mater education

# RMO GASTROENTEROLOGY HANDBOOK

Last updated: 04.01.2021

# Contents

## WELCOME FROM THE MEU

The Medical Education Unit would like to welcome you to this rotation. Please read this handbook in conjunction with the RMO Orientation Handbook which is accessible on the MEU website via Zenworks or http://mededu.matereducation.qld.edu.au/handbooks/

### MEU Contact Details

If you're experiencing difficulty with any aspect of the term, clinical or otherwise, please contact the term supervisor and/or PVMEO as early as possible.

| | |
|---|---|
| Director of Clinical Training (DCT) | ☎ 8229 |
| Medical Education Officer (MEO) | ☎ 8431 |
| Principal Medical Education Officer (PMEO) | ☎ 1560 |
| Medical Education Admin Officer | ☎ 8272 |
| Medical Education Manager | ☎ 8114 |

## INTRODUCTION

The Department of Gastroenterology at the Mater Hospital Brisbane provides tertiary inpatient and outpatient services to patients in the Metro South Health District and from other districts on specific referral. The average number of inpatients is 5-10 and are preferentially admitted to ward 8A. The Department of Gastroenterology sits within the Surgical / Acute stream however the medical consultant team are physician trained and contribute to physician training and the medicine component of undergraduate training for University of Queensland. Gastroenterology patients at times require surgical input from the colorectal and hepatobiliary teams and so close communications are necessary to ensure a smooth patient journey.

## UNIT OVERVIEW

The Gastroenterology unit is a busy department that provides junior medical staff with excellent exposure to common gastroenterological conditions. Common conditions that the RMO will encounter during their term include:

Inflammatory bowel disease

Gastrointestinal tract bleeding

Biliary tract pathology

Acute presentations of liver disease

Complications of cirrhosis including hepatic encephalopathy, ascites, spontaneous bacterial peritonitis and hepatocellular carcinoma.

General gastroenterological complaints including functional disorders

Outpatient Adult and Young Adult clinics provided by the Department include:

General gastroenterology including interventional endoscopy

Pre-endoscopy (consenting)

Inflammatory Bowel Disease

Hepatology

Consultant staff provide ward and after-hours cover on a rotating basis.

| **Medical Staff** | |
|---|---|
| Consultant Staff: available through teleservices | Marianne Mortimore – 0414 366 794<br>(Director of Gastroenterology & Endoscopy )<br>**Interventional team**:<br>Johannes Wittmann – 0411 016 496<br>Kavin Nanda – 0406 209 279<br>Kevin Tang - 0402690884<br>**Hepatology Team:**<br>Paul Clark – 0466 829 979<br>Mazhar Haque – 0402 858 648<br>Katerina Liew – 0422281547<br>Aidan Woodward – 0413 032 736<br>**IBD team:**<br>Jake Begun – 0447 247 283<br>Peter Hendy – 0434 621 495<br>Marianne Mortimore – as above<br>Nicole Walker – 0428 786 427<br>Mariko Howlett – 0418 719 624<br>Yoon Kyo An – 0402 304 989 |
| Registrars 2021 | Szymon Ostowski (Snr Reg 3rd Yr)<br>Anthony Deacon (Advanced Trainee 1st Yr)<br>Ellie Van der List (PHO)<br>Rotating Basic Trainee |
| RMOs | Rotating JHO/SHO (10 weeks) #0074 |
| **Nursing Staff** | |
| NUM Endoscopy | Ruth Ayers (ext 8368 & 0401 101 025) |
| Endoscopy CNC | Ruth Ayers |
| Endoscopy Team Leader | Rotating (ext: 1630) |

| | |
|---|---|
| Endoscopy Admissions | Ext: 1746 |
| Surveillance nurse | Lingwen Meng – ext |
| Hepatology Nursing Help Line | 0434 569 389 |
| Hepatology NP | 0466 829 979 |
| Hepatology CN | Burglind Liddle (ext: 8452) |
| Hepatology CN | Heather Philpot (ext: 6703) |
| IBD Nursing Help Line | Megan McBean (ext: 6703) |
| IBD CNC | 0466 777 857 |
| IBD CN | Heidi Harris (ext: 5834) |
| IBD CN | Kaity Mullen (ext: 8054) |
| NUM 8A | Deb Tanham |
| NUM DPU | Ann Kuhlmann |
| Outpatients RN / Procedure Bookings | Davina Fiorini (ext: 6138) |
| IBD Research Nurse | Natalie Allan (0400 757 347) |
| Research Study Coordinator | Sharyn Grossman (ext: 8195) |
| **Administration Staff** | |
| Endoscopy Service Manager | Anya Tate (ext:6816) |
| Gastroenterology Outpatients Manager | Anya Tate (ext:6816) |
| Departmental Secretary | Sam van Hamersveld (ext: 8196) |
| Endoscopy Bookings Office | Monique Kent (ext:7954) |
| | Elaine McLachlan ( ext:7955) |
| Administration Officer | Kaity McManus (ext:3803) |
| Endoscopy Bookings | Monique Kent (ext:8183) |
| Outpatient Bookings | Carissa Horstmann (ext:5841 |

**IBD Contacts**

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|---|---|---|---|
| Department Fax | 3163 8213 | | |
| IBD Nurses | IBDnursing@mater.org.au | 0466 777 857 | 3163 8054 |
| Hepatology Nurses | Hepatologynurse@mater.org.au | 0434 569 389 | 3163 6703 |
| Outpatients Admin | GastroOPD@mater.org.au | | |
| MFM Case Manager | Barbara.Soong@mater.org.au | | |
| YASU Psychiatrist | Tatjana.Ewais@mater.org.au | | |
| Dietitian | Peter.Collins@mater.org.au | | |

**Endoscopy Contacts**

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|---|---|
| Endoscopy Team Leader | 3163 1630 |
| Endoscopy Bookings Office | adult.endoscopy.bookings@mater.org.au |
| Endoscopy Bookings Nurse | gascolo@mater.org.au 3163 6138 |
| Endoscopy Team Leaders | Adult.endoscopy.team.leader@mater.org.au |
| EndoVault IT Specialist | James.Eales@mater.org.au |
| Endoscopy NUM | Ruth.Ayers@mater.org.au |
| Endoscopy Surveillance Nurse | Lingwen.Meng@mater.org.au |

