

EXPANDED CASE SUMMARIES

Guidance for Trainees

Structure

It is important to be prescriptive enough to provide a working framework making these easier to assess with a degree of uniformity while allowing content entirely to the breadth of understanding of the trainee.

In general

- The case should be chosen because it was of interest, had specific learning points, raised major issues or provided an opportunity to explore an area of interest to the trainee.
- The cases should be distributed across curriculum domains.
- Each case should be between 750 and 1500 words
- The curriculum domain should be stated.
- A focused description of the case itself.
- Discussion of the case with a review of related points of interest.
- Conclusion should emphasize what was learnt from the case.

The content

1. Each is a record / summary of a clinical case with which the trainee has been involved and has been of interest.
2. Should illustrate a problem or series of clinical problems.
3. Should have one or more learning points or points of contention or controversy.
4. Should illustrate trainees ability to relate a case succinctly with relevant information.
5. Should enable the trainee to demonstrate the ability to find and evaluate the literature relevant to that case.
6. Should enable the trainee to discuss the case and the arising issues.
7. Should allow a critical appraisal of the case.
8. Does not need to have rarity value. (In fact common day material with lessons may be better than once in a lifetime occurrences)

Marking scheme

- Criterion 1:* Defining a question. The case title should be informative. Is it a reasonable case? Why the case was chosen should be defined in a single paragraph.
- Criterion 2:* Does the report convey the relevant clinical details succinctly?
- Criterion 3:* Are the important features researched and discussed?
- Criterion 4:* Accessing relevant and up to date information relevant to the case.
- Criterion 5:* Drawing conclusions - limitations of review.

Each criterion will score a maximum of 5 marks and each section should score 3 or above prior to submission:

- 5 Outstanding
- 4 Good
- 3 Pass
- 2 Needs improvement
- 1 Poor – needs complete revision

Detailed instructions

1. Use Arial 12 point for the body
2. Use Arial 16 point for headings and 14 point for subheadings, both in bold
3. Don't use jargon or unnecessary acronyms
4. Define abbreviations the first time if to be used again.
5. References should be in the Vancouver style
6. Use SI units
7. Use a 'spell-checker' and consider the grammar and readability of your prose
8. Discuss suitability of the case with the trainer who will also review what has been written prior to submission

Standard

Should be commensurate with a publication in *Anaesthesia* or *BJA* in terms of use of English and content /references etc. These are usually simple, concise and informative. They should conform to our template. They do not need scarcity value nor need to be original in the context of 'different from all other trainees', but do need to be their own cases, their own experience, their own take on the literature and their own lesson.

Suggested layout

Case number : Title
Clinical problem and domain: <i>1 sentence saying why the case was chosen</i> <i>About 200 words describing the essence of the case as it presented to you.</i>
Management: <i>About 200 words describing the relevant aspects of the resuscitation, stabilization and ongoing management of the case, and the eventual outcome.</i>
Discussion: <i>About 300 words on the clinic-pathological details of the case; discuss the relevant literature.</i>
Lessons learnt: <i>About 100 words on what have you gained from this case and how will your future management be different?</i>
References: <i>Give at least four but no more than ten relevant references that you would consider essential reading.</i>

Example Summary:

NOTE: This example is adapted from Part II of a previous edition of *The CCST in Intensive Care Medicine*. This manual now been replaced by *The CCT in Intensive Care Medicine* and is no longer in print, but can be found on the Faculty's website, www.ficm.ac.uk. Further summary examples are available on the trainee section of the ICS website: www.ics.ac.uk.

Acute Respiratory Failure: the role of steroids in persisting ARDS

Clinical problem

A 40-year-old previously fit merchant banker was admitted with a short history of acute breathlessness, fever, cough and malaise. Clinically he has clear evidence of a right basal pneumonia with associated fever, tachycardia and hypotension.

He was hypoxic and tachypnoeic and rapidly required intubation in the ED and transfer to ICU for full mechanical ventilation. Despite the commencement of intravenous broad-spectrum antibiotics and adequate fluid loading he remained hypotensive and required inotropic support. A fully sensitive pneumococcus was isolated from 4 out of 4 blood cultures the next morning and he was changed to intravenous benzylpenicillin.

Thirty six hours after admission his chest x ray, along with his gas exchange, PEEP requirements and clinical history all fitted with the development of acute respiratory distress syndrome from pneumococcal septicaemia.

128 words

Management

His ventilation deteriorated progressively requiring the sequential introduction of inverse ratio and pressure controlled ventilation, high levels of PEEP and then inhaled nitric oxide. He endured a brief period of prone ventilation but with no improvement in his gas exchange.

A percutaneous tracheostomy was performed on the 8th day of admission. After 10 days he still required an FiO₂ of 0.85 and inverse ratio pressure controlled ventilation but had come off all inotropes after the first 5 days.

