



MANAGEMENT OF ACUTE PELVIC INFLAMMATORY DISEASE

This is the second edition of this guideline, which was previously published in 2003 under the same title.

1. Purpose and scope

Pelvic inflammatory disease (PID) is usually the result of infection ascending from the endocervix causing endometritis, salpingitis, parametritis, oophoritis, tubo-ovarian abscess and/or pelvic peritonitis. While sexually transmitted infections (STIs) such as *Chlamydia trachomatis* and *Neisseria gonorrhoeae* have been identified as causative agents, additional STIs including *Mycoplasma genitalium*, anaerobes and other organisms may also be implicated.¹⁻⁵

PID is a common cause of morbidity and accounts for one in 60 general practitioner consultations by women under the age of 45 years.⁶ Delays of only a few days in receiving appropriate treatment markedly increase the risk of sequelae, which include infertility, ectopic pregnancy and chronic pelvic pain.^{7,8} Sequelae may also have significant healthcare costs.⁹ This guideline applies to women requiring treatment for confirmed or suspected acute PID being treated in an outpatient or inpatient setting by primary and secondary care practitioners.

There are marked variations in the antimicrobial regimens used in the treatment of PID, reflecting uncertainty in the optimal treatment schedule.¹⁰ The guideline contains recommendations for treatment and graded evidence to support their use.

2. Identification and assessment of evidence

A Medline search was performed covering 1963 to August 2007 looking for the following terms in the title or abstract: 'pelvic inflammatory disease', 'adnexitis', 'oophoritis', 'parametritis', 'salpingitis' or 'adnexal disease' (the dataset for 1963-86 was limited to Argonne Information Management journals and human subjects); 7211 citations were identified. A search of the Cochrane database revealed no directly relevant systematic reviews. A search of the Cochrane controlled trials register using a search strategy of 'pelvic inflammatory disease', 'adnexitis', 'oophoritis', 'parametritis', 'salpingitis' or 'adnexal disease' identified 356 citations. The following guidelines and reports were also reviewed: 2006 US Centers for Disease Control STD treatment guidelines,¹¹ Royal College of Obstetrics and Gynaecology Study Group proceedings on PID 1996,¹ 2004 Health Technology Assessment report, *The Clinical Effectiveness and Cost Effectiveness of Antibiotic Regimens for Pelvic Inflammatory Disease*,¹² 2005 UK *National Guidelines on Sexually Transmitted Diseases*¹³ and 2007 European guidelines for the management of pelvic inflammatory disease.¹⁴

The recommendations given in this guideline have been graded according to the guidance for the development of RCOG Green-top Guidelines.

3. Making a diagnosis of acute PID

3.1 Clinical

A low threshold for empiric treatment of PID is recommended because of the lack of definitive clinical diagnostic criteria and because the potential consequences of not treating of PID are significant. In clinically severe cases, referral to hospital for treatment and further investigation is advisable.

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The following clinical features are suggestive of a diagnosis of PID:

- bilateral lower abdominal tenderness (sometimes radiating to the legs)
- abnormal vaginal or cervical discharge
- fever (greater than 38°C)
- abnormal vaginal bleeding (intermenstrual, postcoital or 'breakthrough')
- deep dyspareunia
- cervical motion tenderness on bimanual vaginal examination
- adnexal tenderness on bimanual vaginal examination (with or without a palpable mass).

Clinical symptoms and signs lack sensitivity and specificity (the positive predictive value of a clinical diagnosis is 65–90% compared with laparoscopic diagnosis but laparoscopy may also lack sensitivity).^{15–18} The presence of excess leucocytes on a wet-mount vaginal smear is associated with PID^{19,20} but is also found in women with isolated lower genital tract infection.

Laparoscopy enables specimens to be taken from the fallopian tubes and the pouch of Douglas and can provide information on the severity of the condition.^{2,21} Although it has been considered the gold standard in many studies of treatment regimens, 15–30% of suspected cases may have no laparoscopic evidence of acute infection, despite organisms being identified from the fallopian tubes.^{2,16,17} When there is diagnostic doubt laparoscopy may, however, be useful to exclude alternative pathologies.^{2,17}

Transvaginal ultrasound scanning may be helpful when there is diagnostic difficulty. When supported by power Doppler, it can identify inflamed and dilated tubes and tubo-ovarian masses. It may differentiate PID from acute appendicitis in a minority of cases but there is insufficient evidence to support its routine use.^{22,23} Computed tomography²⁴ and magnetic resonance imaging^{25–27} can assist in making a diagnosis but the evidence is limited. A peripheral blood leucocytosis, elevated erythrocyte sedimentation rate or C-reactive protein also support the diagnosis and can provide a useful measure of disease severity²⁸ but these are non-specific findings. There is insufficient evidence to support endometrial biopsy as a routine diagnostic test at present.^{29,30}

Evidence
level 3

The differential diagnosis of lower abdominal pain in a young woman includes:

- ectopic pregnancy
- acute appendicitis
- endometriosis
- irritable bowel syndrome (and, less commonly, other gastrointestinal disorders)
- complications of an ovarian cyst, such as rupture or torsion
- urinary tract infection
- functional pain (pain of unknown physical origin).