**On-Call Contacts**

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|---|---|
| ED Duty Consultant | 3163 7692 |
| ED SSU Consultant | 3163 2575 |
| Endoscopy TL | 3163 1630 |
| Theatre TL | 3163 1875 (theatre TL will get after hours nurse manager to call in endoscopy nurses for a/h scopes) |
| Duty Anaesthetist | 3163 6647 |

| **Contact Group Name**: | **Colorectal Histology & Radiology MDT (Gastro/Hep Nurses, Regs, Consultants)** |
|---|---|
| An, Yoon-Kyo | Yoon.An@mater.org.au |
| Begun, Jake (MMRI) | jakob.begun@mater.uq.edu.au |
| Chang, Linus | Linus.Chang@mater.org.au |
| Clark, Paul | Paul.Clark@mater.org.au |
| Haque, Mazhar | Mazhar.Haque@mater.org.au |
| Harris, Heidi | Heidi.Harris@mater.org.au |
| Hendy, Peter | Peter.Hendy@mater.org.au |
| Hepatology Nurse | hepatologynurse@mater.org.au |
| Howlett, Mariko | Mariko.Howlett@mater.org.au |
| IBD Nursing | IBDNursing@mater.org.au |
| Mickenbecker, Matthew | Matthew.Mickenbecker@mater.org.au |
| Mortimore, Marianne | Marianne.Mortimore@mater.org.au |
| Nanda, Kavin | Kavin.Nanda@mater.org.au |
| Walker, Nicole | Nicole.Walker2@mater.org.au |
| Wittmann, Johannes | Johannes.Wittmann@mater.org.au |
| Woodward, Aidan | Aidan.Woodward@mater.org.au |

| | |
|---|---|
| **Contact Group Name:** | **Colorectal Pathology & Radiology MDT (Path/Rad)** |
| Bastian-Jordan, Matthew | mdb544@gmail.com |
| Dornbusch, Tracie | Tracie.Dornbusch@qldxray.com.au |
| Natasha Koya | Natasha.koya@qldxray.com.au |
| Lawler, Karen | Karen.Lawler@mater.org.au |
| Liu, John | John.Liu@mater.org.au |
| Lourie, Rohan | Rohan.Lourie@mater.org.au |
| Smith, Deborah | Deborah.Smith@mater.org.au |
| Snell, Cameron | Cameron.Snell@mater.org.au |

| | |
|---|---|
| Contact Group Name: | **IBD Fertility & Maternity Service MDT** |
| An, Yoon-Kyo | Yoon.An@mater.org.au |
| Begun, Jake (MMRI) | jakob.begun@mater.uq.edu.au |
| Ewais, Tatjana | Tatjana.Ewais@mater.org.au |
| Gardener, Glenn | Glenn.Gardener@mater.org.au |
| Harris, Heidi | Heidi.Harris@mater.org.au |
| Howlett, Mariko | Mariko.Howlett@mater.org.au |
| Hendy, Peter | Peter.Hendy@mater.org.au |
| IBD Nursing | IBDNursing@mater.org.au |
| Kumar, Sailesh (MMRI) | Sailesh.kumar@mater.uq.edu.au |
| Mortimore, Marianne | Marianne.Mortimore@mater.org.au |
| Petersen, Scott | Scott.Petersen@mater.org.au |
| **Soong, Barbara (Case Manager)** | Barbara.Soong@mater.org.au |
| Thomas, Joseph | Joseph.Thomas@mater.org.au |
| Walker, Nicole | Nicole.Walker2@mater.org.au |

- Allied Health are critical to the multidisciplinary care of Gastroenterology patients.
- Social work, OT and physiotherapy are allocated to the specific ward.
- Social work input is critical often early in the admission for discharge and advanced healthcare planning and for family liaison.
- The Gastroenterology dietician (Peter Collins) sees both inpatients and outpatients and can be contacted on pager #4440.
- There are dedicated allied health staff for the MYAHCB patients with teams for inpatient and outpatient care.

Inpatients requiring C/L psychiatry review is organised through the PAH psychiatry team. This is organised with a phone call to the psychiatry registrar and requested through a specific psych referral accessible through the homepage under Departments – Clinical Liaison Psychiatry "blue slip referral".

## RMO LEARNING OBJECTIVES

RMOs are expected to develop their own learning objectives for this rotation and discuss and confirm with their Supervisors during the first few days of the term. In general terms, the educational objectives for junior doctors is the assessment and management of gastroenterological and hepatological conditions, within the broader context of the patient's general health and circumstances.

To this end the following skills should be developed:

### History

- Learn to take a relevant and thorough history to allow assessment and treatment of the patient, particularly in the context of liver and or gastrointestinal disease

### Physical examination

- Elicit and interpret important diagnostic clinical signs; e.g. signs of malnutrition, sepsis, jaundice, ascites and encephalopathy
- Recognize and assess intra-abdominal pathology including hepatosplenomegaly, abdominal masses, etc
- Rectal pathology – perianal fistulae, rectal masses, fissures etc
- Recognise signs of haemodynamic, respiratory or neurological instability or decompensation
- Accurately assess a patient's fluid resuscitation status, particularly in the context of liver disease

### Diagnostic tests

- Learn the rational and appropriate use of diagnostic tests for common gastroenterological and hepatological conditions:
    - Liver blood test abnormalities
    - Ascites
    - Hepatocellular carcinoma
    - Malabsorption syndromes and diarrhoea
    - Iron deficiency anaemia
    - Inflammatory bowel disease
    - Gastrointestinal tract bleeding

- Biliary and pancreatic pathology
- Foreign body management (food boluses etc)

- Interpret the following diagnostic tests:
  - Pattern of liver function tests (ie cholestatic, hepatitic vs mixed)
  - Serum markers of liver disease severity (ie albumin, bilirubin and INR/ prothrombin)
  - Viral hepatitis serology
  - Iron studies
  - Faeces microscopy and culture
  - Ascitic fluid analysis
  - Auto-antibody screen
  - Gastrointestinal imaging including abdominal X ray, ultrasound and CT scans, and MRI
  - Endoscopy and colonoscopy results – including endoscopic appearance and histopathology
  - Understand the indications for, preparation required, performance of and possible complications of the following tests:
  - Endoscopy
  - Colonoscopy
  - Endoscopic ultrasound
  - ERCP
  - Small bowel capsule study
  - Liver biopsy
  - Large Volume Paracentesis
- Understand the common scoring systems used to define disease severity including
  - Crohn's disease activity index (CDAI) in Crohn's disease
  - MAYO score in Ulcerative colitis
  - Child Turcotte Pugh score and MELD score in patients with chronic liver disease
  - Maddrey's score in alcoholic hepatitis
  - Staging system for cancers

