EMA Emergency Medicine Australasia

Emergency Medicine Australasia (2024) **36**, 505–511 doi: 10.1111/1742-6723.14421

REVIEW ARTICLE

Review article: Abdominal pain and diabetes mellitus in the emergency department

Sneha KRISHNA, Alex PRINS and Adam MORTON

Mater Health, Brisbane, Queensland, Australia

Abstract

This manuscript seeks to describe diagnostic considerations in individuals with diabetes mellitus presenting to the ED with abdominal pain. It highlights the importance of early investigation with computerised tomography to differentiate aetiologies that compel early surgical intervention from those which may be treated conservatively.

Key words: *abdominal pain, diabetes mellitus, computerised tomography.*

Introduction

Abdominal and pelvic pain accounted for 4.7% of ED presentations in Australia in 2022–2023.[1] Abdominal pain is the most frequent reason for return visits to ED.[2] Abdominal pain may be caused by a wide variety of pathologies of non-gastrointestinal pathology.[3] A questionnaire survey of 8657 individuals living in New South Wales found prevalence rates of abdominal pain or discomfort in the preceding 3 months of 13% in individuals with diabetes mellitus (DM), compared with 10.8% in control subjects (adjusted odds ratio = 1.63).[4]

The prevalence of type 2 diabetes mellitus (T2DM) in Australia increased from 1985 to 3429 per 100 000 population between 1990 and 2019. Incidence rates of type 1 diabetes mellitus (T1DM) remained stable between 2000 and 2018.

This review seeks to address:

1. Conditions related to DM associated with clinical signs of an acute abdomen, but where surgical intervention is not required and/or contraindicated.
2. Abdominal conditions necessitating expeditious surgical intervention because of the significantly higher morbidity and mortality in individuals with DM, compared with individuals without DM.
3. Investigations into abdominal pain may be misleading in individuals with DM (Table 1).
4. Presentations where the opportunity to make an early diagnosis may lead to a significant improvement in patient outcome.
5. To propose an investigative flowchart for the assessment of abdominal pain in DM.

The review highlights the importance of early investigation to define the indications for immediate therapy and/or computerised tomography (CT), and further differentiate aetiologies that compel early surgical intervention from those that may be managed conservatively.

Methods

A literature search was conducted using Medline (OVID) and Google Scholar from their inception until July 2023 using MeSH search terms 'abdominal pain' and 'diabetes mellitus' without language and location limitation. Once duplicates were eliminated, two authors (SN and AM) individually assessed the titles and abstracts for suitability. We excluded studies with unavailable texts and articles on other subjects. Full text of relevant articles was obtained, and reference lists were screened for additional relevant articles. In total, 413 articles were included, and 1605 articles were excluded.

Key findings

- A major challenge in evaluating abdominal pain in individuals with diabetes mellitus is differentiating those with non-surgical pathology from those causes which mandate urgent surgical intervention.
- Clinical signs, laboratory changes, and electrocardiography may be misleading in individuals with diabetes mellitus and abdominal pain.
- Early computerised axial tomography imaging may be useful in differentiating aetiologies of abdominal pain in individuals with diabetes mellitus presenting to the Emergency Department.

Diabetic ketoacidosis

DKA is associated with acute epigastric pain in 48% of adults and 62% of children, and may mimic an acute abdomen, so-called diabetic pseudoperitonitis.[5,6] Epigastric pain may persist for days after correction of metabolic acidosis, suggesting peritoneal irritation by ketone bodies may be the cause of pain.[7] Severe abdominal pain with signs of acute peritonitis leading to laparotomy has also been described with sodium-

Correspondence: Dr Adam Morton, Mater Health, Raymond Terrace, Brisbane, QLD 4101, Australia. Email: adam.morton@mater.org.au

Sneha Krishna, MBBS, Endocrinology Advanced Trainee; Alex Prins, FACEM, Staff Specialist Emergency Medicine; Adam Morton, FRACP, Senior Staff Specialist Endocrinology and Obstetric Medicine.