3.2 Microbiological

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Women with suspected PID should be tested for gonorrhoea and chlamydia.

Although not a prerequisite to justify initial treatment decisions, testing for gonorrhoea and chlamydia in the lower genital tract is recommended. A positive result gives support to the clinical diagnosis of PID and reinforces the need to treat sexual partners. The absence of confirmed infection in the lower genital tract site does not exclude PID.^{2,11,16}

Testing for gonorrhoea should be with an endocervical specimen and tested via culture (direct inoculation on to a culture plate or transport of swab to laboratory within 24 hours) or using a nucleic acid amplification test (NAAT). If gonorrhoea is detected using a NAAT, an additional endocervical swab should be taken for gonococcal culture to allow the reporting of antibiotic sensitivities and revision of therapy if required (women at high risk of gonorrhoea should have an endocervical swab for gonococcal culture taken at their first examination; for example, where the woman's partner has gonorrhoea, clinically severe disease, sexual contact abroad).

Testing for chlamydia should also be from the endocervix, preferably using a NAAT (such as polymerase chain reaction, strand displacement amplification).

Taking an additional sample from the urethra can increase the diagnostic yield for gonorrhoea and chlamydia but is only recommended if the more sensitive NAAT is not available. A first catch urine or self-taken vulvovaginal swab sample provides an alternative sample for some NAATs.³¹

The absence of endocervical or vaginal pus cells on a wet-mount smear has a good negative predictive value (95%) for a diagnosis of PID but their presence is non-specific (poor positive predictive value (17%).^{19,20,32}

Other organisms, including *M. genitalium*,^{4,33-35} have been associated with PID but routine screening is not yet justified because of limited information on prevalence, natural history, treatment and cost effectiveness.³⁶

Further advice on the appropriate testing for STIs is available from the *National Screening and Testing Guidelines for Sexually Transmitted Infections* (www.bashh.org/guidelines).

Evidence level 2+

4. Starting treatment

4.1 How should PID be managed in the outpatient setting?

Information on current and recent medication should be obtained.



Interactions between antibiotic therapy and hormonal contraception and other patient medications should be assessed and appropriate action taken.⁴⁸



Outpatient antibiotic treatment should be commenced as soon as the diagnosis is suspected.

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In mild or moderate PID (in the absence of a tubo-ovarian abscess) there is no difference in outcome when women are treated as outpatients or admitted to hospital.³⁷ It is likely that delaying treatment, especially in chlamydia infections, increases the severity of the condition and the risk of long-term sequelae such as ectopic pregnancy, subfertility and pelvic pain.^{7,8}

Evidence level 1+

Outpatient antibiotic treatment should be based on one of the following regimens:

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- oral ofloxacin 400 mg twice daily plus oral metronidazole 400 mg twice daily for 14 days³⁸⁻⁴¹
- intramuscular ceftriaxone 250 mg single dose,* followed by oral doxycycline 100 mg twice daily plus metronidazole 400 mg twice daily for 14 days.^{38,39,42-44}

* Cefoxitin has a better evidence base for the treatment of PID than ceftriaxone but is not easily available in the UK. Ceftriaxone is therefore recommended.

Broad-spectrum antibiotic therapy is generally required to cover *N. gonorrhoeae*, *C. trachomatis* and anaerobic infection.^{1,2,11} Ofloxacin should be avoided in women who are at high risk of gonococcal PID because of increasing quinolone resistance in the UK. Those women at high risk of acquiring gonorrhoea include those whose partner has gonorrhoea, in clinically severe disease or if there is a history of sexual contact abroad.

Metronidazole may be discontinued in those women with mild or moderate PID who are unable to tolerate it, since its addition provides uncertain additional efficacy in this patient group.

Clinical trial evidence for the following regimen is less strong but it may be used as an alternative to the treatments above:

Evidence level 1-

- intramuscular ceftriaxone 250 mg immediately, followed by azithromycin 1 g/week for 2 weeks.^{45,46}

Although the combination of oral doxycycline and metronidazole (without ceftriaxone) has been used to treat PID in the UK, there are no clinical trials adequately assessing its effectiveness and its use in isolation is not recommended.^{10,39,47} Data supporting azithromycin monotherapy for PID is also limited at present and its use without the addition of ceftriaxone is not recommended.