He was thus commenced on intravenous steroids, methylprednisolone 2 mg/kg daily for 2 weeks, and then a reducing dose over a total of 32 days. This produced a dramatic improvement in his gas exchange. Over the next 36 hours he came off the inhaled nitric oxide and after 3 days of steroid therapy he was on 45% oxygen breathing with 25 cmH₂O pressure support and fully conscious and alert.

147 words

Discussion: Steroids in ARDS

ARDS is a severe and often fatal form of acute microvascular lung injury. Overall mortality remains high at between 40-60%¹ with the majority of patients requiring mechanical ventilation and prolonged intensive care management. Treatment up to the present has been largely supportive.

ARDS is traditionally divided into three phases: exudative, proliferative and fibrotic². The initial 'exudative' phase involves the leakage of proteinaceous fluid and the migration of cells, in particular neutrophils, from the circulation into the interstitium and alveolar space following diffuse damage to the endothelial and epithelial surfaces. The proliferation of fibroblasts and type II pneumocytes characterises the second phase. Activated fibroblasts secrete a number of extracellular matrix proteins, including collagen, within the interstitium but also migrate into the alveolar space where they form attachments to damaged basement membranes³. Unabated, this process leads to established interstitial and intra-alveolar fibrosis.

Approximately 60% of patients with ARDS fail to improve or are deteriorating after one week of ventilation and all of these patients demonstrate mechanical, biochemical and histological evidence of fibrosis^{4,5}. A doubling of lung collagen is observed in patients with ARDS surviving more than two weeks⁴. Progressive hypoxia and a susceptibility to nosocomial infection result in an 80% mortality in this group⁶. Recent evidence suggests mechanical ventilation itself may exacerbate lung injury and stimulate a fibrogenic response in the lung⁷.

There is now evidence that steroids may be of benefit after the initial stages of the illness^{2,8} and this was seen dramatically in this patient who made a huge improvement after their commencement. If steroids are to be used they should be commenced only in those patients who have a significant respiratory dysfunction and have failed to improve more than one week after the onset of ARDS. Trials to date suggest that brief courses of steroids are ineffective, hence treatment should be maintained for more than one week. If a benefit is to be seen this usually occurs within the first five days of institution of steroids.

The mechanism of action for steroids at this stage is unclear but may include effects on permeability, reduction in inflammatory cell load (through both increased apoptosis and/or reduced cellular influx) and reduction in fibroblast proliferation and collagen deposition. The problems with steroids are that they have been shown to make things worse in the initial stages of ARDS and of sepsis and there are concerns that they will lead to increased susceptibility to nosocomial infections. There is a minor risk of steroid psychosis.

415 words

Lessons learnt

I am now aware that there is a place for steroids in the management of this complicated condition but have also been made aware from this literature search that these drugs must be used in clearly defined situations, when there is clinical evidence of non-resolving fibrosis⁸.

46 words

References

1. Suchyta MR, Clemmer TP, Elliott CG, Orme JF, Jr., Weaver LK. The adult respiratory distress syndrome. A report of survival and modifying factors. *Chest* 1992; 101:1074-79
2. Meduri GU, Chinn AJ, Leeper KV, et al. Corticosteroid rescue treatment of progressive fibroproliferation in late ARDS. Patterns of response and predictors of outcome. *Chest* 1994; 105:1516-27
3. Kuhn C, 3rd, Boldt J, King TE, Jr., Crouch E, Vartio T, McDonald JA. An immunohistochemical study of architectural remodelling and connective tissue synthesis in pulmonary fibrosis. *Am Rev Respir Dis* 1989; 140:1693-1703
4. Zapol WM, Trelstad RL, Coffey JW, Tsai I, Salvador RA. Pulmonary fibrosis in severe acute respiratory failure. *Am Rev Respir Dis* 1979; 119:547-54
5. Raghu G, Striker LJ, Hudson LD, Striker GE. Extracellular matrix in normal and fibrotic human lungs. *Am Rev Respir Dis* 1985; 131:281-9
6. Bell RC, Coalson JJ, Smith JD, Johanson WG, Jr. Multiple organ system failure and infection in adult respiratory distress syndrome. *Ann Intern Med* 1983; 99:293-8
7. Parker JC, Hernandez LA, Peevy KJ. Mechanisms of ventilator-induced lung injury. *Crit Care Med* 1993; 21:131-43 (Abs) *Am J Respir Crit Care Med* 14 1997; A613.
8. Meduri GU, Headley AS, Golden E et al. Effect of Prolonged Methylprednisolone therapy in unresolving acute respiratory distress syndrome; a randomised controlled trail. *JAMA* 1998; 280:159-65

216 words

Total words = 952

Appendix of common abbreviations:

ED	Emergency Department
ICU	Intensive Care Unit
ARDS	Acute Respiratory Distress Syndrome