Accepted 10 April 2024

TABLE 1. *Potentially misleading investigations with abdominal pain in diabetes mellitus*

Investigation	Conditions
Elevated lipase in absence of acute pancreatitis	Diabetic ketoacidosis Medications – DPP4i, GLP1 agonists Glycogenic hepatopathy Uncomplicated diabetes mellitus
Normal lipase despite acute pancreatitis	Hypertriglyceridaemia
ECG abnormalities and elevated troponin without myocardial ischaemia	Diabetic ketoacidosis Acute pancreatitis
Lactic acidosis in absence of mesenteric ischaemia or sepsis	Glycogenic hepatopathy Metformin – usually with renal dysfunction

DPP4i, dipeptidyl peptidase 4 inhibitor; GLPA, glucagon-like peptide-1.

glucose transporter protein 2 inhibitor-euglycemic ketoacidosis.[8] In one study of adults with DKA and abdominal pain, 17% of individuals had underlying abdominal pathology, predominantly acute pancreatitis (AP), which is estimated to occur in 10–15% of cases of DKA.[9] Other abdominal pathologies most commonly associated with DKA include cholecystitis, pyelonephritis, appendicitis, mesenteric ischaemia and ischaemic liver injury, although any intra-abdominal pathology may be a precipitant. CT/CT angiography should be considered in any individual with DKA where the former diagnoses are considered, or where the individual is not improving clinically with treatment of DKA. Hypoxic/ischaemic liver injury should be considered in the setting of abdominal pain and markedly elevated aminotransferases with DKA, although this needs to be differentiated from glycogenic hepatopathy (GH).[10]

Acute mesenteric ischaemia

Occlusive and non-occlusive acute mesenteric ischaemia (AMI) have been reported in children, adolescents and adults with DKA and hyperosmolar hyperglycaemic state (HHS).[11,12] The adjusted hazard ratio of DM for AMI was 1.32 in one study.[13] AMI should be suspected where there is ongoing abdominal pain, peritonism, elevated lactate, or failure to correct high anion-gap acidosis with treatment of DKA or HHS.

Acute pancreatitis

A systematic review and meta-analysis of cohort studies found that individuals with DM had a 74% higher risk of AP than individuals who did not have DM.[14] The revised Atlanta classification requires that two or more of the following criteria be met for the diagnosis of AP: (i) abdominal pain suggestive of pancreatitis; (ii) serum amylase or lipase level greater than three times the upper normal value (>3*ULN); or (iii) characteristic imaging findings. Elevation of serum lipase with asymptomatic DM, DKA, fatty pancreas and medications, as well as *in vitro* depression of serum lipase levels with hypertriglyceridemia, complicate the biochemical diagnostic criteria for AP in DM, resulting in greater reliance on imaging. Studies of adult patients presenting with DKA found lipase values >3*ULN in 16–25%, with changes consistent with AP on imaging in 41–52.9% of these patients.[15] Serum lipase >3*ULN was associated with a sensitivity of 81%, specificity of 91% and positive predictive value of 52% for a definite diagnosis of AP. Paediatric DKA was associated with elevated lipase in 31–38%, serum lipase >3*ULN in 13% and AP in 0–2%. Elevation of serum lipase was present in 11.4–20.4% of asymptomatic individuals with T2DM as part of baseline screening for a drug trial, with 0.4–2.1% having a serum lipase >3*ULN.

Dipeptidyl peptidase 4 inhibitors (DPP4i) and glucagon-like peptide-1 receptor agonists (GLP1RAs) are associated with the elevation of serum lipase in 39% and 25–51% of asymptomatic T2DM, respectively.[16] Lipase levels may be increased >7*ULN with DPP4i therapy, and 1.5–8.3% of individuals receiving GLP1RA develop a serum lipase >3*ULN, in the absence of AP.

Hypertriglyceridaemia (HTG) has been reported as an aetiological factor in 0.97–25.3% of cases of AP, with the highest rates in those of Chinese ethnicity.[17] Individuals with T1DM and T2DM are at increased risk of HTG as the enzyme responsible for breakdown of triglycerides, lipoprotein lipase, is insulin dependent. A Chinese study found that 20.6% of patients presenting with HTG-AP had pre-existing DM.[18] Patients with HTG-AP may have normal lipase and amylase levels because of HTG interfering with the laboratory assay. A study of 11 patients with AP associated with DKA found that four (36%) patients had significant HTG, two of whom had normal serum lipase levels.[15]

As a result of the multiple factors that may cause elevation of serum lipase >3*ULN in the absence of pancreatitis in individuals with DM, and the potential for spurious *in vitro* lowering of lipase with HTG, computed tomography may

have a greater role in diagnosing AP in DM.