There are currently no randomised controlled trial data to support the use of an oral (rather than parenteral) cephalosporin as part of the treatment regimen. Tissue levels of the antibiotic are likely to be lower following oral administration.

A detailed explanation of their condition should be provided to women, with particular emphasis on the long-term implications for their health and the health of their partner(s). This should be reinforced with clear and accurate written information.



When giving information to patients, the clinician should consider the following:

- an explanation of what treatment is being given and its possible adverse effects
- that following treatment fertility is usually maintained but there remains a risk of future infertility, chronic pelvic pain or ectopic pregnancy
- repeat episodes of PID are associated with an exponential increase in the risk of infertility
- future use of barrier contraception will significantly reduce the risk of PID
- the need to screen her sexual contacts for infection to prevent her becoming reinfected
- clinically more severe disease is associated with a greater risk of sequelae
- the earlier treatment is given the lower the risk of future fertility problems.

A suitable patient information leaflet for PID is available at: www.rcog.org.uk/resources/public/pdf/Acute_PID_2004.pdf.

4.2 What hospital treatment should be given and when should it be recommended?

Admission to hospital would be appropriate in the following circumstances:¹¹

- surgical emergency cannot be excluded
- clinically severe disease
- tubo-ovarian abscess
- PID in pregnancy
- lack of response to oral therapy
- intolerance to oral therapy.

Inpatient antibiotic treatment should be based on intravenous therapy which should be continued until 24 hours after clinical improvement and followed by oral therapy.

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Recommended regimens are:

- **ceftriaxone 2 g by intravenous infusion daily plus intravenous doxycycline 100 mg twice daily,* followed by oral doxycycline 100 mg twice daily plus oral metronidazole 400 mg twice daily for a total of 14 days** ^{11,38,39,43,44}
* Oral doxycycline may be used if tolerated.
- **intravenous clindamycin 900 mg three times daily plus intravenous gentamicin,* followed by either**
- **oral clindamycin 450 mg four times daily to complete 14 days**
OR
- **oral doxycycline 100 mg twice daily plus oral metronidazole 400 mg twice daily to complete 14 days.** ^{11,39,43,44}
* Gentamicin should be given as a 2 mg/kg loading dose followed by 1.5 mg/kg three times daily [or a single daily dose of 7 mg/kg may be substituted].
- **intravenous ofloxacin 400 mg twice daily plus intravenous metronidazole 500 mg three times daily for 14 days.** ^{38,39,49}

The clinical trial data support the use of cefoxitin for the treatment of PID but this agent is not easily available in the UK so ceftriaxone, which has a similar spectrum of activity, is recommended. An alternative third-generation cephalosporin would also be acceptable.

Intravenous doxycycline is available from IDIS World Medicines (+44 [0] 1932 824000). If parenteral gentamicin is used then serum drug levels and renal function should be monitored.

The choice of an appropriate treatment regimen will be influenced by robust evidence on local antimicrobial sensitivity patterns, robust evidence on the local epidemiology of specific infections, cost, the woman's preference and compliance and severity of disease.

Evidence of the efficacy of antibiotic therapy in preventing the long-term complications of PID is currently limited.

4.3 Treatment in pregnancy and in young women

A pregnancy test should be performed in all women suspected of having PID to help exclude an ectopic pregnancy. When the risk of ectopic pregnancy is judged clinically to be high, the pregnancy test should be repeated 21 days after the date of last unprotected intercourse.

The risk of giving any of the recommended antibiotic regimens in very early pregnancy (before a positive pregnancy test) is low, since significant drug toxicity results in failed implantation (UK National Teratology Information Service).

PID is rare in women with an intrauterine pregnancy except in the case of septic abortion. In septic abortion, the infective organism is unlikely to be a sexually transmitted pathogen. Cervicitis may, however, occur in a pregnancy and is associated with increased maternal and fetal morbidity. Treatment regimens will be dependent upon the organisms isolated. Drugs known to be toxic in pregnancy, such as tetracyclines, should be avoided.

A combination of cefotaxime, azithromycin and metronidazole for 14 days may be used. The risks associated with metronidazole are uncertain but no confirmed associations with adverse outcomes have been reported.

Ofloxacin should be avoided, where possible, in young women, when bone development is still occurring. However, this recommendation is based on data from animal studies and no problems have been reported in human subjects, so the British National Formulary currently recommends that ofloxacin may be used in children where other options are limited. Doxycycline can be safely used in children over the age of 12 years.

A particularly low threshold for diagnosing and treating PID in women under the age of 25 years is appropriate, owing to the higher incidence of disease in this group and the potential impact on future fertility.