## Treatment and management

- Learn the acute management of common GI conditions as listed above

- Learn the management of medical emergencies particularly haemodynamic compromise from haemorrhage, acute liver failure – paracetamol overdose, sepsis
- Develop management plans for multi-discipline management (e.g. surgical, dietetics, physical therapy), patient's discharge, outpatient follow up and ongoing treatment

### Personal skills

- Develop time management and organizational skills
- Develop communication skills with medical colleagues, particularly being able to present a patient's case clearly and concisely to senior medical staff
- Recognise one's own limitations in knowledge and experience and so being aware of when to seek advice
- Develop communication skills with patients and their relatives
- Work as a member of a clinical team, recognising the role and experience of other health care professionals
- Develop responsible attitudes towards patient care

### Procedural skills

- Independent performance of:
    - Venepuncture and cannulation
    - Arterial blood gas collection
    - Venesection
    - Therapeutic and diagnostic abdominal paracentesis – once deemed competent by the Advanced Training registrar or consultant

## RMO DUTIES AND RESPONSIBILITIES

### General To-Do List

- Verdi Results Acknowledgements
- Ozescribe dictations
- Infusions & Prescriptions (your in-tray)
- Thioguanine Monitoring
- MDT preparation - IBD MDT, CRS MDT, FMS MDT

### Clinics

- Adult IBD OPD – Level 3, Main Building
- Young Adult IBD OPD – Level 4, Salmon Building

- DAE OPD – Level 4, Main Building

**Monitoring**

- Follows Mater Protocol
- Spreadsheet to be handed over separately, for patients initiating 6MP/AZA, adding Allo or other ad-hoc

## Ward duties

- Daily review of all inpatients with the formulation of a management plan under the supervision of the registrar and consultant.
- Admissions
    - Emergency admissions in collaboration with the registrar
    - Elective admissions for pre-procedure
- Coordinate patient care with respect to allied health, specialty referrals, investigation ordering and follow-up.
- Notify Hepatology Clinical nurse about **any** Liver patient admissions or discharges (Ph: 6703 or 0434 569 389)
- Notify IBD Clinical nurse about **any** IBD patient admissions or discharges (Ph: 8054 or 0466 777 857).

**Weekend ward round**

Gastroenterology inpatients are reviewed by a member of the treating team each Saturday and Sunday morning. RMOs and registrars share this on a rotating roster. On completion of the round RMOs are advised to contact the consultant on call to handover the patients for the remainder of the weekend. Unstable patients requiring further review are handed over to the medical registrar. The patients for review are placed on the medical handover program "Confluence" accessible through ZENworks and also accessible on L:DRIVE.

## Day procedure unit

- Venesection: Occasionally haemochromatosis patients attend for routine venesection. There is a standard venesection kit available in the infusion unit. The patient's most recent FBC and Iron studies should be reviewed. Target ferritin for venesection is 50-100ug/L. The patient should not be anaemic. Scheduling for subsequent venesections should be booked on the day. Patients should be discussed with the advanced trainee if there is uncertainty about venesection intervals.
- Biologics infusions:
    - Infliximab / Vedolizumab / Ustekinumab are used for moderate-severe UC and Crohn's
    - Patients should be assessed for contraindications pre-dose e.g. intercurrent infection, heart failure

- Pre-medication with 100mg hydrocortisone, 1g paracetamol and 10mg loratadine should be considered for patients who have experienced previous infusion reactions (such as rash, or anaphylactic reaction), an infliximab 'drug holiday' or are not on concurrent immunomodulators.
- For infusion reactions halt the infusion, assess the patient and notify the registrar.
- ***You will be contacted by the infusion to assess patients who have been flagged by the pre-infusion questionnaire as being potentially not suitable for infusion on the day. You should go and assess the patient and then report to the BPT/AT/Fellow/IBD nurse whether safe to proceed with infusion.***

- Iron infusions
  - Ferrinject infusion 1000 mg in 250 mL over 30 minutes.
- Elective Large Volume Paracentesis (see Mater Guideline on Paracentesis)

### Discharge planning and Summaries

Discharge planning is a process that should commence early in an admission and reviewed regularly on ward rounds. Aim to have patients discharged by 10am on their day of discharge. To facilitate this, it is advised to submit discharge prescriptions to the ward pharmacist the previous afternoon and aim to pre-complete as much of the discharge summary as possible.

Discharge summaries should be concise, timely and follow a logical process that the reader will understand. Ideally the patient should leave hospital with a discharge summary completed but if this is not possible the GP can be contacted by phone for urgent matters then 100% of summaries should be completed within 48hours. To capture appropriate DRG coding data the Gastroenterology Clinical Documentation form is a reference http://quality.mater.org.au/docs/Clinical%20Forms/CF-IID-001670.pdf. Clinical Coder, Damian Stewart (ext: 6523) is available for discussion on individual cases.

## SUPERVISION

Supervision is provided by the consultants, advanced training registrar and senior registrar. Your term supervisor will be one of the Ward Consultants and their Registrar Delegates. Please confirm with Dr Marianne Mortimore at the commencement of your term.

### Scope of Practice

RMOs are not permitted to perform any clinical procedure without direct observation, at least in the first instance. The clinical supervisor will then inform you what is to happen in future, with regard to whether or not direct supervision is required. This will be dependent on the skill itself and level of proficiency exhibited.

Procedures performed within the unit include:

- Endoscopy (diagnostic and therapeutic)
- Colonoscopy (diagnostic and therapeutic)
- Flexible sigmoidoscopy
- ERCP
- Endoscopic ultrasound scans +/- FNA
- Insertion of Percutaneous Endoscopic Gastrostomy (PEG) feeding tubes
- Capsule endoscopy
- "FibroScan" – Transient elastography
- Venesection
- Liver biopsy
- Abdominal paracentesis.