Pancreatic cancer

The most common presenting symptoms of pancreatic cancer are weight loss (85%), epigastric pain (79%) and jaundice (56%). A bidirectional relationship exists between pancreatic cancer and DM.[19] Eighty per cent of pancreatic cancer patients have a diagnosis of either hyperglycaemia or T2DM, and patients with new-onset DM are at up to eight times higher likelihood of developing pancreatic cancer within 3 years of the diagnosis of DM.[20] Among individuals with new-onset DM, the highest risk of pancreatic cancer occurred in those with a history of pancreatitis, gallstones, weight loss, a family history of pancreatic cancer and severe/rapidly increasing glycaemia requiring early use of insulin.[20] The development of DM in the setting of pancreatic cancer may be because of a paraneoplastic effect rather than islet cell destruction, given that DM often develops at an early resectable stage of disease.[21] Pancreatic imaging with CT should be performed in any individual presenting with newly diagnosed T2DM with significant weight loss, epigastric pain, previous pancreatitis or rapid requirement for insulin therapy.

Addison's disease

The prevalence of Addison's disease in individuals with T1DM is 0.2%, 200-fold higher than that in the general population.[22] Up to 14% of patients with Addison's disease have associated T1DM. Epigastric pain and tenderness, vomiting, diarrhoea and signs of an acute abdomen may occur with impending Addisonian crisis, and have led to unnecessary surgery.[23,24] Other clinical features of Addison's disease in the individual with T1DM include frequent hypoglycaemia with reduction in insulin requirements, weight loss, fatigue, hypotension, hyperpigmentation, salt craving, hyponatraemia and hyperkalaemia. In any individual with T1DM presenting with otherwise unexplained abdominal pain and features listed above urgent measurement of serum cortisol should be performed, and parenteral glucocorticoid therapy administered pending the results.

Inferior myocardial ischaemia/infarction

Individuals with T2DM have a two- to fourfold increase in coronary heart disease, cardiac death and non-fatal MI. The risk of coronary heart disease is even greater in T1DM. Inferior MI may present with epigastric pain, nausea and vomiting.[25] Cannabinoid hyperemesis syndrome (CHS) has been reported to be complicated by MI secondary to coronary vasospasm.[26] DKA and AP may each be associated with ECG abnormalities mimicking MI and elevated cardiac troponin values, with no evidence of MI on echocardiography and normal coronary angiography.[27–30]

Glycogenic hepatopathy

GH is an uncommon complication of poorly controlled T1DM. Individuals present with right upper quadrant pain, nausea, massive hepatomegaly, markedly elevated transaminases and elevated lactate levels.[31] Serum lipase may be elevated more than 6*ULN in the absence of acute pancreatitis. The presence of lactic acidosis may cause consideration of mesenteric ischaemia or sepsis. Except when presenting with superimposed DKA, individuals with GH are haemodynamically stable and do not appear unwell.

Cannabinoid hyperemesis syndrome

CHS is characterised by cyclical episodes of nausea, vomiting and abdominal pain. The prevalence of past-30-day cannabis use among individuals with DM in the United States increased from 1.7% to 5.8% between 2005 and 2018. Cannabis use in T1DM is associated with a twofold increase in the risk of DKA and poor glycaemic control.[32] Diagnosis of CHS in T1DM is often delayed, with a minimum of 12 hospital admissions over 2–13 years from the first presentation before the diagnosis was made, with individuals having undergone multiple imaging and endoscopy studies during this time.[33] Several diagnostic algorithms have been proposed for the diagnosis of CHS, including the Rome IV criteria. Assessment of chronicity and recent use of cannabis is hampered by positive results for tetrahydrocannabinol on urine testing up to 6 weeks after last use.