4.4 Treatment in a woman with an intrauterine contraceptive device

Consideration should be given to removing an intrauterine contraceptive device (IUD) in women presenting with PID, especially if symptoms have not resolved within 72 hours. B

The randomised controlled trial evidence for whether an IUD should be left in place or removed in women presenting with PID is limited.^{50,51} Removal of the IUD should be considered and may be associated with better short-term clinical outcomes but the decision to remove it needs to be balanced against the risk of pregnancy in those who have had otherwise unprotected intercourse in the preceding 7 days. Hormonal emergency contraception may be appropriate for some women in this situation. Evidence level 1+

5. Other modes of treatment

Surgical treatment should be considered in severe cases or where there is clear evidence of a pelvic abscess. B

Consider drainage of an abscess and in noting its position, the possibility that the abscess may have arisen from the appendix or colon. ✓

Laparoscopy may help early resolution of the disease by division of adhesions and drainage of pelvic abscesses.⁵² Ultrasound-guided aspiration of pelvic fluid collections is less invasive and may be equally effective.^{53,54} Evidence level 3

6. Management of sexual partner(s) of women with PID

When a sexually transmitted infection is either proven or likely to be the cause of PID, the current sexual partner(s) should be contacted and offered health advice and screening for gonorrhoea and chlamydia. B

Other recent sexual partners may also be offered screening. Tracing of sexual partners within a 6-month period of the onset of symptoms is recommended but this time period may be influenced by the sexual history. The risk of detecting STIs in the partners of women with PID is high.² Women should be advised to avoid intercourse until they and their partner have completed the treatment course. Gonorrhoea diagnosed in their sexual partner should be treated appropriately and concurrently with the index woman. Concurrent empirical treatment for chlamydia is recommended for all sexual partners, owing to the variable sensitivity of currently available diagnostic tests. If adequate screening for gonorrhoea and chlamydia in the sexual partner(s) is not possible, empirical therapy for both gonorrhoea and chlamydia should be given.^{55,56} Currently recommended regimens are available at www.bashh.org. Tracing of sexual partners is not required where a non-sexually transmitted pathogen has been clearly identified as the cause of infection. Evidence level 3

Referral of the index woman and her partner to a genitourinary medicine/sexual health clinic is recommended to facilitate contact tracing and infection screening.



7. Review of women with PID

In the outpatient setting, review at 72 hours is recommended, particularly for those with a moderate or severe clinical presentation.



Failure to improve clinically suggests the need for further investigation, to exclude competing diagnoses, and may require admission for parenteral therapy and/or surgical intervention.

Further review 4–6 weeks after therapy may be useful to ensure:

- adequate clinical response to treatment
- compliance with oral antibiotics
- screening and treatment of sexual contacts
- awareness of the significance of PID and its sequelae
- that a repeat pregnancy test is negative, if clinically indicated.

Repeat testing for gonorrhoea after treatment is recommended in those initially found to be infected unless sensitivity testing of the isolate confirms sensitivity to the prescribed antibiotic.

Repeat testing for chlamydia and gonorrhoea is appropriate in those in whom persisting symptoms, compliance with antibiotics and/or tracing of sexual contacts indicate the possibility of persisting or recurrent infection.

Evidence level 4

A repeat chlamydia and gonorrhoea test is not otherwise required.

8. Women who are infected with HIV

Women with PID who are also infected with HIV should be treated with the same antibiotic regimens as women who are HIV negative.



Women who are infected with HIV may have clinically more severe PID but respond equally well to treatment as women who are not infected.^{57–59} Standard antibiotic treatment as outlined above is therefore appropriate and hospital admission is only required for those with clinically severe disease. Potential interactions between antibiotics and antiretroviral medication need to be considered on an individual basis (information on drug interactions with antiretroviral drugs is available at www.hiv-druginteractions.org).

Evidence level 3

Women with HIV should be managed in conjunction with their HIV physician.



9. Contraception options and PID

Women on hormonal contraception presenting with breakthrough bleeding should be screened for genital tract infection, especially *C. trachomatis*.



The use of the combined oral contraceptive pill has usually been regarded as protective against symptomatic PID.⁶⁰ Retrospective case-control and prospective studies have, however, shown an association with an increased incidence of asymptomatic cervical infection with *C. trachomatis*.⁶¹ This has led to the suggestion that the oral contraception may mask endometritis.⁶²

An IUD only increases the risk of developing PID in the first few weeks after insertion.⁶³ One European randomised trial compared efficacy and continuation rates of copper-containing IUDs and the levonorgestrel-releasing intrauterine system (LNG-IUS). At 3 years, there were significantly fewer removals for PID in the LNG-IUS group.⁶⁴

All women diagnosed with PID should be provided with information about future contraceptive options and should be assisted in making an informed choice.