## UNIT ORIENTATION

### Orientation to the Ward

- RMOs participate in unit orientation together to receive a consistent message for the term. The following areas will be covered:
- Handover
- Weekend rosters
- Term learning objectives
- Unit policies and procedures
- How daily clinical handover is conducted and
- Miscellaneous (tour of the department, introductions to staff, location of resus trolley).
- Reporting lines

### Team Orientation

Your term supervisor will conduct a face-to-face team orientation with you within the first three days of the term. The following areas will be covered:

- Reporting lines
- Daily roster - where to be & when, e.g., ward rounds
- Discussion and documentation of your individual learning objectives for the term (see the 'term learning plan' below and start of term orientation checklist)
- Assessment
- Handover with the previous junior doctor
- Start of term checklist

## UNIT POLICIES AND PROCEDURES

### Acute Severe Colitis

Patients presenting with bloody diarrhoea should be referred to the gastroenterology unit for consideration of admission. Commonly such patients will have inflammatory bowel disease, although infection and ischaemia are other possibilities.

Acute Severe colitis is a potentially life threatening condition. A proportion of ulcerative colitis patients present as acute severe colitis and 50% of these go on to need a colectomy in their lifetime.

Acute severe colitis is defined by the Truelove and Witts' criteria as:

1. >6 bloody stools/day

And 1 or more of:

- Temp >37.8°C
- Heart rate >90bpm
- Haemoglobin <105g/dL
- CRP>45mg/L / ESR >30mm/hr

Patients who meet criteria for Acute Severe Colitis should be admitted to hospital for intensive treatment with intravenous steroids to obtain remission. Treatment during the admission should be goal directed with the aim of reduction in bloody stool frequency and signs of systemic toxicity within 3-5 days.

Patients admitted with severe colitis require:

- Intravenous steroids (usually hydrocortisone 100mg QID) unless contraindicated
- Stool pathogen PCR, M/C/S and *C. diff* toxin
- Unprepped flexible sigmoidoscopy within 24-48 hours to assess disease and exclude CMV
- Regular monitoring of ELFTs (including magnesium) and CRP
- Proactive electrolyte replacement
- DVT prophylaxis (e.g. clexane 40mg daily)
- Stool chart (patient to self-record timing of BM, consistency, bleeding)
- Baseline abdominal XR to asses for megacolon, disease extent and proximal faecal loading.
- Avoidance of narcotic analgesia, NSAIDs and antispasmodics (e.g. buscopan) as this increases risk of megacolon and colectomy.
- IBD nurse education and review
- Dietician review

Patients who fail to respond clinically or have a persisting high CRP >45 on day 3 of steroids require rescue therapy with either cyclosporin or infliximab. Patients failing iv steroid therapy should be discussed with a member of the IBD consultant team. In addition, patients requiring rescue therapy should have a Colorectal surgical and stomal therapist review with the view to colectomy if medical treatment fails.

**Upper Gastrointestinal Bleeding**

http://quality.mater.org.au/docs/policies/GD-CLN-900023.pdf

**Paracentesis in Patients with Liver Disease**

http://quality.mater.org.au/docs/policies/GD-CLN-900022.pdf

# UNIT EDUCATION AND TRAINING OPPORTUNITIES

## Unit Education

The learning opportunities include the practical opportunities to achieve the RMOs & available learning objectives.

There are additional opportunities to participate in audit, clinical presentations at the unit meeting and original research projects. Please approach your supervising consultants if you wish to participate in a research or audit opportunity.

## Multidisciplinary Meetings

The following MDTs attended by the Gastroenterology Department:

- IBD MDT weekly Tuesday from 12-1pm (coordinated by IBD Snr Reg)
- Gastroenterology MDT weekly Wednesday from 12-1pm.
- Gastroenterology Morbidity & Mortality occurs quarterly in place of the Gastro MDT from 12-1pm
- Hepatobiliary MDT on every second Wednesday Morning from 7.30am (Coordinated by Adv Trainee)
- Liver Meeting occurs 4 weekly on an alternate Wednesday to the HB MDT from 8.00am
- Histology/radiology MDTs fortnightly on Friday from 12-1pm. (Coordinated by IBD Snr Reg)
- Complex patient MDTs monthly on Friday (week 3) 1-2pm (coordinated by IBD Snr Reg)
- IBD-MFM MDT quarterly on Mondays (coordinated by IBD Snr Reg)

**IBD-MDT**

- Tuesday 12:15 – 13:00,
- MDT for inpatients, complex patients, patients with new issues
    - IBD Consultants, Registrars, IBD Nurses, Allied Health, +/- Colorectal
    - Occasionally QCH attend to handover complex transition patients
- Coordinator duties:
    - Receive emailed referrals and check AT1/PHO for inpatients / consults
    - Prepare list Monday or Tuesday AM
    - People email referrals to you, check with AT1/PHO re ward inpatients
    - Action tasks if required or email responsible person to action task
    - Dictate chart letter using ozescribe (ok to dictate bare minimum and copy/paste MDT document into ozescribe web editor – e.g. "Title of letter: IBD MDT Tue DD/MM/YYYY, next

paragraph, NAME was discussed at the MDT and the discussion & recommendations follow below, documentation to be. Inserted")

**Combined colorectal histology/radiology MDT**

- Fortnightly Friday 12:00 – 12:30 (week 1 and 3)
  MDT for histology and radiology – commonly IBD imaging, resection specimens, incongruous histology, liver histology (most liver imaging goes to HPB MDT which the AT1 coordinates)
- Coordinator duties:
  - Send out list by Tuesday PM to allow radiologist/pathologist time to review, inevitably add-ons do occur
  - Action tasks or email responsible person to action
  - Dictate letter to referring responsible clinician summarising discussion and recommendations
  - Meeting is followed at 12.30 by colorectal cancer MDT
  - Generally any colorectal cancers emailed separately to Lisa Welsh as the Colorectal Cancer MDT is minuted and documented formally for billing purposes and follow-up (Lisa.Welsh@mater.org.au and MDT.Coordinator@mater.org.au)

**IBD-Maternal-foetal medicine MDT**

- Quarterly Mondays, Time varies, Level 3 Main Building, Conference Room 4
- MDT for all pregnant IBD patients and patients planning on pregnancy
- Coordinator duties:
  - Prepare and send out list 1-2 weeks prior to MDT to allow IBD & MFM Consultants time to prepare any issues they want discussed or flag any patients not on the MDT that need discussion
  - Action tasks and document as per IBD-MDT

The schedule for these meetings is located in the notice board inside the Gastro office, level 4 Salmon building, located adjacent to the sleep studies clinic.

