Problem Solving in Infection

EDITED BY
STEPHANIE DANCER AND
R. ANDREW SEATON

CLINICAL PUBLISHING

To our long-suffering families at home, namely, husband George and sons Benjamin and Christopher at the Lifeboat station on Glasgow Green; and the lovely Emma and beautiful daughters Kate, Charlotte and Maisie in sunny Bearsden.

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Preface

This collection of clinical cases has been written by specialists and practitioners in infectious diseases, clinical microbiology and others with an interest in infection within their own speciality. Contributors are based at UK hospitals and further afield. The material is aimed at medical students, microbiology and infectious diseases trainees and generalist clinicians. We hope it will also be useful to other medical and non-medical professionals working, training or just interested in infection-related specialties. The cases draw on real-life clinical situations within adult hospital practice. Whilst the majority of cases relate to commonly encountered or less common but serious infections, we have also included rarer or emerging infections, particularly when they illustrate broader principles of infection management and prevention. We hope these cases will help bring the reader up to date in some of the key areas relating to contemporary diagnosis and management of infection in the setting of a developed healthcare system. Most importantly, we hope the reader will be informed, entertained and stimulated to read more about the fascinating subject of infectious diseases. We would like to take the opportunity to thank all our friends and colleagues at home and abroad who have made this book possible, including a special thank-you to Jane Pennington, who should take full responsibility for its genesis.

SJD
RAS
November, 2011

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Abbreviations

3GC	third-generation cephalosporins
AFFB	acid-fast bacilli stain
AIDS	acquired immune deficiency syndrome
APRV	airway pressure release ventilation
ARDS	acute respiratory distress syndrome
ART	antiretroviral therapy
AXR	abdominal X-ray
AZT	zidovudine
BAL	bronchoalveolar lavage
BHIVA	British HIV Association
BM	bacterial meningitis
BNF	British National Formulary
BP	blood pressure
CA-MRSA	community-acquired methicillin-resistant *Staphylococcus aureus*
CAP	community-acquired pneumonia
CDAD	*Clostridium difficile*-associated diarrhoea
CDC	Centers for Disease Control and Prevention
CDI	*Clostridium difficile* infection
CHB	chronic hepatitis B
CMV	cytomegalovirus
CNS	coagulase-negative staphylococci
COPD	chronic obstructive pulmonary disease
CPIS	Clinical Pulmonary Infection Score
CRP	C-reactive protein
CSF	cerebrospinal fluid
CT	computed tomography
CXR	chest X-ray
DDD	defined daily dose
DF2	Dysgonic Fermenter type 2
DFI	diabetic foot infections
DIC	disseminated intravascular coagulation
DOT	directly observed therapy
EBV	Epstein–Barr virus
ECMO	extracorporeal membrane oxygenation
EEG	electroencephalography
eGFR	estimated glomerular filtration rate
EIA	enzyme immunoassay
ELISA	enzyme-linked immunosorbent assay
ENT	ear, nose and throat
ESBL	extended spectrum β-lactamase
FiO_2	inspired oxygen concentration
G6PD	glucose 6-phosphate dehydrogenase
GAS	Group A beta-haemolytic *Streptococcus*
GBS	group B *Streptococcus*
GCS	Glasgow Coma Scale
GI	gastrointestinal
GMP	good manufacturing practice
GP	general practitioner
HAART	highly active antiretroviral therapy
HAI	healthcare-associated infection
HA-MRSA	healthcare-associated methicillin-resistant *Staphylococcus aureus*
HAV	hepatitis A virus
HBsAg	hepatitis B surface antigen
HBV	hepatitis B virus
HCV	hepatitis C virus
HDV	hepatitis Delta virus
Hep B	hepatitis B
Hep C	hepatitis C
HFOV	high-frequency oscillatory ventilation
HIV	human immunodeficiency virus
HSE	herpes simplex encephalitis
HSV	herpes simplex virus
HUS	haemolytic uraemic syndrome
I&D	incision and drainage
IAP	intravenous antibiotic prophylaxis
ICN	infection control nurse
ICP	intracranial pressure
ICU	intensive care unit
IDU	injecting drug use
IDUs	injecting drug users