If a woman is likely to be at risk of future PID and requests an IUD for contraception, the LNG-IUS would be the most appropriate choice⁶³



10. Auditable standards

Little is known about the long-term outcomes, in relation to future fertility, ectopic pregnancy and chronic pelvic pain, following the treatment of PID. Appropriate short-term audit outcomes include:¹³

1. Proportion of women receiving treatment with a recommended regimen – target 95%.
2. Proportion of women referred for tracing of sexual contacts – target 95%.
3. Proportion of named male contacts in STI associated PID confirmed to have been screened for infection and/or treated – target 60%.
4. Proportion of women having an adequate sexual history documented – target 95%.
5. Proportion of women in whom microbiological investigations have been taken – target 90%.

For service organisation, please see: Royal College of Obstetricians and Gynaecologists. *Standards in Gynaecology. Report of a Working Party*. 2008 [[www.rcog.org.uk/resources/public/pdf/GYNStandardsWPR\(web\)0608.pdf](http://www.rcog.org.uk/resources/public/pdf/GYNStandardsWPR(web)0608.pdf)].

References

1. Recommendations arising from the 31st Study Group: The Prevention of Pelvic Infection. In: Templeton A, editor. *The Prevention of Pelvic Infection*. London: RCOG Press; 1996. 267–70.
2. Bevan CD, Johal BJ, Mumtaz G, Ridgway GL, Siddle NC. Clinical, laparoscopic and microbiological findings in acute salpingitis: report on a United Kingdom cohort. *Br J Obstet Gynaecol* 1995;102:407–14.
3. Baveja G, Saini S, Sangwan K, Arora DR. A study of bacterial pathogens in acute pelvic inflammatory disease. *J Commun Dis* 2001;33:121–5.
4. Simms I, Eastick K, Mallinson H, Thomas K, Gohhale R, Hay PE, *et al*. Associations between *Mycoplasma genitalium*, *Chlamydia trachomatis* and pelvic inflammatory disease. *Sex Transm Infect* 2003;79:154–6.
5. Haggerty CL, Hillier SL, Bass DC, Ness RB. PID Evaluation and Clinical Health study investigators. Bacterial vaginosis and anaerobic bacteria are associated with endometritis. *Clin Infect Dis* 2004;39:990–5.
6. Simms I, Vickers MR, Stephenson J, Rogers PA, Nicoll A. National assessment of PID diagnosis, treatment and management in general practice: England and Wales. *Int J STD AIDS* 2000;11:440–4.
7. Hillis SD, Joesoef R, Marchbanks PA, Wasserheit JN, Cates W Jr, Westrom L, *et al*. Delayed care of pelvic inflammatory disease as a risk factor for impaired fertility. *Am J Obstet Gynecol* 1993;168:1503–9.
8. Leping LA, Hillis SD, Marchbanks PA, Joesoef MR, Peterson HB, Westrom L. Severity of pelvic inflammatory disease as a predictor of the probability of live birth. *Am J Obstet Gynecol* 1998;178:977–81.
9. Yeh JM, Hook EW 3rd, Goldie SJ. A refined estimate of the average lifetime cost of pelvic inflammatory disease. *Sex Transm Dis* 2003;30:369–8.
10. Ross JD. Outpatient antibiotics for pelvic inflammatory disease. *BMJ* 2001;322:251–2.
11. US Centers for Disease Control. Sexually Transmitted Diseases Treatment Guidelines 2006. *MMWR Morb Mort Wkly Rep* 2006;55(RR-11):1–94.
12. Meads C, Knight T, Hyde C, Wilson J. The clinical effectiveness and cost effectiveness of antibiotic regimens for pelvic inflammatory disease. 2004. West Midlands Health Technology Assessment Collaboration, University of Birmingham [www.rep.bham.ac.uk/2004/Pelvic_Inflammatory_Disease.pdf].
13. Ross JDC. British Association for Sexual Health and HIV UK National Guidelines for the Management of Pelvic Inflammatory Disease. 2005 [www.bashh.org/documents/118/118.pdf].
14. Ross JD, Judlin P, Nilas L. European guideline for the management of pelvic inflammatory disease. *Int J STD AIDS* 2007;18:662–6.
15. Gaitan H, Angel E, Diaz R, Parada A, Sanchez L, Vargas C. Accuracy of five different diagnostic techniques in mild-to-moderate pelvic inflammatory disease. *Infect Dis Obstet Gynecol*. 2002;10:171–80.
16. Morcos R, Frost N, Hnat M, Petrunak A, Caldito G. Laparoscopic versus clinical diagnosis of acute pelvic inflammatory disease. *J Reprod Med* 1993;38:53–6.
17. Cibula D, Kuzel D, Fucikova Z, Svabik K, Zivny J. Acute exacerbation of recurrent pelvic inflammatory disease. Laparoscopic findings in 141 women with a clinical diagnosis. *J Reprod Med* 2001;46:49–53.
18. Molander P, Finne P, Sjoberg J, Sellors J, Paavonen J. Observer agreement with laparoscopic diagnosis of pelvic inflammatory disease using photographs. *Obstet Gynecol* 2003;101:875–80.
19. Peipert JE, Ness RB, Blume J, Soper DE, Holley R, Randall H, *et al*. Clinical predictors of endometritis in women with symptoms and signs of pelvic inflammatory disease. *Am J Obstet Gynecol* 2001;184:856–63.