The RMO may also be required to present an educational piece at the Wednesday Gastro meetings, one of the Consultants will complete the roster for the new year and pair each presenter with a consultant as mentor.

## UNIT ROSTER & TIMETABLES

### RMO Timetable

Timing for twice weekly consultant ward rounds is a guide and should be discussed with consultant on-call at the commencement of the week. There are separate ward rounds for Liver patients and IBD / general gastroenterology. Interventional and procedural patient management is supervised by the procedural list responsible for the case or their delegate. Please see / email the departmental secretary (Sam Van Hammersveld) for current RMO timetable.

## UNIT TIMETABLE

The Master Gastro Roster is updated and managed by Gastroenterology Senior Administration contact (Sam).

This Roster manages the on call rosters for the department. All leave is recorded in the Service Calendar. The Service Manager will provide you with access to this upon commencement.

### Kronos

Staff will be required to manage their own swaps between themselves and leave cover if they are unable to fulfil their designated allocation. All changes are to be communicated ASAP to the Snr Administrative person to ensure that the roster is updated and that the Hospital switch is updated with any changes.

It is the responsibility of all RMOs to ensure your hours are entered into Kronos as per the roster by the end of each fortnight.

Contract hours for registrars are 76 hours per fortnight, 7:36 per day and with one of the following set patterns – 8:00-16:36 and 1 RDO / month. Set patterns for these regular hours will be entered into your Kronos pattern. Please note that no start before 7:00am will be approved unless instructed by Director. 2020 has seen the introduction of a longer day and an RDO for each Registrar.

Any additional hours done outside of these regular hours are considered OT and will need prior approval by Director/ Consultant. You will need to ensure that all appropriate codes for overtime are included in Kronos and a clear description of the reason for the overtime is provided including UR's/ Patient Names and or brief description.

Without Consultant endorsement & UR numbers, overtime hours will be considered unauthorised and will not be paid.

**ANY UNROSTERED OVERTIME** will need to be authorised (as per RMO Enterprise Agreement Section 5.6) by the relevant Consultant/Director on each occasion.

If you need help managing tasks, speak to the team early; do not wait until your shift ends to speak up.

After 6pm only the on-call registrar is expected to be working; all other registrars should have completed their days work and left. Where this isn't possible, the Consultant or Head of Unit needs to have endorsed the additional hours before the registrar's time sheet can be authorised.

No Fatigue – always ensure that you take the minimum 10 hour break – only return fatigue leave in exceptional circumstances. Please ensure that if this going to affect you, you bring this to the attention of the Director immediately.

Any other variances to your roster (i.e. sick leave or PDL taken) will need to be added to Kronos before the end of the fortnight. If in doubt please email Sam for assistance. In the event that you miss the cut off, Sam will take care of this for you.

On the Monday after each fortnight the Unit Director will check and approve your Kronos timecard.

Please note that registrar timesheets should be completed by 10am Monday of new fortnight.

### ARL/ PDL & Sick Leave

If you wish to apply for ANY leave please ensure that you email Snr Admin (Sam) your request.

Policy stipulates that there is a minimum of 6 weeks between your request and your anticipated leave date. If your leave conflicts with on call or clinics you will need to liaise with your colleagues to arrange swaps to ensure that you are covered.

## A JUNIOR DOCTOR'S GUIDE TO RECOGNISING AND MANAGING DECOMPENSATED LIVER DISEASE

### Chronic liver disease

Prevalence of chronic liver disease is increasing with a rise in hospital admissions for complications leading to chronic liver disease being one of the most common causes of premature mortality. The most common causes of chronic liver disease in Western populations are alcohol, non-alcoholic fatty liver disease (NAFLD) from conditions such as obesity or diabetes, and viral hepatitis. It is important to remember that co-factors often exist (e.g. the patient with NAFLD who drinks heavily) and that these should be concurrently managed.

### Decompensated cirrhosis

This is one of the most common reasons for hospital admission for liver disease. Decompensated cirrhosis is defined as an acute deterioration in liver function in a patient with cirrhosis. It can manifest as increasing or new onset jaundice, ascites, hepatic encephalopathy (HE) or renal impairment. In addition, patients can present with portal hypertensive complications such as GI bleeding from varices. The most frequent cause of decompensation is infection or sepsis. The overall risk of death during an admission with decompensated cirrhosis is 10-20% and 50% at two years 1. If there is organ failure, then "acute on chronic liver failure (ACLF)" is diagnosed. The development of 1, 2, or 3 organ system failures is associated with a high mortality of 21%, 55% and >90% respectively 1. These are some of the sickest patients on the medical wards.

### What leads to an acute admission?

Sepsis, such as from spontaneous bacterial peritonitis (SBP) or other causes of infection, toxins (medications, particularly sedatives, diuretics or drugs which constipate), GI bleeding (variceal and non-variceal), thrombosis, alcohol withdrawal, constipation, acute alcohol hepatitis, dehydration and hyponatraemia can all precipitate a decompensation. Patients will often have multiple precipitants and co-contributing factors at any one time (e.g. SBP, hyponatraemia and alcoholic hepatitis). It is important to remember that they may present atypically (e.g. sepsis/ bacteraemia without fever).

### Cause of death

Patients with cirrhosis die from GI bleeds, sepsis and renal failure. They can be admitted with these complications but they can also develop them during their hospital admission. These complications can easily be missed and delays in appropriate diagnosis, prevention and management of decompensation are associated with poor survival.

### Principals of acute care

Common precipitants should be sought in every patient and treated accordingly. These include: GI bleeding (variceal & non-variceal), sepsis (SBP, chest, urine, skin, cholangitis, other; always look for bacteraemia), alcohol hepatitis, portal vein thrombosis, hepatocellular carcinoma (HCC), drugs (opiates, benzodiazepines, NSAIDs, paracetamol, alcohol), ischemic liver injury (hypotension- any cause), dehydration (change in diuretics?) and constipation.

With every patient always **look** for a precipitant, **treat** the cause and aim to **prevent** it from occurring. Our cirrhosis acute care bundle originated with the British Society of Gastroenterology and has been validated multiple times and supports their use to assist in the timely investigation, diagnosis and management of these complex patients (for the bundle see appendix 1) [2,3,4,& 5]. It should be completed for every decompensated cirrhotic patient admitted to hospital.

