. Length of time on dialysis is the strongest independent modifiable risk factor for renal transplant outcome. Having an available living donor provides the opportunity for a pre-emptive transplant (i.e. avoidance of dialysis altogether). This offers a distinct

advantage, acknowledged in the NICE guideline, especially for patients with diabetic nephropathy (who typically have limited survival on dialysis), multiple comorbid potential recipients, and older individuals all of whom will particularly benefit from minimisation of time on dialysis. In the UK, 40% of living donor transplants in 2017-18 were pre-emptive, compared with 16% of transplants from deceased donation.[6]

'The longer a recipient has been dialysis dependent before transplantation the poorer the outcome'

A small but important exception to the premise of a healthier recipient cohort in living donor transplantation is the group with such complexity that they are considered unsuitable for an emergency surgical procedure (i.e. a deceased donor transplant). With a carefully planned elective transplant from a living donor, they can potentially benefit from transplantation, without this option the risks are prohibitive. Careful counselling of both the potential donor and recipient is imperative.

RISKS AND COMPLICATIONS OF LIVING KIDNEY DONATION

The risks of donating and living with one kidney are categorised according to the temporal association with donation.

Short-term risks

These are complications related to:

- **General anaesthesia**
- **Any surgical procedure** i.e. pain, nausea, infection, thrombosis, nerve injury
- **Nephrectomy** i.e. vascular, bowel, splenic, or thoracic injury

Almost all centres now offer minimally invasive laparoscopic surgery for donors. The risk of a major complication, including conversion to open surgery is 1-2%.[9] The risk of death is typically quoted as 1 in 3,000, based on data on more than 6,000 living donors in the USA.[10]

Medium-term risks

Following the initial recovery period after nephrectomy, few living donors have any persistent problems. For the minority that do, one of the most common for men who have had a left kidney removed is testicular discomfort. The venous drainage of the left testis is conventionally into the left renal vein, the disruption to this can result in venous engorgement, manifest as swelling and tenderness of the testis. This typically settles over a few weeks or months as collateral drainage develops, with supportive treatment only required.

Persistent pain and symptoms of irritable bowel can also occur in a small minority of donors, and as with any surgery, an incisional hernia may complicate recovery. While not sinister, the added morbidity of these medium-term complications is unwelcome for previously healthy donors.

Long-term risks

Hypertension, renal failure, and premature death are potential long-term risks. The challenge in drawing conclusions about the magnitude of any additional risk is the observational nature of many reports, and the validity of comparison with the general population is limited by the inclusion in the latter group of people who, by virtue of comorbidities or lifestyle, would not have been considered suitable donors.

More recently several large studies have made efforts to recruit healthy non donors as controls. This year a systematic review and meta-analysis, which included approximately 118,000 donors and a similar number of non donors has concluded that, despite higher diastolic blood pressure (BP) readings, lower eGFR, increased relative risk for ESRD, and pre-eclampsia, the absolute risks for such adverse outcomes in a living donor is low.[11]

The distinction between relative and absolute risk is important; if an individual's lifetime risk of renal failure is 0.05%, then even a ten-fold increase in relative risk means that the absolute risk of ESRD is still only 0.5%. While it is accepted that there is an increased long-term risk of ESRD in living donors crucially the absolute risk in studies remains very low at $\leq$ 0.5%.

Similarly, although the relative risk of pre-eclampsia is considered two fold higher than in a healthy non donor, there are good maternal and fetal outcomes reported in pregnancies after living donation. The evidence does not indicate an increased risk of low birthweight or prematurity.

It is postulated that the mechanism of pre-eclampsia in this setting is different (i.e. not related to placental insufficiency) and subsequently not deleterious to fetal outcome.

The applicability of historic studies to today's potential living donors is uncertain, as the demographic profile has evolved as donors now are older, and the potential donor population is increasingly more likely to be obese, have impaired glucose metabolism, and hypertension.

Risks of renal failure vary between ethnic groups, another factor for which long-term data are scarce.

Overall, despite an increased relative risk of long-term complications, there must be appreciation of the excellent outcomes for those who have donated after being assessed in accordance with national and internationally agreed guidelines.[12]

For the vast majority of living donors the experience is a positive one, often associated with improved physical and psychological wellbeing. There are definitely donors who have increased their life expectancy by modifying their lifestyle in order to become eligible to donate.

The benefits of seeing a close relative or friend transformed by transplantation cannot be overstated, and the risk a potential donor considers acceptable is frequently different from that of the medical profession.