20. Hakakha MM, Davis J, Korst LM, Silverman NS. Leukorrhea and bacterial vaginosis as in-office predictors of cervical infection in high-risk women. *Obstet Gynecol* 2002;100:808-12.
21. Kinghorn GR, Duerden BI, Hafiz S. Clinical and microbiological investigation of women with acute salpingitis and their consorts. *Br J Obstet Gynaecol* 1986;93:869-80.
22. Molander P, Sjoberg J, Paavonen J, Cacciatore B. Transvaginal power Doppler findings in laparoscopically proven acute pelvic inflammatory disease. *Ultrasound Obstet Gynecol* 2001;17:233-8.
23. Taipale P, Tarjanne H, Ylostalo P. Transvaginal sonography in suspected pelvic inflammatory disease. *Ultrasound Obstet Gynecol* 1995;6:430-4.
24. Bennett GL, Slywotzky CM, Giovanniello G. Gynecologic causes of acute pelvic pain: spectrum of CT findings. *Radiographics* 2002;22:785-801.
25. Tukeya TA, Aronen HJ, Karjalainen PT, Molander P, Paavonen T, Paavonen J. MR imaging in pelvic inflammatory disease: comparison with laparoscopy and US. *Radiology* 1999;210:209-16.
26. Imaoka I, Wada A, Matsuo M, Yoshida M, Kitagaki H, Sugimura K. MR imaging of disorders associated with female infertility: use in diagnosis, treatment, and management. *Radiographics* 2003;23:1401-21.
27. Nishino M, Hayakawa K, Iwasaku K, Takasu K. Magnetic resonance imaging findings in gynecologic emergencies. *J Comput Assist Tomogr* 2003;27:564-70.
28. Miettinen AK, Heinonen PK, Laippala P, Paavonen J. Test performance of erythrocyte sedimentation rate and C-reactive protein in assessing the severity of acute pelvic inflammatory disease. *Am J Obstet Gynecol* 1993;169:1143-9.
29. Wiesenfeld HC, Hillier SL, Krohn MA, Amortegui AJ, Heine RP, Landers DV, et al. Lower genital tract infection and endometritis: insight into subclinical pelvic inflammatory disease. *Obstet Gynecol* 2002;100:456-63.
30. Ross JD. What is endometritis and does it require treatment? *Sex Transm Infect* 2004;80:252-3.
31. Ross JD, Ison CA. UK National Screening and Testing Guidelines for STIs. *Sex Transm Infect* 2006;82 Suppl IV:1-5.
32. Yudin MH, Hillier SL, Wiesenfeld HC, Krohn MA, Amortegui AA, Sweet RL. Vaginal polymorphonuclear leukocytes and bacterial vaginosis as markers for histologic endometritis among women without symptoms of pelvic inflammatory disease. *Am J Obstet Gynecol* 2003;188:318-23.
33. Clausen HF, Fedder J, Drasbek M, Nielsen PK, Toft B, Ingerslev HJ, et al. Serological investigation of Mycoplasma genitalium in infertile women. *Hum Reprod* 2001;16:1866-74.
34. Jensen JS. Mycoplasma genitalium infections. Diagnosis, clinical aspects, and pathogenesis. *Dan Med Bull* 2006;53:1-27.
35. Haggerty CL, Totten PA, Astete SG, Ness RB. Mycoplasma genitalium among women with nongonococcal, nonchlamydial pelvic inflammatory disease. *Infect Dis Obstet Gynecol* 2006;30:184:1-5.
36. Ross JDC, Jensen JS. Mycoplasma genitalium as a sexually transmitted infection: Implications for screening, testing, and treatment. *Sex Transm Infect* 2006;82:269-71.
37. Ness RB, Soper DE, Holley RL, Peipert J, Randall H, Sweet RL, et al. Effectiveness of inpatient and outpatient treatment strategies for women with pelvic inflammatory disease: results from the Pelvic Inflammatory Disease Evaluation and Clinical Health (PEACH) Randomized Trial. *Am J Obstet Gynecol* 2002;186:929-37.
38. Martens MG, Gordon S, Yarborough DR, Faro S, Binder D, Berkeley A. Multicenter randomized trial of ofloxacin versus cefoxitin and doxycycline in outpatient treatment of pelvic inflammatory disease. Ambulatory PID Research Group. *Southern Med J* 1993;86:604-10.
39. Walker CK, Kahn JG, Washington AE, Peterson HB, Sweet RL. Pelvic inflammatory disease: metaanalysis of antimicrobial regimen efficacy. *J Infect Dis* 1993;168:969-78.
40. Soper DE, Brockwell NJ, Dalton HP. Microbial etiology of urban emergency department acute salpingitis: treatment with ofloxacin. *Am J Obstet Gynecol* 1992;167:653-60.
41. Peipert JF, Sweet RL, Walker CK, Kahn J, Rielly-Gauvin K. Evaluation of ofloxacin in the treatment of laparoscopically documented acute pelvic inflammatory disease (salpingitis). *Infect Dis Obstet Gynecol* 1999;7:138-44.