### Pathophysiology of portal hypertension

Portal hypertension is characterized by a pathologic increase in portal venous pressure resulting from reduced portal blood flow, increased resistance and subsequent increased pressure. In liver cirrhosis, increased intrahepatic vascular resistance to the portal flow elevates portal pressure and leads to portal hypertension. Once portal hypertension develops, it influences extrahepatic vascular beds in the splanchnic and systemic circulations. This leads to the formation of an extensive network of portosystemic collaterals that divert a significant proportion of portal blood to the systemic circulation, bypassing the liver, leading to collateral vessel formation and arterial vasodilation. This further exacerbates portal hypertension and leads to the complications of chronic liver disease you will encounter on the ward and which are discussed in detail below.

### Acute variceal bleed in cirrhosis

Varices occur when the hepatic venous pressure gradient is greater than >10mmHg. They are present in 50% of cirrhotic patients with a bleeding rate of 5-15% per year 6. The mortality associated with a variceal bleed is approximately 15% at 6 weeks 6.

Patients can present in a variety of ways: shocked, not shocked, haematemesis, melaena and with anaemia. They can also present in a variety of circumstances: patient known to have varices, patient not known to have varices, patient with decompensated liver disease and patient not known to have liver disease.

Management is as for any GI bleed: airway, breathing and circulatory assessment and decide on an appropriate level of care; transfuse as necessary; support abnormal clotting and/or thrombocytopenia; and, request endoscopy. Always remember that patients may be more shocked than you realise, particularly if they are known to have varices and are on a beta-blocker for primary or secondary prophylaxis of variceal haemorrhage.

Management specific to AVB includes the following:

- Care with transfusion: in haemodynamically stable patients a transfusion threshold of <70g/dl has demonstrated significantly improved outcomes for re-bleeding, morbidity and mortality compared to a more liberal strategy 7. Patients should be transfused with a post-transfusion target of approximately 80g/dl (range 70-90g/dl).
- Fresh frozen plasma and platelet transfusions if required, targeting an INR <2.0 8, platelet count >50 and fibrinogen >1.5g/dl 1 & 9.
- Actively seek out infection: send blood cultures, urine culture, order a CXR and perform a diagnostic ascitic tap if the patient has ascites. Infection is the precipitating event in 20% of patients with AVB 10.
- Consider acute alcohol hepatitis and portal vein thrombosis (will need an abdominal US once acute bleed addressed) as potential triggers.
- Irrespective of whether infection is present, all cirrhotic patient's benefit from a prophylactic five to seven day course of a broad spectrum antibiotic through a reduction in rates of re-bleeding, sepsis

and mortality 1,6,9,8 & 11. A third generation cephalosporin such as ceftriaxone 1 gram OD IV is an appropriate choice. Ciprofloxacin 500 mg BD PO is an appropriate oral step-down.

- Use splanchnic vasoconstrictors: commence a continuous IV infusion of octreotide for 72h at a rate of 50mcg/h following a 50mcg bolus 12. Dilute 0.5 mg octreotide into 50ml 0.9% saline to give a final concentration of 10 mcg/ml. Run at 5ml/h I.E. 50mcg/h.
- Stat erythromycin 250mg IV immediately prior to endoscopy is useful to clear the stomach of blood 13. If not readily available, metoclopramide 10-20mg IV in addition to, or as an alternative, should be prescribed. Remember to monitor the patients ECG as both drugs are associated with prolongation of the QTc interval.
- Patients should be commenced on IV PPI (80mg pantoprazole stat followed by 40mg BD) as this helps reduce the incidence of post banding ulceration 14.

With respect to endoscopy timing, unstable patients should be offered urgent endoscopy immediately after resuscitation and only if haemodynamically stable 1, 8 & 9. Haemodynamically unstable patients may require placement of a Sengstaken tube. All other patients should undergo endoscopy within 12h 1, 8 & 9. In patients who continue to bleed despite band ligation, a trans-jugular intrahepatic porto-sytemic shunt (TIPS) procedure can be considered.

Empirical treatment for HE should be commenced in all patients 6.

**Ascites**

Ascites is the most common complication of cirrhosis. It is usually a sign of advancing cirrhosis but other important triggers for the development of ascites include portal vein thrombosis and HCC. These should be actively looked for in every patient presenting acutely to hospital.

Ascites itself is a poor prognostic indicator with 50% mortality at 2 years 1. There is a spectrum of severity which is graded as follows:

- Grade I- radiologically present, not clinically
- Grade II- modest, clinically evident
- Grade III- large volume, with distended, tympanic abdomen

Ascites can also be described as complicated (SBP, renal dysfunction (AKI vs. HRS), uncomplicated (diuretic controlled, not infected, normal renal function) and/ or refractory (diuretic intolerant, diuretic resistant).

**Spontaneous bacterial peritonitis**

SBP is a life threatening complication of ascites related cirrhosis. It has a 30 day mortality in the region of 30% and a 70% recurrence rate within the first year 1, 6 & 15. At any one time it represents 20% of hospitalised patients admitted with cirrhosis and ascites. Fever, pain and peritonism are **often absent**. SBP can be present despite **normal CRP and WCC**. Therefore the diagnosis must be excluded in all cirrhotic patients hospitalised with ascites by performing a diagnostic abdominal paracentesis 1, 6 & 15.

**Diagnostic paracentesis**

This is a mandatory, urgent investigation, for anyone admitted to hospital with decompensation and ascites. It is important in determining the aetiology in those with new ascites and in diagnosing infective complications in those with chronic liver disease. Informed consent should be sought from the patient wherever possible; however, it should not delay performing the procedure if consent cannot be obtained (e.g. unable to consent because of HE). Coagulopathy and thrombocytopenia (both very common in cirrhotic patients) are themselves not absolute contraindications as the incidence of bleeding complications from the procedure has been shown to be very low [1]. Paracentesis should be avoided in DIC [1].

The left lower quadrant is the preferred site given the thinner abdominal wall (landmarks: 3cm above and 3cm medial to the anterior superior iliac crest; stay lateral of the rectus sheath in order to avoid the inferior epigastric artery). Aseptic technique should be used throughout. Skin preparation should be with 70% alcohol/ 2% chlorhexidine and a sterile field should be employed. If the patient has obvious ascites then there is no requirement for US guidance/ US marking prior to performing a diagnostic tap. An 18g needle should be advanced with a 20-50ml syringe using the landmarks previously described.