A person from one ethnic group can potentially donate to a friend or partner from another ethnic group. A genetic relationship with the recipient is not a prerequisite to being a living donor, and even an emotional association is no longer essential. In the past decade non directed altruistic living donors, those that do not have a particular person to donate to, known as Good Samaritan donors, have made an important contribution to transplantation with more than 80 donating annually in the UK. Each can trigger a chain of transplants by entry into the UK Kidney Sharing Scheme.[13]

More recently direct solicitation of a living donor using social media has resulted in directed altruistic donation, approximately a dozen times per annum in the UK.

ASSESSMENT OF POTENTIAL LIVING DONORS

The purpose of assessment is to ensure, as far as possible, that the risks for that individual in being a living donor are acceptably low. It is not essential to have perfect health in order to be a donor. Table 2, p22, summarises the key aspects of assessment.

Consideration of long-term risks to renal function is key. First, it must be established that there are two kidneys and no current concern in relation to »

Table 2

Assessing suitability for living kidney donation

General anaesthesia/ laparoscopic surgery	**Obesity**		**Smoking**		**Previous thrombosis**	
	Increased perioperative risks		Increased perioperative risks		Increased perioperative risks	
	Current renal function		**Risk for renal disease**			
Renal function after donation	Creatinine Isotopic GFR	Urinalysis	Obesity Blood pressure Diabetes	Familial disease	Stone disease	
	Technical		**Immunological**		**Disease transmission**	
Kidney for transplant into recipient	Vascular anatomy		Blood group	HLA type	Infective	Malignant

renal function. Second, there needs to be determination of any particular risks for this individual in terms of the development of renal disease in the long term, which means that the person should not be left with a single kidney.

One increasingly frequent concern is obese individuals. Persistent hyperfiltration in a single kidney, together with the concomitant higher risk of hypertension and diabetes will compromise long-term function. While some individuals are sufficiently motivated to lose a substantial quantity of weight to permit donation, maintaining such weight loss lifelong is crucial.

Occasionally, the risk of familial renal disease is of sufficient concern to preclude donation.

The final part of the assessment process mandates consideration of three areas:

- Technical feasibility of donation and implantation
- The risk of transmissible disease (malignancy or infection)
- The compatibility of blood group and HLA loci between donor and recipient.

Surgical teams rarely decline a donor on a technical basis as there are a number of reconstructive techniques and options to overcome vascular complexity. With the possibility of living donor exchange programmes in many countries, the finding of incompatibility no longer precludes donation, provided that the donor is willing for entry into such a sharing scheme.

Figure 1, p19, illustrates the typical minimal assessment process. How this is organised varies between centres, it is possible to have a streamlined donor journey which is popular with potential donors by adopting the ethos of making it as easy as possible to donate.[14] Additional investigations are required if initial screening reveals another potentially relevant pathology.

POTENTIAL COMPLICATIONS IN RECIPIENTS

There are a number of technical and immunological issues that can be problematic in the immediate post-transplant period, when the patient is still in hospital. The most common early complications following discharge are infection (most typically urine), wound dehiscence, and poor glycaemic control. Transplant recipients are under close review by the transplant team in this period, but primary care teams may be asked by medical staff to assist in care.

In the medium term the most common cause of death in transplant recipients is cardiovascular disease, and management of risk factors is an important aspect of GP care.

With time, avoidance of malignancy becomes the most likely determinant of survival. Minimisation of immuno-suppression is the remit of the transplant clinicians; encouraging adherence to screening protocols and having a high index of suspicion for potential neoplasia that of the primary care team.

Particular relevant practical care for transplant recipients relates to their ongoing immunosuppressed state: live vaccines should not be administered and infections must be treated swiftly and for longer as these patients can develop sepsis and become critically unwell quickly.

MONITORING OF DONORS AND RECIPIENTS

Living kidney donors are typically followed by the transplant centre in the early weeks after donation, and lifelong annual follow-up is recommended. The arrangements for this vary between regions. Historically, when there were very small numbers of living donors, this was often by clinic attendance at the transplant unit. With rising numbers, (there are now more than 1,000 living donors annually in the UK), and increasing time from donation, alternative processes are being put in place in some areas. The key aspects for donor follow-up are BP, urine protein estimation, and serum creatinine/eGFR.

Box 2

Renal function after living kidney donation

Reduced nephron mass
Serum creatinine will be substantially higher and eGFR substantially lower than previously

- A lower eGFR in this instance does NOT represent chronic kidney disease
- The remaining kidney is perfectly good, it has been carefully assessed
- The change in renal function simply reflects reduced nephron mass

There is a normal variability in serum creatinine (and hence eGFR) of at least 10% in all individuals, e.g. if creatinine is 130 μmol/L then values between 115 μmol/L and 145 μmol/L are essentially the same

- Additional investigation or referral is not required unless there is a progressive decline

A lower eGFR therefore should not cause alarm unless there are other health concerns or it continues to decline

key points

SELECTED BY

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GP, Suffolk, UK

Kidney transplantation is the optimum form of renal replacement therapy for suitable patients with end-stage renal disease. A successful transplant represents a much closer approximation to true renal replacement with excretion of waste products, regulation of electrolytes and other substances, than dialysis. The longer a recipient has been dialysis dependent before transplantation the poorer the outcome. Length of time on dialysis is the strongest independent modifiable risk factor for renal transplant outcome.

