Inflammatory arthritis such as rheumatoid and psoriatic arthritis are chronic diseases requiring long-term management. The overall goal of treatment is to achieve remission or sustained low disease activity.\(^1\) The majority of patients are managed effectively using disease-modifying antirheumatic drugs (DMARDs).

Traditionally, patients have been managed in secondary care by planned consultation with a rheumatologist every 3–6 months. This system of follow-up accounts for >75% of a rheumatologist’s workload\(^2\) and evidence has shown that up to 45% of routine consultations are undertaken without demonstrated patient need.\(^3\) Projected future shortages of specialist physicians including rheumatologists mean this arrangement may be untenable in the near future.\(^3\) Greater emphasis has therefore been placed on managing patients with stable inflammatory arthritis within the community using shared-care arrangements. Innovations within primary care have developed the role of specialist nurse practitioners and interface teams responsible for the continued monitoring of DMARDs.

Shared care is defined as:

‘... the joint participation of GPs and specialists in the planned delivery of care for patients with a chronic condition, informed by an enhanced information exchange, over and above the routine discharge/referral letters.’\(^*\)

This model has been used successfully for >10 years in chronic conditions including asthma and diabetes. Studies have consistently demonstrated that versions of shared care are not inferior to traditional follow-up in controlling disease activity in patients with inflammatory arthritis.\(^3,4\) Furthermore, symptom control facilitated by shared care is likely to be more cost-effective than treatment delivered in secondary care in 60–90% of cases.\(^3\)

In the UK it is becoming common to manage patients with stable inflammatory arthritis within primary care using a shared-care arrangement, once therapy is well established and low disease activity persistent (typically >3 months). Figure 1 shows a patient journey from initial symptoms to the shared-care domain.

The British Society for Rheumatology has produced guidelines on monitoring and prescribing of DMARDs to aid implementation of effective shared care.\(^8\) This has been consolidated by the National Institute for Health and Care Excellence (NICE) Clinical Knowledge Summaries.\(^3\) Together these form the basis of inflammatory arthritis shared-care guidelines.

**RECOMMENDATIONS FOR DMARD MONITORING IN SHARED CARE**

DMARDs are essential in arresting the disease process in inflammatory arthritis. Early initiation is crucial to prevent significant disability and sustained use is vital to maintain disease suppression. However, many DMARDs have potentially severe adverse effects, requiring long-term monitoring. The most frequently prescribed DMARDs include methotrexate, azathioprine, sulfasalazine, and hydroxychloroquine.

Vaccinations should always be offered to prevent serious infections in patients receiving DMARDs. A pneumococcal vaccine should ideally be given 2–4 weeks before initiating DMARD therapy. This should be repeated at 10-yearly intervals (5-yearly if given after DMARD initiation).\(^9\) An annual influenza vaccination should also be given;\(^7\) live vaccines (for example, rubella and yellow fever) should be avoided.

**Methotrexate.** This has become an anchor DMARD in the treatment of inflammatory arthritis. Patients are usually initiated on 7.5–10 mg orally once weekly, increased incrementally to a maximum dose of
**Table 1. Blood monitoring requirements for commonly used DMARDs (adapted from NICE Clinical Knowledge Summaries)**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Monitoring</th>
<th>Full blood count</th>
<th>Urea &amp; electrolytes</th>
<th>Liver function tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methotrexate</td>
<td>2-weekly until 6 weeks after the last dose increase, monthly thereafter</td>
<td>White blood cells &lt;3.5 × 10^9/L</td>
<td>Moderate renal impairment (&lt;50 mL/minute)</td>
<td>AST or ALT &gt; twice the upper limit of normal</td>
</tr>
<tr>
<td>Sulfasalazine</td>
<td>Fortnightly for the first 3 months, then every 4 weeks for the second 3 months and 3-monthly thereafter</td>
<td>White blood cells &lt;3.5 × 10^9/L</td>
<td>Only required monthly for the first 3 months, clinically indicated thereafter</td>
<td>AST or ALT &gt; twice the upper limit of normal</td>
</tr>
</tbody>
</table>
| Azathioprine  | Weekly for 6 weeks and continue every 2 weeks until dose is stable for 6 weeks then monthly  
In patients heterozygous for TPMT, monitoring should be monthly (all tests) | White blood cells <3.5 × 10^9/L | Only required 6-monthly | AST or ALT > twice the upper limit of normal |

*aIf mean cell volume (MCV) >105 fL, check folate, vitamin B12, and thyroid-stimulating hormone. If abnormal treat any underlying abnormality. If normal discuss with specialist team. ALT= alanine aminotransferase. AST = aspartate aminotransferase. DMARD = disease-modifying antirheumatic drugs. eGFR = estimated glomerular filtration rate. TPMT = thiopurine methyltransferase.