42. Arredondo JL, Diaz V, Gaitan H, Maradiegue E, Oyarzun E, Paz R, et al. Oral clindamycin and ciprofloxacin versus intramuscular ceftriaxone and oral doxycycline in the treatment of mild-to-moderate pelvic inflammatory disease in outpatients. *Clin Infect Dis* 1997;24:170-8.
43. Hemsell DL, Little BB, Faro S, Sweet RL, Ledger WJ, Berkeley AS, et al. Comparison of three regimens recommended by the Centers for Disease Control and Prevention for the treatment of women hospitalized with acute pelvic inflammatory disease. *Clin Infect Dis* 1994;19:720-7.
44. The European Study Group. Comparative evaluation of clindamycin/gentamicin and cefoxitin/doxycycline for treatment of pelvic inflammatory disease: a multi-center trial. *Acta Obstet Gynecol Scand* 1992;71:129-34.
45. Savaris RF, Teixeira LM, Torres TG, Edelweiss MI, Moncada J, Schachter J. Comparing ceftriaxone plus azithromycin or doxycycline for pelvic inflammatory disease: a randomized controlled trial. *Obstet Gynecol* 2007;110:53-60.
46. Bevan CD, Ridgway GL, Rothermel CD. Efficacy and safety of azithromycin as monotherapy or combined with metronidazole compared with two standard multidrug regimens for the treatment of acute pelvic inflammatory disease. *J Int Med Res* 2003;31:45-54.
47. Piyadigamage A, Wilson J. Improvement in the clinical cure rate of outpatient management of pelvic inflammatory disease following a change in therapy. *Sex Transm Infect* 2005;81:233-5.
48. FFPRHC Guidance: Drug interactions with hormonal contraception. *J Fam Plann Reprod Health Care* 2005;31:139-51.
49. Witte EH, Peters AA, Smit IB, van der Linden MC, Mouton RP, van der Meer JW, et al. A comparison of pefloxacin/metronidazole and doxycycline/metronidazole in the treatment of laparoscopically confirmed acute pelvic inflammatory disease. *Eur J Obstet Gynecol Reprod Biol* 1993;50:153-8.
50. Soderberg G, Lindgren S. Influence of an intrauterine device on the course of an acute salpingitis. *Contraception* 1981;24:137-143.
51. Altunyurt S, Demir N, Posaci C. A randomized controlled trial of coil removal prior to treatment of pelvic inflammatory disease. *Eur J Obstet Gynecol Reprod Biol* 2003;107:81-4.
52. Reich H, McGlynn F. Laparoscopic treatment of tuboovarian and pelvic abscess. *J Reprod Med* 1987;32:747-52.
53. Aboughar MA, Mansour RT, Serour GI. Ultrasonographically guided transvaginal aspiration of tuboovarian abscesses and pyosalpinges: an optional treatment for acute pelvic inflammatory disease. *Am J Obstet Gynecol* 1995;172:1501-3.
54. Corsi PJ, Johnson SC, Gonik B, Hendrix SL, McNeeley SG Jr., Diamond MP. Transvaginal ultrasound-guided aspiration of pelvic abscesses. *Infect Dis Obstet Gynecol* 1999;7:216-21.
55. Haddon L, Heason J, Fay T, McPherson M, Carlin EM, Jushuf IH. Managing STIs identified after testing outside genitourinary medicine departments: one model of care. *Sex Transm Infect* 1998;74:256-7.
56. Groom TM, Stewart P, Kruger H, Bell G. The value of a screen and treat policy for Chlamydia trachomatis in women attending for termination of pregnancy. *J Fam Plann Reprod Health Care* 2001;27:69-72.
57. Bukusi EA, Cohen CR, Stevens CE, Sinei S, Reilly M, Grieco V, et al. Effects of human immunodeficiency virus 1 infection on microbial origins of pelvic inflammatory disease and on efficacy of ambulatory oral therapy. *Am J Obstet Gynecol* 1999;181:1374-81.
58. Cohen CR, Sinei S, Reilly M, Bukusi E, Eschenbach D, Holmes KK, et al. Effect of human immunodeficiency virus type 1 infection upon acute salpingitis: a laparoscopic study. *J Infect Dis* 1998;178:1352-8.
59. Irwin KL, Moorman AC, O'Sullivan MJ, Sperling R, Koestler ME, Soto I, et al. Influence of human immunodeficiency virus infection on pelvic inflammatory disease. *Obstet Gynecol* 2000;95:525-34.
60. Vessey MP. Epidemiologic studies of oral contraception. *Int J Fertil* 1989;34 Suppl:64-70.
61. Cottingham J, Hunter D. Chlamydia trachomatis and oral contraceptive use: a quantitative review. *Genitourin Med* 1992;68:209-16.