Blood culture bottles should be inoculated as this doubles organism identification in SBP from 40% to 80% compared to sending a universal container only (make sure they are labelled as containing ascitic fluid) 16. A universal container and EDTA tube should also be sent for cell count, microscopy, total protein and albumin. If this is the patient's first presentation, cytology should also be requested; however, there is no need to send this routinely in patients with recurrent presentations, unless you feel it is clinically indicated.

**Interpretation of diagnostic abdominal paracentesis**

The serum ascites- albumin gradient (SAAG) is very useful to establish a group of causes 17:

- SAAG >= 11g/L: cirrhosis, cardiac failure, nephrotic syndrome
- SAAG < 11g/L: malignancy, TB, pancreatitis

The ascitic fluid total protein is also useful in determining the risk of SBP. SBP is uncommon with ascitic fluid total protein >15g/L; however, there is a significantly increased risk in those patients with ascitic protein < 10g/L 1, 6 & 15.

SBP is an important infection to diagnose promptly. It is diagnosed when the absolute leukocyte count is 250 cells/ cm3 (0.25 x 109/L) and >=50% neutrophils.

With respect to culture results, organisms in SBP are usually a gram negative monoculture. If there are multiple organisms, secondary peritonitis should be suspected (e.g. perforated viscus, cholangitis, incarcerated hernia, and diverticulitis). Gram positive organisms are most likely to be seen after hospitalisation, instrumentation and antibiotic prophylaxis.

**Other investigations**

In those with new or worsening ascites always remember to look for a cause. A cause may be suggested from the history but it is important to always perform an abdominal US looking for portal vein thrombosis or a new HCC (check AFP). HCC is an important consideration in those patients who do not attend follow-up appointments/ have been lost to follow-up, which is not uncommon in this vulnerable patient group.

**Management of ascites specific to SBP**

If SBP is suspected on the basis of ascitic fluid cell counts:

- 3rd Generation cephalosporin empirically (ceftriaxone 1 grams OD) with antibiotic choice rationalised on the basis of culture results 1, 6 & 15. Antibiotics should be continued for five to seven days.
- Withhold all diuretics and nephrotoxic drugs and prescribe IV albumin at a dose of 1.5 g/ kg on the day of admission and 1 g/kg on day 3 to support renal function. Albumin administration in SBP has been shown to reduce mortality through a reduction in the incidence of hepatorenal syndrome (HRS) 18.
- A repeat diagnostic ascitic tap should be performed at 48hrs to ensure adequate response (defined as a 50% fall in neutrophil count in the ascitic fluid) 1, 6 & 15. Antibiotic choice may need to be escalated if this has not occurred.
- All patients diagnosed with a first episode of SBP should receive secondary antibiotic prophylaxis on discharge (Bactrim 800/160mg OD (monitor renal function); norfloxacin 400mg OD) for life as this reduces recurrent episodes and mortality 1, 15 & 19.
- Once SBP has been successfully treated, management of ascites should be as detailed below. Definitive management of ascites with large volume abdominal paracentesis and/ or commencement of diuretics is rarely required before day five of hospital admission in patients with SBP, as there is the very real risk of causing/ exacerbating renal dysfunction and/ or precipitating HRS 1, 6 & 15.

**General management of ascites**

Management of ascites in those without SBP/ once SBP adequately treated1, 15:

- Review medications and cease anything that may be contributing to ascites formation. NSAIDs, ACE inhibitors, alpha-blockers and aminoglycosides should all be avoided.
- Assess fluid status, renal function and commence daily weights. Unless the patient has heart failure there is no requirement to fluid restrict. In ascites secondary to liver disease the aim is to cause a naturesis, not a diuresis.
- Sodium restriction (2000mg/day)/ salt-to-tolerance diet.
- Involve dietician. Patients with chronic liver disease almost universally have sarcopenia and protein energy malnutrition and will benefit from their input.
- If no renal impairment or hyponatraemia, commence an aldosterone antagonist. Spironolactone 50mg od can be commenced and up titrated in 50mg increments every 3-4 days (no more than two increments per week) if renal function and potassium allow. Frusemide is added to prevent hyperkalaemia and allow further up titration of spironolactone at a ratio of 40mg frusemide: 100mg spironolactone. Up to 400mg spironolactone can be given daily; however, it is rare to need to go above 200mg daily.
- Target weight loss should be 0.5kg/d in those with ascites only and 1 kg/d in those with ascites and significant peripheral oedema. Higher weight loss targets risk precipitating renal dysfunction and further decompensation.

- Large volume abdominal paracentesis should be should be performed if ascites is tense, causing respiratory compromise or where diuretic therapy cannot be used. It is imperative to carefully assess intravascular volume beforehand and to counterbalance fluid shifts and the potential for renal dysfunction/ precipitation of HRS with 100ml 20% albumin for every 2.5 litres of fluid drained after the first 5 litres. Drains should be left in for no longer than 6h and no more than 12 litres of fluid should be drained in one sitting. Nursing staff should be instructed to remove the drain when either one of these end points is reached.
- Ensure renal function is monitored daily when up-titrating diuretics and/ or performing large volume abdominal paracentesis.

## Hepatorenal Syndrome

HRS is functional renal failure caused by intra-renal vasoconstriction in patients with end-stage liver disease and circulatory dysfunction. Circulatory dysfunction is caused by splanchnic vasodilation and insufficient cardiac output leading to effective hypovolaemia. HRS occurs spontaneously with deteriorating liver function or secondary to a precipitating event such as bacterial infection.

Ascites is the first part of a spectrum of renal impairment severity in cirrhosis. The onset of HRS is a very poor prognostic sign. Type 1 HRS, which is associated with a rapid deterioration in renal function, has a very poor short term prognosis. Type 2 HRS is associated with a slower deterioration and a poor medium term prognosis. With type 2 HRS the presentation acutely is usually with ascites and mild to moderate renal impairment.