Diabetic gastroparesis

A study of 134 individuals with diabetic gastroparesis found that 89% reported abdominal pain, with pain being severe in 34% of cases.[34] Abdominal pain was aggravated by eating in 51%, occurred at night in 51%, and interfered with sleep in 37%. Acute episodes of abdominal pain were reported by 72% of individuals. Severity of pain was not related to the degree of delayed gastric emptying for solids or liquids on scintigraphy. Factors associated with excruciating abdominal pain included female sex, younger age, use of narcotic pain medication, acute *versus* insidious onset and increasing gastroparesis cardinal symptom index. Four studies found the use of haloperidol reduced rates of hospital admission, amount of opioid administered, rates of rescue therapy, and mean pain and nausea scores in individuals presenting to an ED with symptomatic diabetic gastroparesis, non-specific abdominal pain, or undifferentiated intractable vomiting.[35] In patients with gastroparesis presenting to the ED, droperidol reduced opioid use, improved pain control and decreased antiemetic use without any change in length of stay or hospital admission rate.[36]

Coeliac disease

The prevalence of celiac disease in patients with T1DM is approximately 3–8%, significantly higher than in the general population (1%). Undiagnosed coeliac disease is an important cause of recurrent abdominal pain in children, adolescents and adults.[37–40] A study of children and adolescents presenting with

recurrent abdominal pain found coeliac disease to be the underlying cause in 10% of individuals.[37] A case–control study found that adult coeliac disease accounted for 3% of adults who presented to a University hospital with unselected acute abdominal pain.[38] Elevated hepatic transaminases occur in 4–9% of individuals with coeliac disease.

Thoracic or lumbar polyradiculopathy

Diabetic neuropathy may affect intercostal nerve roots resulting in severe, constant, chronic, band-like upper or lower abdominal pain.[41] Pain is typically unilateral, described as burning or aching, worse at night and associated with dysaesthesia. The anterior abdomen is always involved, with pain in the corresponding back region in 60% of cases. Abdominal pain may be associated with significant unexplained weight loss suggestive of underlying malignancy. Occasionally, radiculopathy may be accompanied by diabetic amyotrophy with severe thigh pain, weakness and muscle atrophy. Physical examination may reveal unilateral abdominal wall paresis and protrusion and hypoaesthesia or hyperaesthesia in multiple adjacent dermatomes. Diagnosis is made by paraspinal electromyography. Imaging must be performed to exclude spinal compression or intrabdominal malignancy.

Emphysematous cholecystitis

Emphysematous cholecystitis (EC) is caused by secondary infection of the gallbladder wall with gas-forming organisms such as Clostridia, predisposing to gangrene and perforation. The mortality rate with EC is 15%.[42] It typically affects men in their fifth to seventh decade, and 30–50% have DM. EC usually results from thrombosis or occlusion of the cystic artery with ischaemic necrosis of the gallbladder wall. Initial presentation is often indistinguishable from uncomplicated acute cholecystitis. Clues to the diagnosis include mild to moderate unconjugated hyperbilirubinemia because of clostridial-induced haemolysis, and rarely crepitus in the abdominal wall. Prompt diagnosis is essential as early cholecystectomy may reduce morbidity and mortality. CT is the most sensitive modality for the detection of intraluminal or intramural gallbladder gas.[43] US may be misleading because of air in the gallbladder wall being erroneously attributed to overlying bowel gas.

Emphysematous pyelonephritis

Emphysematous pyelonephritis is a gas-producing, necrotizing infection involving the renal parenchyma and occasionally perirenal tissue. It is associated with a mortality rate of 12.5%.[44] Major risk factors include DM (80–96% of cases) and urinary tract obstruction (22%). Clinical features are indistinguishable from acute pyelonephritis, and diagnosis is best made with CT showing air in the collecting system, renal parenchyma and/or surrounding tissues. Management requires relief of obstruction if present, drainage of gas and/or purulent material, empiric antibiotics, supportive care for septic shock, acute kidney injury and disseminated