IE	infective endocarditis
IgG	immunoglobulin G
IgM	immunoglobulin M
IJV	internal jugular vein
IL-6	interleukin-6
ILI	influenza-like illness
IRIS	immune reconstitution inflammatory syndrome
ITU	Intensive Therapy Unit
IV	intravenous
IVIG	intravenous immunoglobulin G
LD	Legionnaires' disease
LDH	lactate dehydrogenase
LGV	lymphogranuloma venereum
LOC	level of consciousness
LP	lumbar puncture
MAOI	monoamine oxidase inhibitor
MDRTB	multidrug-resistant tuberculosis
MHC	major histocompatibility complex
MIC	minimum inhibitory concentration
MRI	magnetic resonance imaging
MRSA	methicillin-resistant *Staphylococcus aureus*
MSM	men who have sex with men
MSQ	mental state questionnaire
MSSA	methicillin-susceptible *Staphylococcus aureus*
MSSU	mid-stream specimen of urine
NAAT	nucleic acid amplification test
NF	necrotizing fasciitis
NICE	National Institute for Health and Clinical Excellence
NMB	neuromuscular blocking agent
NSTI	necrotizing soft tissue infection
OHPAT	outpatient and home parenteral antimicrobial therapy
OI	opportunistic infections
PaO_2	partial pressure of arterial oxygen
PCIRV	pressure control inverse ratio ventilation
PCP	*Pneumocystis jirovecii* pneumonia
PCR	polymerase chain reaction
PEG-INF	pegylated interferon
PEP	post-exposure prophylaxis
PHI	primary HIV infection
PJI	prosthetic joint infection
pO_2	oxygen pressure
PPROM	pre-term premature rupture of membranes
PVL	Panton-Valentine leukocidin
RBC	red blood cell
rBPI	recombinant bactericidal/permeability-increasing protein
RCOG	Royal College of Obstetricians and Gynaecologists
RCT	randomized controlled trial
RDT	rapid diagnostic test
RT-PCR	reverse transcriptase–polymerase chain reaction
SARS	severe acute respiratory syndrome
SBP	spontaneous bacterial peritonitis
SDD	selective digestive tract decontamination
SIRS	systemic inflammatory response syndrome
SpO_2	saturation of peripheral oxygen
SSC	Surviving Sepsis Campaign
SSTI	skin and soft tissue infection
STEC	Shiga toxin-producing *Escherichia coli*
STI	sexually transmitted infection
STSS	streptococcal toxic shock syndrome
TB	tuberculosis
TNF-α	tumour necrosis factor alpha
TOE	trans-oesophageal echocardiogram
TPPA	*Treponema pallidum* particle agglutination
TSS	toxic shock syndrome
TTE	trans-thoracic echocardiogram
URTI	upper respiratory tract infection
UTI	urinary tract infection
VAP	ventilator-associated pneumonia
VDRL	venereal disease research laboratory
VP	ventriculoperitoneal
VRE	vancomycin-resistant enterococci
VT	tidal volume
VTEC	verocytotoxin-producing *Escherichia coli*
WCC	white blood cell count
WHO	World Health Organization
XDRTB	extensively drug-resistant tuberculosis

Principles

THE PATIENT WITH SEPSIS

PROBLEM

1 Assessment of the Patient with Sepsis

R. Andrew Seaton

Case History

A previously healthy 19-year-old woman presented to her general practitioner with fever, myalgia, sore throat and fatigue. Her temperature was 38.2°C, heart rate 100 beats/min and respiratory rate 22 breaths/min. The pharynx was inflamed with moderate exudate. Blood pressure was 100/60 mmHg. She looked well and viral upper respiratory tract infection was suspected. A throat swab for bacteria and viruses was performed and analgesics prescribed. Twelve hours later she collapsed and was taken to the emergency department. A widespread macular rash was evident on the trunk (Figure 1.1), heart rate was 130 beats/min, respiratory rate 34 breaths/min, blood pressure 70/40 mmHg and temperature 39°C. *Streptococcus pyogenes* was isolated from the throat swab.

