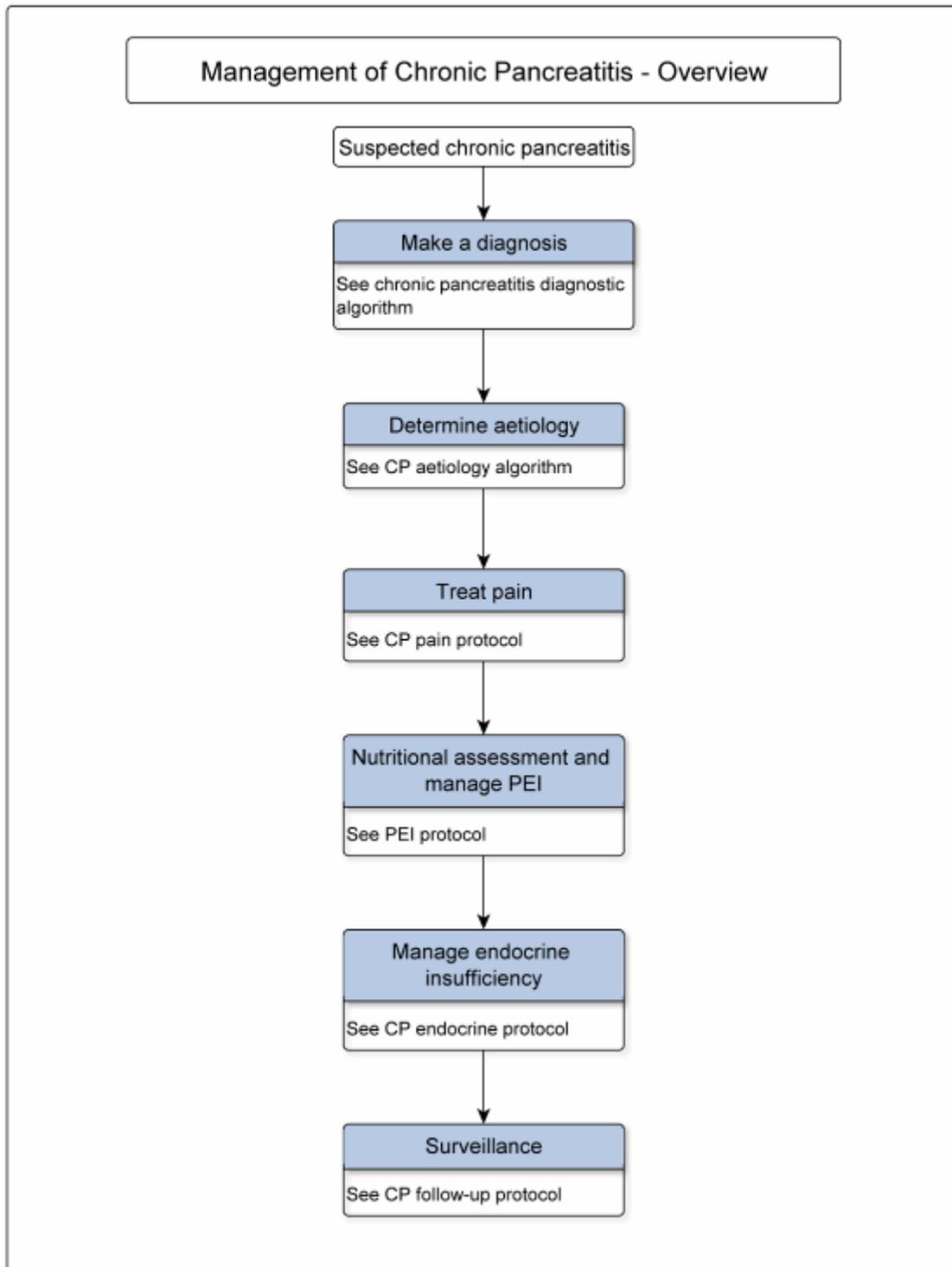

**Greater Manchester and Cheshire HPB Unit
Guidelines for the Assessment &
Management of Hepatobiliary and
Pancreatic Disease
Chapter 11**