62. Rice PA, Schachter J. Pathogenesis of pelvic inflammatory disease. What are the questions? *JAMA* 1991;266:2587-93.
63. Grimes DA. Intrauterine device and upper-genital-tract infection. *Lancet* 2000;356:1013-19.
64. Toivonen J, Luukkainen T, Allonen H. Protective effect of intrauterine release of levonorgestrel on pelvic infection: three years' comparative experience with levonorgestrel- and copper-releasing intrauterine devices. *Obstet Gynecol* 1991;77:261-4.

APPENDIX

Clinical guidelines are: 'systematically developed statements which assist clinicians and patients in making decisions about appropriate treatment for specific conditions'. Each guideline is systematically developed using a standardised methodology. Exact details of this process can be found in Clinical Governance Advice No. 1: *Development of RCOG Green-top Guidelines* (available on the RCOG website at www.rcog.org.uk/index.asp?PageID=75). These recommendations are not intended to dictate an exclusive course of management or treatment. They must be evaluated with reference to individual patient needs, resources and limitations unique to the institution and variations in local populations. It is hoped that this process of local ownership will help to incorporate these guidelines into routine practice. Attention is drawn to areas of clinical uncertainty where further research may be indicated.

The evidence used in this guideline was graded using the scheme below and the recommendations formulated in a similar fashion with a standardised grading scheme.

Classification of evidence levels	Grades of recommendations
1++ High-quality meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a very low risk of bias	A At least one meta-analysis, systematic reviews or randomised controlled trial rated as 1++ and directly applicable to the target population; or A systematic review of randomised controlled trials or a body of evidence consisting principally of studies rated as 1+, directly applicable to the target population and demonstrating overall consistency of results
1+ Well-conducted meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a low risk of bias	B A body of evidence including studies rated as 2++ directly applicable to the target population and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 1++ or 1+
1- Meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a high risk of bias	C A body of evidence including studies rated as 2+ directly applicable to the target population and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 2++
2++ High-quality systematic reviews of case-control or cohort studies or high-quality case-control or cohort studies with a very low risk of confounding, bias or chance and a high probability that the relationship is causal	D Evidence level 3 or 4; or Extrapolated evidence from studies rated as 2+
2+ Well-conducted case-control or cohort studies with a low risk of confounding, bias or chance and a moderate probability that the relationship is causal	Good practice point
2- Case-control or cohort studies with a high risk of confounding, bias or chance and a significant risk that the relationship is not causal	<input checked="" type="checkbox"/> Recommended best practice based on the clinical experience of the guideline development group
3 Non-analytical studies; e.g. case reports, case series	
4 Expert opinion	

This guideline was produced on behalf of the Guidelines Committee of the Royal College of Obstetricians and Gynaecologists and the British Association for Sexual Health and HIV (BASHH) by:

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The final version is the responsibility of the Guidelines Committee of the RCOG.

The guideline review process will commence in 2011
unless otherwise indicated

The following changes were made to the published document January 2009:

Section 4.4 Treatment in a woman with an intrauterine contraceptive device

Consideration should be given to removing an intrauterine contraceptive device (IUD) in women presenting with PID, especially if symptoms have not resolved within 72 hours.

Section 5. Other modes of treatment

Consider drainage of an abscess and in noting its position, the possibility that the abscess may have arisen from the appendix or colon.

DISCLAIMER

The Royal College of Obstetricians and Gynaecologists produces guidelines as an educational aid to good clinical practice. They present recognised methods and techniques of clinical practice, based on published evidence, for consideration by obstetricians and gynaecologists and other relevant health professionals. The ultimate judgement regarding a particular clinical procedure or treatment plan must be made by the doctor or other attendant in the light of clinical data presented by the patient and the diagnostic and treatment options available.

This means that RCOG Guidelines are unlike protocols or guidelines issued by employers, as they are not intended to be prescriptive directions defining a single course of management. Departure from the local prescriptive protocols or guidelines should be fully documented in the patient's case notes at the time the relevant decision is taken.