What are the key clinical indicators of infection and its severity?

What is the likely cause of this person's collapse?

How should this patient be managed?

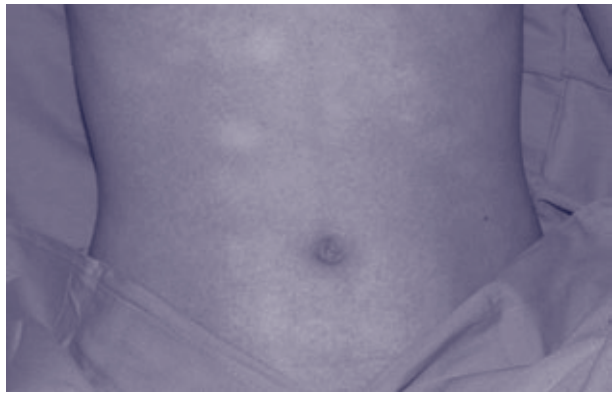

Figure 1.1 A widespread macular rash on the trunk (*see inside front cover for colour version*).

Background

The systemic inflammatory response syndrome (SIRS) reflects the host immune response, including the release of pro-inflammatory cytokines, to a variety of stimuli (infection, ischaemia, chemical toxins, vasculitis and trauma). SIRS is defined by at least two key clinical (fever, hypothermia, tachycardia and tachypnoea) or laboratory (neutrophil leukocytosis or leukopenia) signs of inflammation (Table 1.1). Clinical symptoms and signs of infection in conjunction with SIRS define 'sepsis'. Severe sepsis is sepsis with associated organ dysfunction, hypoperfusion or hypotension. Originally proposed to give consistency in patient assessment for clinical trials in severe infection, these criteria have been shown to correlate well with outcome in everyday clinical practice with mortality increasing with sepsis severity. Septic shock has the poorest prognosis with mortality >50%. In general, sepsis syndrome is a manifestation of serious bacterial infection but it may also be a manifestation of other infections including viraemia (e.g. influenza, measles), parasitaemia (e.g. falciparum malaria) and fungaemia (e.g. candidaemia). Bacterial sepsis is associated with bacteraemia and may be triggered by a number of mechanisms including release of endotoxin (e.g. in Gram-negative infection) or exotoxin release as is seen in staphylococcal or streptococcal toxic shock syndrome (TSS). Early recognition of SIRS, sepsis and severe sepsis should institute prompt targeted antimicrobial therapy against the most likely organisms. Choice of agent will be guided by the anatomical and systemic manifestations of infection and associated epidemiological risk factors (Table 1.2). In true community-acquired sepsis in the UK, the most commonly isolated organisms from blood cultures are *Escherichia coli* (urinary tract source), *Streptococcus pneumoniae* (pneumonia) and *Staphylococcus aureus* (soft tissue or primary bacteraemia). *Streptococcus pyogenes* (Group A beta-haemolytic *Streptococcus* [GAS]) and *Neisseria meningitidis* are important additional risks particularly in younger adults and children.

Toxic shock syndrome

TSS is a severe and life-threatening condition caused typically by either GAS or methicillin-sensitive or methicillin-resistant *Staphylococcus aureus*. TSS has also been described

Table 1.1 Symptoms and signs of the systemic inflammatory response syndrome (SIRS)