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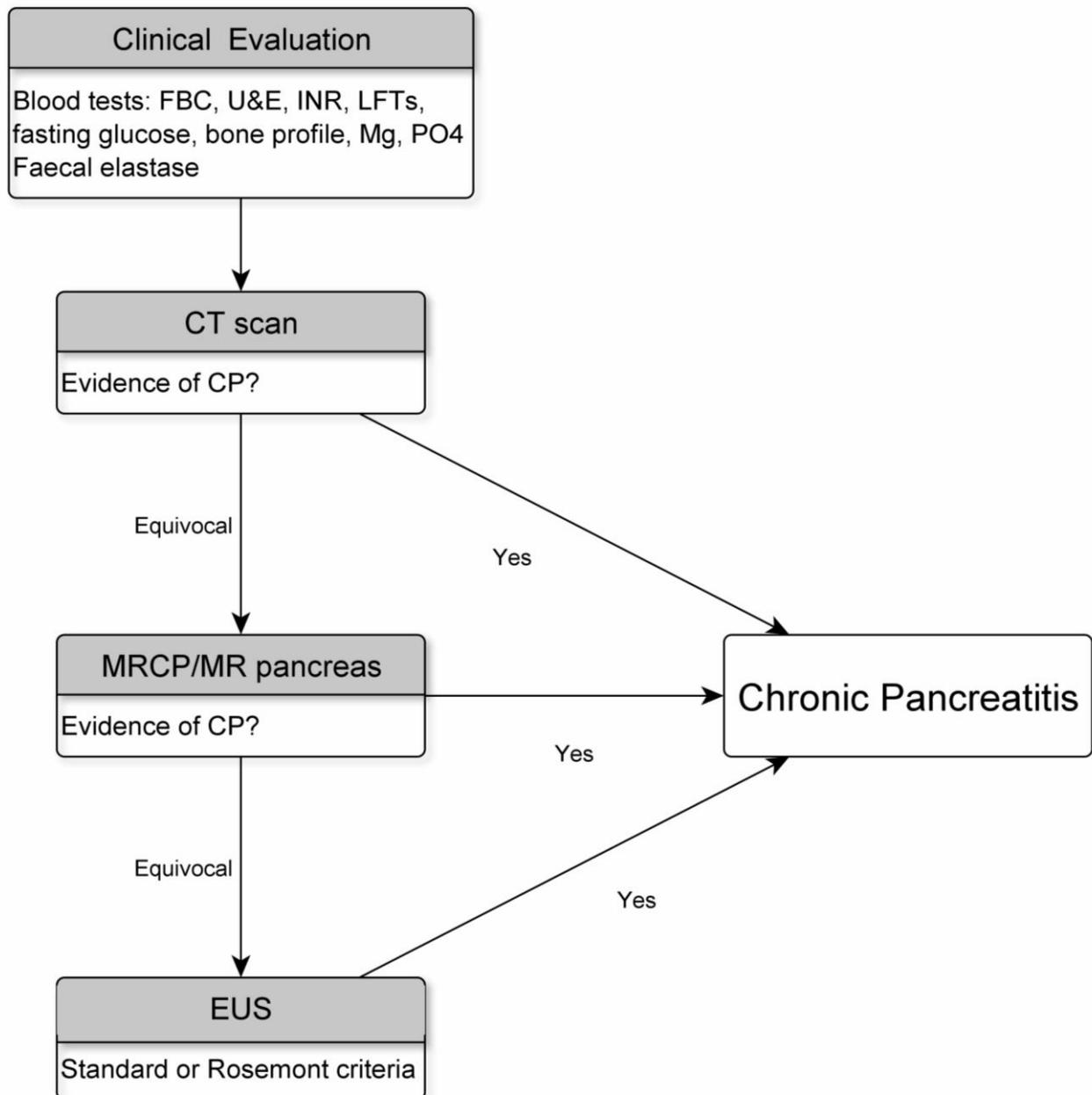
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11. Chronic Pancreatitis