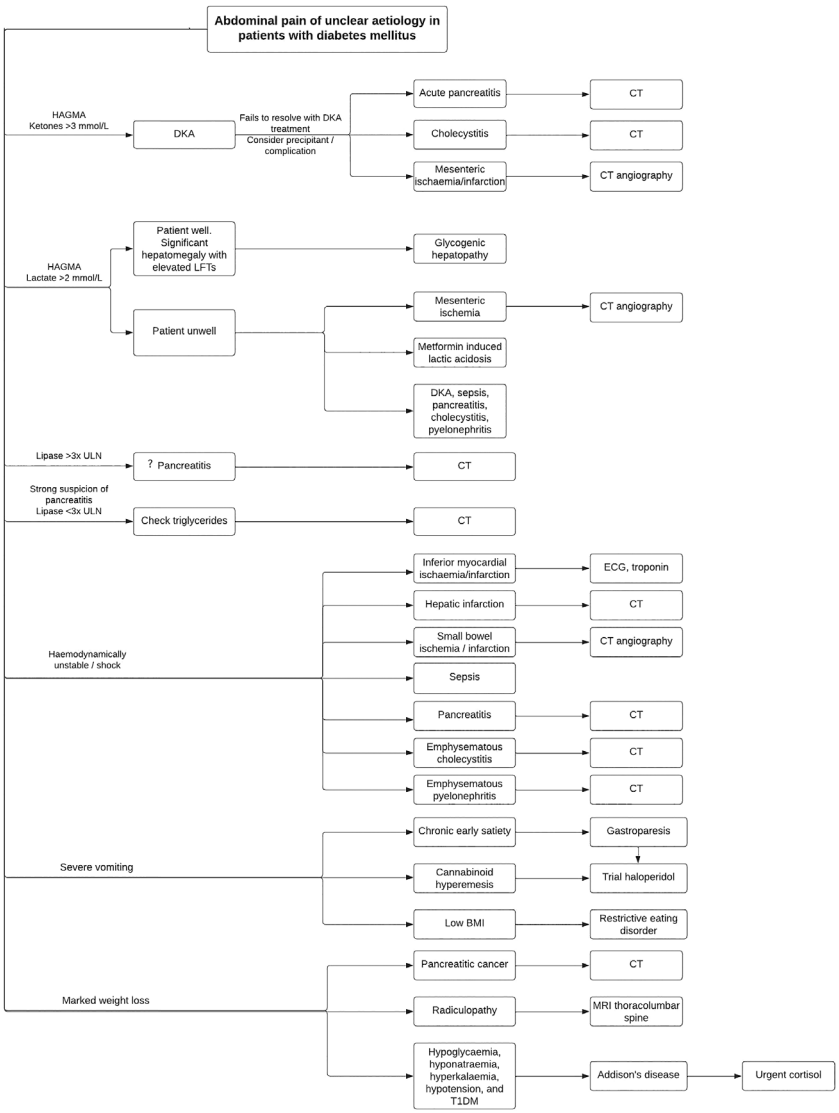

Figure 1. *Flowchart regarding differential diagnosis of abdominal pain in diabetes mellitus.*

intravascular coagulation where present and occasionally nephrectomy. The most common organisms are *Escherichia coli* and *Klebsiella pneumoniae*. Initial antibiotic therapy should be with an antipseudomonal carbapenem and vancomycin in critically ill patients or those with obstruction. In less unwell individuals, ceftriaxone or piperacillin/tazobactam is a reasonable initial choice.

Liver abscess

The risk of bacterial liver abscess in DM is approximately 3.6–11 times higher than in individuals without DM.[45] Rates are highest in individuals from Asian countries. The diagnosis should be considered where there is unexplained right upper abdominal pain, rigors and fever. Individuals with poorly controlled DM are prone to emphysematous liver abscesses, which are associated with a mortality rate of 27–30% because of the risk of septic shock and abscess rupture.

Post-bariatric surgery

Two-thirds of Australian adults are overweight or obese. From 2005–2006 to 2014–2015, the number of bariatric surgery separations in Australia increased from 9300 to 22 700.[46] T2DM was recorded as an additional diagnosis in 16.7% of cases.[46] Complications of bariatric surgery presenting with abdominal pain include internal hernia, small bowel obstruction, intussusception and cholelithiasis. A high index of suspicion is required because of the non-specific nature of presentation. This is particularly important during pregnancy, where symptoms may be attributed to physiological gestational pain. A systematic review of 120 pregnant women who required emergency abdominal surgery for complications related to previous bariatric surgery reported maternal death in three (2.5%) cases, and foetal death in nine cases (7.5%).[47] A low threshold for imaging with point-of-care US, MRI or CT should be employed in pregnant women with abdominal pain where there is a previous history of bariatric surgery.