SIRS if ≥2 of:		SEPSIS	Severe SEPSIS = SEPSIS + one of:
Tachycardia	Heart rate >90 beats/min	SIRS + symptoms/signs of infection (e.g. rigors, dysuria, erythema, purulent sputum, presence of pus, etc.)	Hypotension: systolic blood pressure ≤90 mmHg despite fluid resuscitation or vasopressor/inotropic support required
Tachypnoea	Respiratory rate >20 breaths/min		Adult respiratory distress syndrome: acute onset of diffuse pulmonary infiltrates and hypoxaemia in the absence of cardiac failure or by evidence of diffuse capillary leak manifested by acute onset of generalized or pleural oedema
Fever or hypothermia	Temperature <36°C or >38°C		Acidosis (blood lactate >4 mmol/l)
			Coagulopathy (disseminated intravascular coagulation or thrombocytopenia, platelet count $<100 \times 10^9$/l)
Leukocytosis or leukopenia	Peripheral white blood cell count <4 or $>12 \times 10^9$/l or band forms present on blood film		Renal failure: oliguria or rising creatinine
			Hepatic dysfunction: increasing bilirubin or transaminases
			Acute alteration in mental status

Table 1.2 Examples of epidemiological risk factors for infection

Risk factor	Nature of risk/infection type	Notable organisms
Healthcare-associated infection (including recent hospitalization or surgery)	Vascular device, urinary catheter related, metalwork/surgery associated, ventilator associated or antibiotic therapy induced. Bloodstream, urinary tract, respiratory tract and enteric infections	*Staphylococcus aureus* (including MRSA), coagulase-negative staphylococci, enterococci, *Clostridium difficile*, resistant Gram-negative infection (e.g. extended spectrum β-lactamase-producing organisms)
Immunosuppressive states and agents	Multiple risks including chemotherapy, biological agents, other rheumatic disease-modifying agents, transplantation antirejection therapy, corticosteroids, malignancy, advanced HIV	Additional risk of opportunistic organisms including low-virulence bacteria, fungi (*Candida, Aspergillus*) and viruses (cytomegalovirus)
Diabetes	Risk factors include vascular disease, impaired humoral immunity, neuropathy. Soft tissue, osteomyelitis, pneumonia, urinary tract	*S. aureus*, streptococci, pneumococci, fungi (*Candida, Aspergillus*)
Parenteral drug misuse	Soft tissue infection, vascular infection, endocarditis, pneumonia, blood-borne virus	*S. aureus*, streptococci, clostridial species, pneumococci
Alcohol excess and chronic liver disease	Pneumonia, spontaneous bacterial peritonitis, meningitis	Pneumococci, listeriosis, streptococci, Gram-negative infection
Occupation and environment	Environmental and zoonotic risk	Leptospirosis, *E. coli* O157, *Coxiella burnetii*
Travel	Nature/region of travel/activities, travel itinerary, rural versus urban, immunization and prophylaxis history	Malaria, enteric fever, Dengue, Rickettsia, meningococci, leptospirosis, imported resistant bacteria (e.g. penicillin-resistant pneumococci

HIV, human immunodeficiency virus; MRSA, methicillin-resistant *Staphylococcus aureus*

with other beta-haemolytic streptococci. In both GAS and *S. aureus* TSS, pyrogenic exotoxins or superantigens are produced which interact directly with the host class II major histocompatibility complex (MHC) molecules. These bind to T-cell receptors and trigger massive polyclonal T-cell activation and a resultant cytokine storm including tumour necrosis factor alpha (TNF-α) and interleukin-6 (IL-6). In GAS TSS, the streptococcal M protein within the cell wall binds to host complement regulators and impedes phagocytosis by mononuclear cells. Clinically, GAS and *S. aureus* TSS may be difficult to differentiate as both are characterized by hypotension and erythroderma. GAS TSS is associated with higher mortality (up to 80%) and there should be clinical and microbiological evidence of GAS, usually a necrotizing soft tissue infection or pharyngitis. Strict definition of GAS TSS requires the presence of hypotension and at least two other features of multiorgan dysfunction (Table 1.3). Lack of multiorgan involvement and hypotension differentiates 'scarlet fever' from GAS TSS. Staphylococcal TSS is less common and has an attributable mortality of about 5%. It typically occurs in young women and is associated with tampon use, menses and intravaginal contraceptive devices but may also complicate skin infections, particularly after surgical procedures. Microbiological tests are typically negative. The diagnosis of staphylococcal TSS is confirmed in the presence of fever, macular rash (which desquamates 1–2 weeks after onset), hypotension and evidence of multiorgan involvement and no positive microbiological cultures (excepting *S. aureus*) (Table 1.3). In the illustrative case, isolation of GAS from the throat swab differentiates between streptococcal and staphylococcal TSS.