## Pathophysiology

The initial event is splanchnic arterial vasodilation, which causes effective hypovolemia. This is compensated for by an increased cardiac output (hyper dynamic circulation). However, as the disease progresses, splanchnic arterial vasodilation increases and cardiac output decreases, leading to deterioration of circulatory function and stimulation of the renin-angiotensin-aldosterone system (RAAS), sympathetic nervous system, and antidiuretic hormone. Activation of the RAAS represents a homeostatic response to counterbalance the vasodilation, arterial hypotension and renal hypoperfusion observed in portal hypertension. When circulatory dysfunction is moderate, this leads patients to develop sodium retention. When it is severe, patients develop a profound impairment in free water excretion and dilutional hyponatremia. Finally, when it is extreme, patients present with HRS. This explains why medications which affect renal perfusion (ACE inhibitors & NSAIDs through inhibition ACE and prostaglandin synthesis respectively) can contribute to ascites formation and/or precipitate precarious renal injury in cirrhotic patients.

## Diagnosis of HRS

HRS is a diagnosis of exclusion in cirrhotic patients with renal dysfunction. All five of the following features need to be present 20:

1. Ascites secondary to cirrhosis
2. AKI according to the ICA-AKI criteria
3. Absence of known causes of renal failure (e.g. shock, ongoing UTI/sepsis, nephrotoxic drugs, diuretics) or renal calculi/obstruction
4. Proteinuria <0.5g/d, no active urinary sediment and normal renal US
5. No sustained improvement of renal function at 48 hours after withdrawal of nephrotoxic medications, albumin support (1.0 g/kg per day) and fluid resuscitation.

## Management

Management is difficult. In patients with type I HRS who are not candidates for transplant, the diagnosis of HRS is dire and likely to be a terminal event and if not already addressed, ceilings of care should be discussed with the patient and/or family. The diagnosis of type II HRS should also be a prompt for advanced care planning in those patients not a candidate for transplant.

Basic management involves an assessment of renal perfusion. Is this patient shocked? All anti-hypertensives and diuretic medications should be ceased. Ensure the patient has not been prescribed NSAIDs or other nephrotoxic medications. A careful assessment of intravascular volume needs to be made. If the patient is obviously intravascularly depleted prescribe one to two 500ml bottles of 4% albumin solution to expand the plasma volume. Ongoing maintenance IV fluids, if required, should be salt poor if the patient's serum sodium allows. 100ml 20% albumin at a dose of 1g/kg/day should be prescribed to support the patient's intravascular volume. Daily weights and a urinary catheter should be placed to allow accurate recording of the patient's fluid balance. These patients are at high risk of acute pulmonary oedema (APO) and need ongoing regular fluid balance assessments and consideration given to performing 12h U+E. An extensive septic screen should be sent, urine sent for active sediment and PCR and a renal US requested in order to exclude precipitating events/ establish another cause. If the patient is a potential transplant candidate, early discussion with ICU for consideration of vasopressor support (terlipressin is first choice having been shown to be superior to noradrenaline) is indicated 21.

### Hepatic encephalopathy

HE is brain dysfunction secondary to liver insufficiency and/ or primary sclerosing cholangitis. It manifests as a wide spectrum of neurological and psychological abnormalities, ranging from subclinical alterations to coma. Overt HE is present in approximately 10% of patients with cirrhosis at diagnosis and will appear in 40% at some point. It often recurs 22. Very mild HE is hard to detect clinically at the bedside but is present in most patients when complexed psychometric tests orientated at attention and visuospatial awareness are performed. Overt HE is much more obvious with personality changes, sleep-wake reversal, motor system abnormalities and extra-pyramidal symptoms, asterixis, stupor and coma. Precipitating factors are always present when hepatic HE is secondary to cirrhosis: look, treat and prevent!

### Grading of hepatic encephalopathy

HE is graded according to the West-Haven classification system:

- Grade 0- Lack of clinically detectable changes in behaviour or mental status.
- Grade 1- Mild confusion, slowing of ability to perform mental tasks.
- Grade 2- Lethargy, disorientation, inappropriate behaviour.
- Grade 3- Somnolent but can be aroused, marked confusion, incomprehensible speech
- Grade 4- Coma

### Precipitants

There is a wide differential diagnosis which must be considered and treated if present. Infection is a common precipitant and often under recognised, as HE may be the only manifestation of sepsis. A thorough septic screen should be performed in all patients (CXR, urine, blood cultures, sputum cultures, abdominal US, diagnostic abdominal paracentesis if ascites, thorough skin check). GI bleeding, constipation, diuretic overuse, drugs (opiates, benzodiazepines), alcohol and electrolyte abnormalities are the other common precipitants which should be looked for.

It is important to always consider the possibility of a subdural haematoma as a potential cause for a deterioration in a cirrhotic patient's mental status, particularly if they have risk factors (e.g. alcoholic liver disease with a history of ongoing alcohol consumption and falls). Have a low threshold for ordering a CT brain.

**Management**

Unconscious patients require protection of their airway and early ICU involvement. All potential causes should be sought and treated. Once a full septic screen has been performed it is reasonable to give a stat dose of a broad spectrum antibiotic, such as a 3rd generation cephalosporin, pending culture results. This can be deescalated if needed once culture results are available.

In the interim, empirical treatment for HE should be commenced 1 & 22.

- Lactulose 30ml PO QID aiming for 2-3 soft bowel motions per day
- If nil by mouth, 300ml lactulose PR QID is an alternative
- If ongoing HE after 24-48h consider starting rifaximin 550mg PO BD23
- Nutritional support and early involvement of the dietician

**Malnutrition, sarcopenia and nutritional deficiencies**

Malnutrition has been reported to occur in 20% patients with compensated cirrhosis and in greater than 50% of those with decompensated liver disease 24. Malnutrition and sarcopenia (muscle mass loss) are associated with a higher rate of complications such as infections, HE and ascites, and have been shown to be independent predictors of mortality 24. Given these observations, malnutrition and sarcopenia should be recognised as complications of cirrhosis, which in turn lead to worse outcomes for patients with cirrhosis 24. Therefore all patients with decompensated liver disease should see a dietitian for a detailed assessment of nutrition, including obese patients, within 24h of admission to hospital 1, 24. All patients with a history of alcohol excess and patients at risk of refeeding, should receive high dose IV thiamine and multivitamins, ideally as combined formulations such as Pabrinex (vials I & II IV TDS), or, if not available, high dose IV thiamine (300mg IV TDS) and Cernavit (one vial IV OD). Daily monitoring of electrolytes should be undertaken, with particular attention paid to serum calcium, magnesium and phosphate, with aggressive IV replacement prescribed if required. In those patients with decompensated cirrhosis not meeting their daily nutritional requirements following review and intervention, early consideration in consultation with the dietician should be given to supplementary NG feeding.

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