25 mg once weekly according to response. Incorrect prescribing, mainly associated with an inappropriate frequency, has been associated with several fatalities. Occasionally, patients may be prescribed subcutaneous methotrexate injections if they develop severe nausea while taking oral therapy. Folic acid is prescribed once weekly to reduce potential adverse effects. After a minimum of 8 weeks of treatment, monitoring may be transferred from secondary care; this is typically when treatment should be withheld, an urgent full blood count taken, and any blood test anomalies discussed with the specialist team.

**Sulfasalazine.** This is a prodrug broken down within the gut to sulfapyridine and 5-aminosalicylic acid. Therapy is typically initiated at 500 mg twice daily, increased by 500 mg increments weekly; a typical maintenance dose is 2–3 grams daily (divided doses). Clinical effect is often delayed and may not occur for 3 months. Monitoring is transferred once the patient is stable and there are no significant adverse effects.

Common adverse effects include nausea, dizziness, and headache. These are frequently transient and treatment should be continued unless severe or protracted. Allergic reactions including skin rashes are common, particularly if the patient has had previous reactions to sulphur-containing drugs (for example, co-trimoxazole). In patients presenting with abnormal bruising, pallor, severe sore throat, fever, or malaise, treatment should be withheld and an urgent full blood count taken. Patients should be asked at each consultation about rash or oral ulceration and any unusual results or symptoms should be discussed with the specialist team.

**Azathioprine.** This is a potent immunosuppressant used as a ‘steroid-sparing’ agent. It is rapidly converted in vivo to 6-mercaptopurine. Thiopurine methyltransferase (TPMT) enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azathioprine. Genetic polymorphisms of TPMT enzyme is a key enzyme in the metabolism of azath


10. The management of any chronic illness presents a challenge for both patients and healthcare professionals. Primary care is the ideal place for the holistic management of chronic illness as the primary care consultation provides the appropriate framework for addressing the chronic nature of inflammatory arthritis and overlapping comorbidities such as hypertension, diabetes, and hyperlipidaemia.

CONCLUSION

Patients with chronic conditions are the most frequent users of primary care. The management of any chronic illness presents a challenge for both patients and healthcare professionals. Primary care is the ideal place for the holistic management of chronic illness as the primary care consultation provides the appropriate framework for addressing the chronic nature of inflammatory arthritis and overlapping comorbidities such as hypertension, diabetes, and hyperlipidaemia.

It is vital that GPs are using shared-care arrangements effectively and appropriately and highly focused education sessions led by disease specialists may be a useful tool in achieving this.

INTERVENTION

Potential obstacles for the implementation of effective shared care for inflammatory arthritis include unfamiliarity of GPs with prescribing and monitoring requirements of commonly used DMARDs. To address this issue, in collaboration with the Hammersmith and Fulham Clinical Commissioning Group, a short educational course was developed for GPs (and repeated at regular intervals).

The educational course was designed to provide an interactive forum promoting discussion and knowledge exchange between participants. Succinct lectures were given covering the most commonly prescribed DMARDs with integrated case studies illustrating key management points. Each lecture was followed by group discussion of clinical scenarios with a consultant rheumatologist directing the sessions. To gauge impact, pre-course and post-course assessment questionnaires were completed.

To date, 29 participants have completed the educational sessions and this intervention is currently ongoing. Pre-course questionnaires indicated that the majority of course attendees had received some (n = 19) or very little (n = 7) training beforehand about managing DMARD therapy. Following the session, more than 95% of GPs reported a significant increase in knowledge of the topic relevant to their practice as a result of attendance. Over 85% of participants felt that this enhanced knowledge would significantly improve their clinical practice.

Funding

This work was undertaken by the National Clinical Guideline Centre for Acute and Chronic Conditions, which received funding from the National Institute for Health and Care Excellence. The views expressed in this publication are those of the authors and not necessarily those of NICE.

Provenance

Freely submitted; externally peer reviewed.

Competing interests

The authors have no competing interests to declare.

Acknowledgements

We would like to thank Dr David Mummery, Ms Sophie Ruiz, and the Hammersmith and Fulham Clinical Commissioning Group for their help and support developing the educational sessions.

Discuss this article

Contribute and read comments about this article: bjgp.org/letters