Table 1.3 Clinical features of GAS and *S. aureus* TSS

	GAS TSS	*S. aureus* TSS
Hypotension	Always	Always
Fever	Usual	Always
Generalized erythroderma	Usual	Always
Desquamation	May occur	Usual 1–2 weeks after onset
Soft tissue infection	If soft tissue primary site: necrotizing fasciitis, myositis or gangrene	Infrequently primary site. May occur post-operatively
Case definition	**Hypotension, microbiological evidence of GAS infection and ≥2 of:** rash, soft tissue necrosis, renal dysfunction, coagulopathy (thrombocytopenia or disseminated intravascular coagulation), hepatic dysfunction, ARDS, new-onset confusion	**Fever, rash (with desquamation) and hypotension. Other pathogens excluded although *S. aureus* may be isolated and ≥3 of: vomiting/diarrhoea at onset, severe myalgia or creatinine phosphokinase twice normal, mucous membrane hyperaemia, renal dysfunction,** thrombocytopenia, hepatic dysfunction, central nervous system involvement

ARDS, acute respiratory distress syndrome

Management of sepsis and TSS

In the septic patient, blood culture is mandatory as is rapid identification and removal of any potential source of infection (e.g. infected vascular catheter or abscess collection). Empirical parenteral antimicrobial therapy should be administered promptly

after blood cultures. Notably in severe streptococcal infections, where there is a large bacterial load, a high rate of treatment failure with penicillin has been observed. In animal models, clindamycin use has been associated with considerably greater success than penicillin, particularly when therapy is delayed. In these circumstances a stationary growth phase of GAS is reached quickly and *in vitro* experiments have demonstrated that critical penicillin-binding proteins are not expressed at this time, so potentially rendering β-lactam antibiotics ineffective. In contrast to β-lactams, clindamycin maintains activity against GAS irrespective of the growth phase. It is also a potent suppressor of bacterial toxin synthesis and may facilitate phagocytosis of *S. pyogenes* by inhibiting M protein synthesis.

Early physiological supportive measures, in addition to antibiotic management, are extremely important in sepsis including TSS. Severe sepsis should be managed in a high-dependency or intensive care setting. Supportive measures include intravascular volume resuscitation with crystalloid or colloid to improve filling pressure ('goal-directed therapy'), optimization of oxygenation and ventilation (due to the associated ventilation–perfusion abnormalities), correction of acidosis and renal and inotrope/vasopressor support. Adjunctive measures which have been shown to be of benefit in subsets of patients with severe sepsis in the intensive care setting include stress-dose corticosteroids (in adults with septic shock only after blood pressure has been shown to be poorly responsive to fluid and vasopressor therapy) and recombinant activated protein C (in adult patients judged to be at high risk for death by the presence of multiorgan failure and without contraindications based on bleeding risk).

Recent Developments

Despite prompt antibiotic therapy, mortality in GAS TSS remains high and there is a need for specific adjuvant therapy. Lack of protective humoral immunity against GAS virulence factors is thought to contribute to susceptibility to invasive infection and so adjunctive human polyspecific intravenous immunoglobulin G (IVIG) therapy has been proposed. IVIG is able to neutralize a wide variety of superantigens and to facilitate opsonization of GAS. To date the only placebo-controlled randomized trial of IVIG in GAS TSS was terminated prematurely because of slow patient recruitment. Although only 21 patients were enrolled in this multicentre study, there was a 3.6-fold increase in mortality in placebo recipients and a significant increase in plasma neutralizing activity against superantigens expressed by autologous isolates in IVIG-treated patients. Although administration of IVIG therapy for GAS TSS is now widely practised, larger studies are required to corroborate these findings and to better guide clinicians in IVIG use.