11.1. *Overview of management*



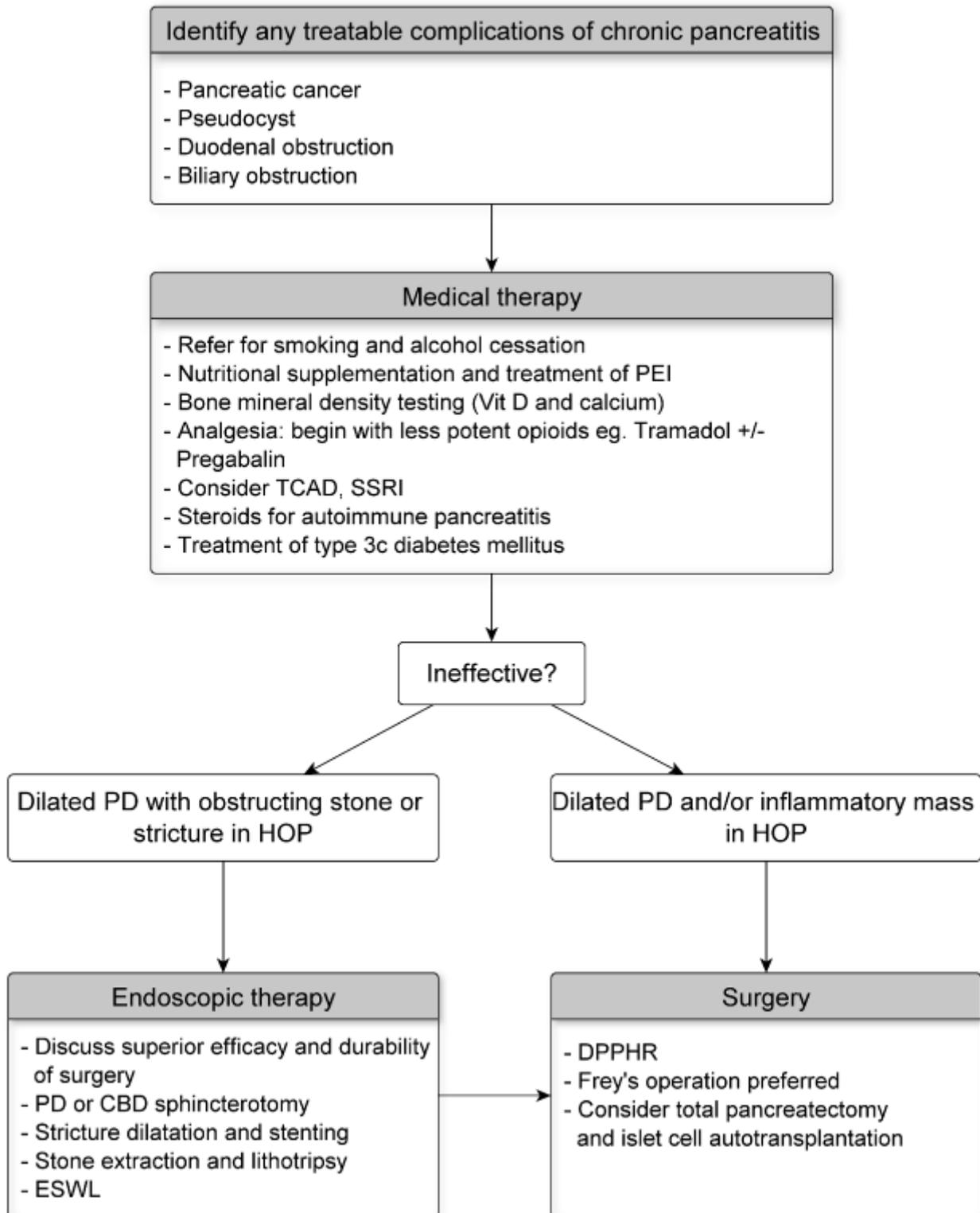
11.2. Diagnostic algorithm for chronic pancreatitis