Post-transplant antirejection drugs reduce the body's normal immunosurveillance function thereby increasing the risk of de novo and recurrent malignancy. Also, patients with current or recurrent infections must have careful consideration of the risk-benefit ratio associated with transplantation. Live vaccines should not be administered. Infections must be treated swiftly and for longer as these patients can develop sepsis and become critically unwell quickly.

The demography of donors has changed substantially from the early years of transplantation, when the typical deceased donor was young and male. Today, many have preexisting comorbidities. Living donor transplantation on the other hand is associated with better graft and patient survival than transplantation from a deceased donor. A genetic relationship with the recipient is not a prerequisite to becoming a living donor.

Having a living donor offers the opportunity of pre-emptive transplant (i.e. avoidance of dialysis altogether). In the UK, 40% of living donor transplants in 2017-18 were pre-emptive, compared with 16% of transplants from deceased donors. Almost all centres now offer minimally invasive laparoscopic surgery for donors.

The risk of a major complication for donors is 1-2%, and risk of death is around 1 in 3,000. Following nephrectomy, few living donors have persistent problems. In the longer term, hypertension, renal failure, and premature death are potential risks. All donors should be followed up with BP, urine protein, and serum creatinine/eGFR testing.
Every living donor will have a lower eGFR after donation which is completely different from the same eGFR in someone with two kidneys. For the vast majority of living donors the experience is a positive one, often associated with improved physical and psychological wellbeing.

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Box 3

Essential information for patients about living kidney donation

- Living donor kidney transplantation is the very best treatment for end-stage renal disease
- It is possible to avoid dialysis completely if there is a suitable available donor
- A donor does not have to be genetically related to the recipient
- There are options for living donation even if the donor is a different blood group or ethnic group from the recipient
- All potential donors undergo a thorough assessment
- Nobody is permitted to donate unless their short- and long-term risks are low
- The absolute risk of a serious complication for living donors is small
- All donors are offered lifelong follow-up
- It is possible to donate a kidney to a stranger
- Typically the experience of being a living donor is positive

Every living donor will have a lower eGFR after donation. Substantial anxiety is caused when donors are told they have CKD. It is important to note that a lower eGFR in a donor, with one kidney, is completely different from the same eGFR in someone with two kidneys, as this will represent a disease process in the latter case but not in a donor.

Box 2, p22, summarises the advice given to primary care colleagues by one transplant unit. If the eGFR trend does cause concern, referral back to the nephrology/transplant team is appropriate and typically welcomed by both the donor and the nephrologist. Box 3, above, summarises information about living kidney donation.

Recipients attend nephrology services lifelong, not least as virtually all still have a below normal GFR. However, they require the expertise of primary care for the non-renal aspects of health.

CONCLUSION

There are significant advantages with kidney transplantation compared with dialysis, and importantly in graft and recipient survival with living compared with deceased donor transplantation. Outcomes for donors are good when suitability is determined in accordance with established guidelines.

Appreciation of the benefits of living donor transplantation and facilitation of the process will result in an increased number of living donors. It is typically a positive experience.

Competing interests: None

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Useful information

Kidney Research UK
www.kidneyresearchuk.org/health-information/kidney-transplantation

NHS Blood and Transplant information on living donation
www.organdonation.nhs.uk/about-donation/living-donation

Information on non-directed altruistic living kidney donation
www.giveakidney.org

Information on donation and transplantation
www.donatelife.co.uk

CPD prompt and framework
Journal article

The Practitioner

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

CPD prompt and framework
Case review

The Practitioner

Clinical case for review

Reason for selection of the case for reflection

Evidence base used to reflect on your practice

What went well?

What could have been done better?

What were the causes of the condition/event?

What learning needs arise from this case for (a) you and (b) for the practice?

Summary of any discussion of the case with peers

What changes/actions have resulted to your personal practice?

What changes/actions have resulted to the practice?

How/when will you review these changes?

Time credits	Impact credits	Total credits

CPD prompt and framework
Audit

The Practitioner

Title

Reason for the audit (target to improve or implement)

Define criterion (or criteria) to be met

Set the standard and its basis

Plan the audit process and any involvement of peers

Time credits **Impact credits** **Total credits**

Reflection on Table 1 results

What changes were implemented to improve results?

Reflection on Table 2 results and review with peers

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					