Conclusion

Significant improvements have been achieved in the support of patients with severe sepsis in recent years; however, mortality remains high. It therefore remains critical that early signs of sepsis are recognized quickly both in the community and hospital and that appropriate investigations and management are initiated rapidly before severe sepsis ensues.

Further Reading

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PROBLEM

2 Assessing Sepsis in the Elderly

Helen Slavin, Adam Bowman

Case History

A 78-year-old man resident in a nursing home presented to the emergency department. He had previously had a stroke and had a long-term urethral catheter *in situ*. He had been unwell for several days and on initial assessment was confused and drowsy with a blood pressure of 90/40 mmHg and temperature of 37.5°C. His dipstick urinalysis was positive for blood, leukocytes and nitrites. Intravenous co-amoxiclav was started for a presumed urinary tract infection and he initially improved. Several days later, however, he became unwell again, with fever and profuse diarrhoea.

What clinical features are there in this man's initial presentation to suggest an underlying diagnosis of sepsis?

Was his initial antibiotic therapy appropriate and are there any special considerations that should guide empirical antimicrobial prescribing in patients such as this?

Background

Sepsis is a major cause of morbidity and mortality in the elderly population and mortality due to sepsis increases in a linear manner with age. Sepsis is a leading cause of hospitaliza-

tion in this age group. Common sites of infection in elderly patients include the respiratory tract (50% of all cases of pneumonia occur in patients over 65 years of age), urinary tract, and skin and soft tissues. The initial approach to investigation and management of the older patient with sepsis is in essence the same as that for any patient presenting with presumed sepsis. There are, however, important subtleties and pitfalls to be aware of when dealing with sepsis in this patient population.

In elderly patients, multiple predisposing factors provide increased exposure to pathogens and allow infection and subsequent sepsis to develop. Extrinsic factors include comorbidity, poor functional status, immobility, institutionalization, repeated invasive procedures (such as catheterization) and recurrent hospital admissions. Intrinsic factors include age-related changes in immune function, altered homeostasis and decreased cardiopulmonary reserve.

All of the factors noted above have been identified as contributors to the development of sepsis in the elderly; it is readily apparent that many of these apply to the very typical patient outlined above.

It has long been recognized that sepsis may present atypically in elderly patients and this can pose a significant diagnostic challenge to clinicians. Changes in immune function with increasing age (immunosenescence) and altered homeostatic mechanisms mean that the typical presenting features of sepsis may not be present in older patients. Fever response, for example, often the cardinal presenting sign suggesting sepsis, may be blunted or absent, and a significant proportion of elderly patients with bacteraemia will never exhibit a significant fever. It follows therefore that absence of fever cannot rule out infection in these patients. The clinical presentation of sepsis in elderly patients may be very non-specific with weakness, general malaise and falls being common presenting features. Delirium (acute confusional state) is an extremely common presenting feature of sepsis in older adults, particularly in those with multiple comorbidities or pre-existing cognitive impairment. It is important to determine if the confusion is of new onset or worsening of a long-standing confusional state. In this regard, confirmatory history from a carer is essential. Any acute change in cognition or change in functional status in an elderly patient should alert the clinician to the possibility of underlying infection and initial investigations should reflect this.

Initial assessment and management

Regardless of age, initial investigation of the patient presenting with sepsis should be directed towards confirming the diagnosis of infection (routine blood tests, blood cultures, urine cultures, etc.), assessing for possible sources of infection (clinical examination, chest X-ray, etc.) and assessing the severity of sepsis. Urinary catheters (as in this case) are frequently colonized with bacteria, making assessment of urinalysis and culture difficult.

The initial management of sepsis in the elderly should follow the guidelines outlined by the Surviving Sepsis Campaign, with the emphasis (as in any septic patient) on maintaining perfusion pressure via fluid resuscitation ± inotropic support, source identification and early administration of appropriate empirical antibiotic therapy. Removal or replacement of medical devices which may be a potential source of infection, such as intravascular or (as in this case) urinary catheters, should be considered in all patients with sepsis and is mandatory if the source of sepsis is not known.