11.3. *Aetiological Classification of Chronic Pancreatitis (TIGAR-O system)*

T	Toxic - Metabolic	Alcohol, Smoking
I	Idiopathic	Including tropical CP
G	Genetic	PRSSI, SPINK-1, CFTR (Refer to EUROPAC)
A	Autoimmune	Type 1, 2 (See AIP algorithms)
R	Recurrent acute severe pancreatitis	Post-necrotic acute pancreatitis
O	Obstructive	Duct obstruction (tumours), SOD, Pancreas divisum

11.4. *Treatment algorithm for chronic pancreatitis*



11.5. *Nutritional Assessment, Treatment of PEI and Type 3c DM*

Suspect pancreatic endocrine insufficiency [PEI] in

- Acute pancreatitis with extensive necrosis
- Chronic pancreatitis
- Pancreatic cancer with obstruction of the pancreatic duct

Assessment of PEI

- History and clinical evaluation
- Measure weight and BMI
- Suspect PEI if clinical features of malabsorption:
 - o diarrhoea
 - o steatorrhoea
 - o weight loss
 - o metabolic bone disease
 - o vitamin or mineral deficiency
- Blood tests: FBC, U&E, LFTs, INR, Calcium, Phosphate, Magnesium, Vitamin D
- Faecal elastase
- Baseline bone mineral density testing

Management of PEI

- Creon 40,000 units, 2 capsules with meals and 1 capsule with snacks
- Add PPI if continued symptoms of malabsorption
- Vitamin D and calcium supplementation
- Consider dietician referral and nutritional supplementation