Medications

Metformin therapy is associated with a relative risk for abdominal pain of 1.5 compared with placebo, along with increased risks of nausea, vomiting and diarrhoea.[48] Metformin-associated lactic acidosis (MALA) is characterised by abdominal pain, vomiting, hypothermia, hypotensive circulatory failure and oliguric acute kidney injury (AKI). Abdominal examination may reveal diffuse guarding and absent bowel sounds prompting laparotomy. Biochemistry reveals severe high anion-gap acidosis, elevated lactate and AKI. Mortality with MALA has been reported to be as high as 50%.[49] Abdominal pain occurs in 1–10%, and nausea and diarrhoea in 10–20% of individuals on commencing GLP1RA.[50]

Conclusion

A major challenge in assessing abdominal pain in DM in the ED is rapidly differentiating patients who require acute intervention from patients with non-surgical conditions presenting with clinical signs and/or biochemical changes suggestive of surgical pathology. A proposed diagnostic pathway is presented in Figure 1. The difficulties in differentiating disorders on clinical signs and biochemical criteria highlight the value of early CT in clarifying diagnosis. Many case reports have described unnecessary laparoscopy or laparotomy in individuals with pseudo peritonism in the setting of uncomplicated DKA, Addison's disease or radiculopathy, or because of lactic acidosis and marked transaminitis with GH. Conversely, early surgical intervention is critical in EC/pyelonephritis and mesenteric ischaemia, and urgent glucocorticoid replacement is required in Addison's disease to avoid morbidity and mortality. CT should also be considered in individuals presenting with abdominal pain in the setting of newly/recently diagnosed DM and significant weight loss, as early identification of pancreatic cancer may improve the possibility of curative resection.

Competing interests

None declared.

Data availability statement

Data sharing is not applicable to this article as no new data were created or analysed in this study.

References

1. Australian Government. Australian Institute of Health and Welfare. Emergency Department Care; 2024. Available from URL: https://www.aihw.gov.au/reports-data/myhospitals/sectors/emergency-department-care.
2. Hooker EA, Mallow PJ, Oglesby MM. Characteristics and trends of emergency department visits in the United States (2010-2014). *J. Emerg. Med.* 2019; **56**: 344–51.
3. Murali N, El Hayek SM. Abdominal pain mimics. *Emerg. Med. Clin. North Am.* 2021; **39**: 839–50.
4. Bytzer P, Talley NJ, Leemon M, Young LJ, Jones MP, Horowitz M. Prevalence of gastrointestinal symptoms associated with diabetes mellitus: a population-based survey of 15,000 adults. *Arch. Intern. Med.* 2001; **161**: 1989–96.
5. Umpierrez G, Freire AX. Abdominal pain in patients with hyperglycemic crises. *J. Crit. Care* 2002; **17**: 63–7.
6. Xin Y, Yang M, Chen XJ, Tong YJ, Zhang LH. Clinical features at the onset of childhood type 1 diabetes mellitus in Shenyang, China. *J. Paediatr. Child Health* 2010; **46**: 171–5.
7. Csomor J, Jirkovska J, Vedralova L *et al.* Diabetic ketoacidosis with an acute abdomen as a first manifestation of type 1 diabetes mellitus. *Acta Endocrinol.* 2017; **13**: 509–11.
8. Wang Q, Wu K, Luo X *et al.* Dapagliflozin-associated euglycemic diabetic ketoacidosis presenting with severe abdominal pain mimicking acute peritonitis. *Cureus* 2022; **14**: e22229.