Initial empirical antibiotic choices are similar in all patients regardless of age, with a few important additional points to consider in older patients. In patients with significant

comorbidity, repeated antimicrobial exposure and recurrent hospitalization are common and therefore levels of antimicrobial resistance may be higher. Changes in pharmacokinetics with increasing age may make antibiotic dosing more difficult and older patients are at higher risk of developing side effects and complications related to antimicrobial use. Frail elderly patients are at a particularly high risk of developing *Clostridium difficile* infection which is frequently associated with poor outcome in this patient group. This is due to a combination of increased prevalence of risk factors which predispose to *C. difficile* infection (comorbidities, recurrent hospital admission, use of agents to suppress gastric acid, etc.) and the presence of factors known to be associated with poor outcomes (hypoalbuminaemia, renal failure, functional impairment, etc.), all of which are more common in frail elderly people. Empirical antimicrobial regimens for these patients should take all of the above into account, as well as likely pathogen and source of infection, and agents less likely to encourage *C. difficile* should be used. Initial antibiotic cover should be rationalized to targeted therapy as soon as culture results allow, and antibiotic regimens limiting the use of antibiotics associated with increased risk of healthcare-associated infections (quinolones, cephalosporins, co-amoxiclav, etc.) should be considered.

Further Considerations

In the case above, where urinary sepsis was suspected, the urinary catheter should be removed and replaced. Although intravenous co-amoxiclav successfully treated this man's urinary sepsis, its use may well have contributed to the subsequent development of *C. difficile*-associated diarrhoea. It would have been preferable in this case to have used an age-, weight- and creatinine-adjusted intravenous dose of gentamicin as empirical cover pending blood and urine culture results. Gentamicin-induced renal, oto- and vestibular toxicity in the elderly is well recognized but can be minimized with short-duration therapy (72 hours or less) and by careful monitoring of blood concentrations and renal function. A switch to an appropriate oral agent may be possible at that stage. Other agents useful in lower urinary tract infection and less likely to encourage *C. difficile* include nitrofurantoin, trimethoprim and co-trimoxazole. Oral options for upper urinary tract infections with lower propensity for *C. difficile* infection are more limited but include co-trimoxazole and amoxicillin following isolation of a susceptible organism.

While many elderly patients presenting with sepsis will respond well to the management strategies outlined above, it is worth remembering that an episode of severe sepsis/septic shock will be a terminal event for a proportion of elderly patients. In these cases appropriate management will involve end-of-life care, which may in turn involve decisions regarding withholding or withdrawal of treatment. Such decisions invariably raise a number of ethical issues and should always be made by senior members of the medical team, taking cognisance of the patient's pre-expressed wishes.

Conclusion

- Sepsis is common in elderly patients.
- The clinical presentation may be atypical leading to difficulty in making the diagnosis.

- Delirium is an extremely common presenting feature and its presence should always prompt a search for underlying infection.
- The principles of initial assessment and management of sepsis are the same in all patients regardless of age.

Further Reading

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PROBLEM

3 Updating Infection Prevention and Control

Stephanie J. Dancer

Case History

Dr Barry is a senior physician in a district general hospital. He has been asked to review the delivery of infection control in the hospital following a request from the hospital's Board of Directors. He has no previous experience other than awareness of screening activities for methicillin-resistant *Staphylococcus aureus* (MRSA). There is one infection control nurse (ICN) assisted by a part-time staff nurse on secondment. There is also an on-site microbiology laboratory but the microbiologist is off on long-term sick leave with cover obtained from another healthcare Trust. How should Dr Barry proceed? The year before, there was an extensive outbreak of norovirus affecting the whole hospital, and rates of MRSA and *Clostridium difficile* infection have recently escalated on the long-term care wards.

What is the first thing that Dr Barry should do?

What are the most important issues to tackle?

What longer-term plans could be initiated?