Type 3c DM

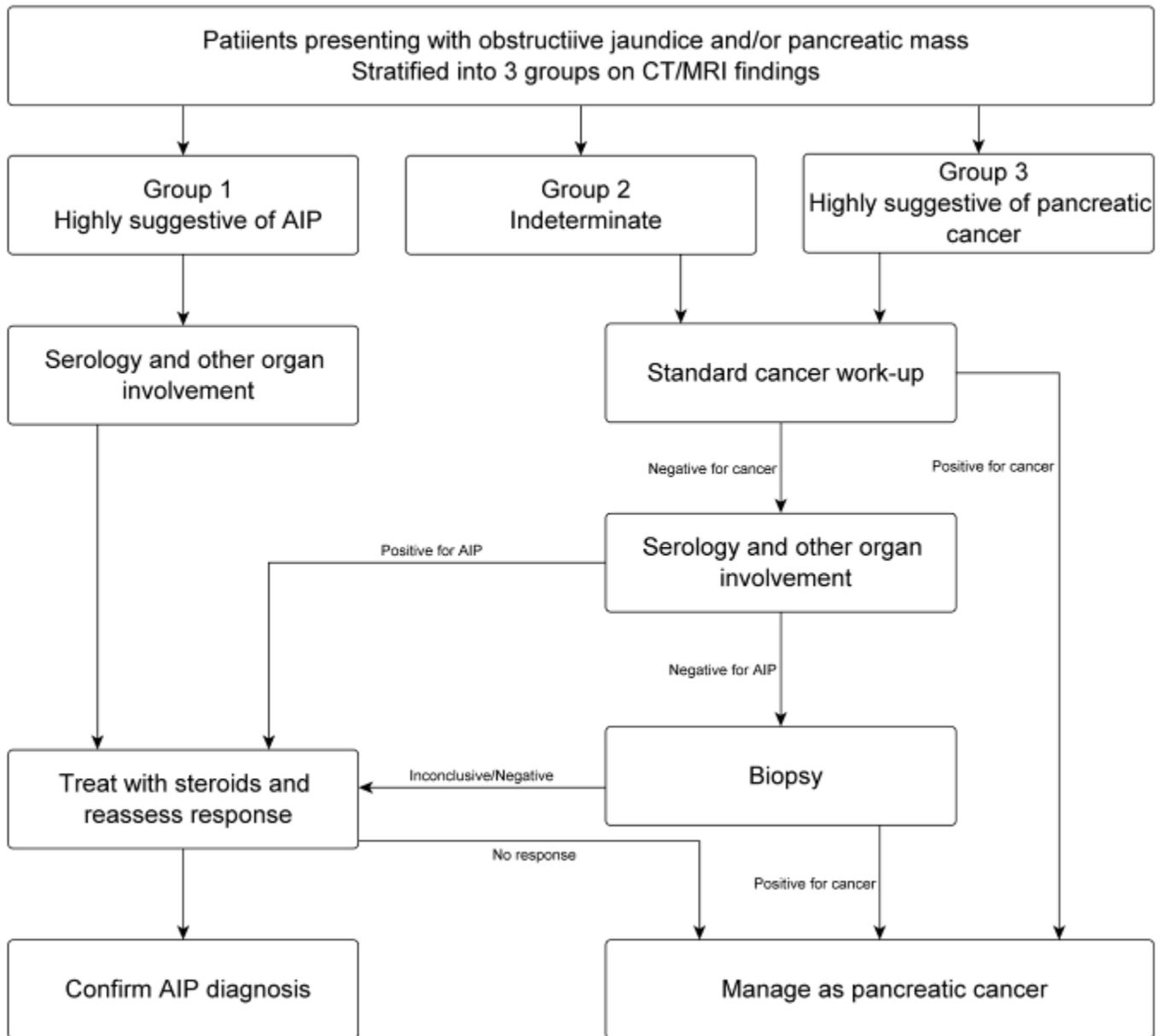
- Measure fasting glucose and HbA1c (baseline and annual)
- Glucose tolerance test for equivocal results
- Refer to specialist diabetologist
- Metformin may reduce the long-term risk of cancer

11.6. *Autoimmune Pancreatitis – Principles of Diagnosis*

Principles of diagnosis when distinguishing autoimmune pancreatitis [AIP] from pancreatic cancer

1. Clinical presentations not suggestive of AIP include: marked cachexia, anorexia and severe pain requiring opiates.
2. A thorough negative work up for other aetiologies should be undertaken, in particular for pancreatic or biliary cancer.
3. Histological diagnosis of AIP requires preservation of tissue architecture (showing lymphoplasmacytic infiltrate with >10 IgG4 positive cells/High Power Field), which renders FNA less helpful for diagnosis.
4. Steroid therapy should only be commenced when other aetiologies for pancreatic disease have been excluded, and only in those patients whose response may be adequately assessed. It should not be used as a substitute for a thorough search for the aetiology.
5. Objective improvement in the appearance of the pancreas on cross-sectional imaging should be evident within 2 weeks of steroid use. Subjective improvement in symptoms or even a decline in serum IgG4 levels can occur in pancreatic cancer or lymphoma and should not be used as response criteria.
6. In AIP, CA 19-9 levels drop with treatment; a rising CA 19-9 suggests this diagnosis is incorrect.
7. The diagnosis of AIP is difficult. Use of the agreed diagnostic pathway and a multidisciplinary approach should be taken with each patient, to ensure that pancreatic cancer patients are not treated with steroids and, conversely, AIP patients not treated with cancer surgery.

Strategy for distinguishing AIP from pancreatic cancer (based on the Mayo Clinic strategy)



11.7. Autoimmune Pancreatitis – Treatment Algorithm