9. Pant N, Kadaria D, Murillo LC, Yataco JC, Headley AS, Freire AX. Abdominal pathology in patients with diabetes ketoacidosis. *Am. J. Med. Sci.* 2012; **344**: 341–4.
10. Chen M, Croxson S. Triad: diabetic ketoacidosis, elevated liver enzymes and abdominal pain – think liver infarct! *Pract. Diab. Int.* 2007; **24**: 302–3.
11. Vivino GR, Crofton NA, Mussarat S. Diabetic ketoacidosis and acute mesenteric ischemia in adults: an underreported association. *Cureus* 2022; **14**: e29053.
12. Frontino G, Di Tonno R, Castorani V *et al*. Non-occlusive mesenteric ischemia in children with diabetic ketoacidosis: case report and review of literature. *Front. Endocrinol.* 2022; **13**: 900325.
13. Chiu YW, Wu CS, Chen PC, Wei YC, Hsu LY, Wang SH. Risk of acute mesenteric ischemia in patients with diabetes: a population-based cohort study in Taiwan. *Atherosclerosis* 2020; **296**: 18–24.
14. Aune D, Mahamat-Saleh Y, Norat T, Riboli E. Diabetes mellitus and the risk of pancreatitis: a systematic review and meta-analysis of cohort studies. *Pancreatology* 2020; **20**: 602–7.
15. Nair S, Yadav D, Pitchumoni CS. Association of diabetic ketoacidosis and acute pancreatitis: observations in 100 consecutive episodes of DKA. *Am. J. Gastroenterol.* 2000; **95**: 2795–800.
16. Lando HM, Alattar M, Dua AP. Elevated amylase and lipase levels in patients using glucagonlike peptide-1 receptor agonists or dipeptidyl-peptidase-4 inhibitors in the outpatient setting. *Endocr. Pract.* 2012; **18**: 472–7.
17. Lai T, Li J, Zhou Z *et al*. Etiological changes and prognosis of hospitalized patients with acute pancreatitis over a 15-year period. *Dig. Dis. Sci.* 2023; **69**: 56–65.
18. Tai WP, Lin XC, Liu H *et al*. A retrospective research of the characteristic of hypertriglyceridemic pancreatitis in Beijing, China. *Gastroenterol. Res. Pract.* 2016; **2016**: 6263095.
19. Li J, Cao G, Ma Q, Liu H, Li W, Han L. The bidirectional interation between pancreatic cancer and diabetes. *World J. Surg. Oncol.* 2012; **10**: 171.
20. Mellenthin C, Balaban VD, Dugic A, Cullati S. Risk factors for pancreatic cancer in patients with new-onset diabetes: a systematic review and meta-analysis. *Cancers* 2022; **14**: 4684.
21. Pelaez-Luna M, Takahashi N, Fletcher JG, Chari ST. Resectability of presymptomatic pancreatic cancer and its relationship to onset of diabetes: a retrospective review of CT scans and fasting glucose values prior to diagnosis. *Am. J. Gastroenterol.* 2007; **102**: 2157–63.
22. Nederstigt C, Uitbeijerse BS, Janssen LGM, Corssmit EPM, de Koning EJP, Dekkers OM. Associated auto-immune disease in type 1 diabetes patients: a systematic review and meta-analysis. *Eur. J. Endocrinol.* 2019; **180**: 135–44.
23. Saint-Pol AL, Morvan T, Maurette P. Adrenocortical insufficiency and abdominal pain. *Ann. Fr. Anesth. Reanim.* 2010; **29**: 494–6.
24. Thyo AA, Lausten SB. Addison's disease causing acute abdomen. *Ugeskr. Laeger* 2012; **174**: 1458–9.
25. Culic V, Miric D, Eterovic D. Correlation between symptomatology and site of acute myocardial infarction. *Int. J. Cardiol.* 2001; **77**: 163–8.
26. Pierard S, Hantson P. Coronary vasospasm complicating cannabinoid hyperemesis syndrome. *J. Cardiol. Cases* 2017; **15**: 115–8.
27. Aksakal E, Duman H, Ulus T, Bayram E. Acute inferior pseudoinfarction pattern in a patient with normokalemia and diabetic ketoacidosis. *Am. J. Emerg. Med.* 2009; **27**: e3–5.
28. Zaki HA, Shaban EE, Shaban AE, Elmoheen A. High troponin-T in acute biliary pancreatitis: is it a real myocardial injury? *Cureus* 2021; **13**: e18637.
29. Pourfridoni M, Khan MAB, Khalil-Khan A, Mohammad Bagheri Rafsanjani A, Askarpour H. Elevated troponin level and nonspecific ST-segment and T-wave changes in a suspected acute pancreatitis patient, post-SARS-Cov-2 infection: a case report. *Clin. Case Rep.* 2022; **10**: e6628.
30. Ho JSY, Mui B, Sia CH *et al*. A 78-year-old male with inferior ST-segment elevation on electrocardiogram, diabetic ketoacidosis and acute pancreatitis. *Cardiovasc. Endocrinol. Metab.* 2020; **9**: 186–8.
31. Fitzpatrick E, Cotoi C, Quaglia A, Sakellariou S, Ford-Adams ME, Hadzic N. Hepatopathy of Mauriac syndrome: a retrospective review from a tertiary liver centre. *Arch. Dis. Child.* 2014; **99**: 354–7.
32. Akturk HK, Taylor DD, Camsari UM, Rewers A, Kinney GL, Shah VN. Association between cannabis use and risk for diabetic ketoacidosis in adults with type 1 diabetes. *JAMA Intern. Med.* 2019; **179**: 115–8.
33. Aung ETBT, Wace P, Weston PJ. Cannabinoid hyperemesis syndrome as a cause of recurrent vomiting in type 1 diabetes. *Pract. Diabetes* 2021; **38**: 22–4.
34. Parkman HP, Wilson LA, Hasler WL *et al*. Abdominal pain in patients with gastroparesis: associations with gastroparesis symptoms, etiology of gastroparesis, gastric emptying, somatization, and quality of life. *Dig. Dis. Sci.* 2019; **64**: 2242–55.
35. Schwartz BE, Baker KK, Bleinberger AJ, Lleshi A, Cruz-Cano R. Intravenous haloperidol for the treatment of intractable vomiting, cyclical vomiting, and gastroparesis. *World J. Emerg. Med.* 2021; **12**: 228–31.
36. Stirrup N, Jones G, Arthur J, Lewis Z. Droperidol undermining gastroparesis symptoms (DRUGS) in the emergency department. *Am. J. Emerg. Med.* 2024; **75**: 42–5.
37. Letizia MTC, Belfiore I, Pelliano V, Piccirillo M, Rinaldi F, Sabatino V. Recurrent abdominal pain and celiac disease. *Dig. Liver Dis.* 2013; **45**: e288.
38. Sanders DS, Hopper AD, Azmy IA, Hurlstone DP. Is there an association between adult coeliac disease and non-specific abdominal pain? *Scand. J. Gastroenterol.* 2007; **42**: 896–7 author reply 98.
39. Madacsy L, Arato A, Korner A. Celiac disease as a frequent cause of abdominal symptoms in children with insulin-dependent diabetes

mellitus. *Clin. Pediatr.* 1997; **36**: 185–6.
40. Toyoshima MT, Queiroz MS, Silva ME, Correa-Giannella ML, Nery M. Celiac crisis in an adult type 1 diabetes mellitus patient: a rare manifestation of celiac disease. *Arq. Bras. Endocrinol. Metabol.* 2013; **57**: 650–2.
41. Kikta DG, Breuer AC, Wilbourn AJ. Thoracic root pain in diabetes: the spectrum of clinical and electromyographic findings. *Ann. Neurol.* 1982; **11**: 80–5.
42. Mentzer RM Jr, Golden GT, Chandler JG, Horsley JS 3rd. A comparative appraisal of emphysematous cholecystitis. *Am. J. Surg.* 1975; **129**: 10–5.
43. McMillin K. Computed tomography of emphysematous cholecystitis. *J. Comput. Assist. Tomogr.* 1985; **9**: 330–2.
44. Desai R, Batura D. A systematic review and meta-analysis of risk factors and treatment choices in emphysematous pyelonephritis. *Int. Urol. Nephrol.* 2022; **54**: 717–36.
45. Wang F, Yu J, Chen W, Mo Z, Zhang Y. Clinical characteristics of diabetes complicated by bacterial liver abscess and nondiabetes-associated liver abscess. *Dis. Markers* 2022; **2022**: 7512736.
46. Australian Government. Australian Institute of Health and Welfare. Weight Loss Surgery in Australia 2014–15: Australian Hospital Statistics; 2017: 1–96. Available from URL: https://www.aihw.gov.au/getmedia/52999476-6016-456e-8dab-.
47. Petrucciani N, Ciangura C, Debs T *et al*. Management of surgical complications of previous bariatric surgery in pregnant women. A systematic review from the BARIA-MAT Study Group. *Surg. Obes. Relat. Dis.* 2020; **16**: 312–31.
48. Nabrdalik K, Skonieczna-Zydecka K, Irlik K *et al*. Gastrointestinal adverse events of metformin treatment in patients with type 2 diabetes mellitus: a systematic review, meta-analysis and meta-regression of randomized controlled trials. *Front. Endocrinol.* 2022; **13**: 975912.
49. Kopec KT, Kowalski MJ. Metformin-associated lactic acidosis (MALA): case files of the Einstein Medical Center medical toxicology fellowship. *J. Med. Toxicol.* 2013; **9**: 61–6.
50. Filippatos TD, Panagiotopoulou TV, Elisaf MS. Adverse effects of GLP-1 receptor agonists. *Rev. Diabet. Stud.* 2014; **11**: 202–30.