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INTRAMURAL DUODENUM HAEMATOMA IN A CHILD WITH REPEATED ATTACKS OF PANCREATITIS

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A b s t r a c t

In this publication we present the case report of a child born in 1998, who was admitted to our department several times with repeated attacks of pancreatitis. During the hospitalisation the patient was checked up for ERCP, among other examinations. During further hospitalisations with the same diagnosis a tumorous formation of duodenal haematoma appeared, which was the reason for a surgery revision with the presurgery and histological diagnosis of intramural duodenum haematoma. This diagnosis is rare in children and is usually associated with a blunt trauma in the abdomen, which was not the case. In the discussion we consider the possible causes of this disease.

K e y w o r d s

Paediatric pancreatitis, ERCP, Intramural duodenal haematoma

INTRODUCTION

Pancreatitis is a relatively rare cause of pain in the abdomen in children. It is described mainly in association with metabolic diseases (*1*), diseases of the biliary tract (pigment stones in haemolytic disease, a choledochal cyst, parasites), virus infection (cytomegalovirus, varicella, coxsackievirus B), bacterial infection (Salmonella, Mycoplasmas), anomalies in the pancreatic duct (pancreas divisum), familial chronic pancreatitis, toxins (alcohol, boric acid), and trauma (blunt abdominal trauma, surgery, ERCP) (*2*). Metabolic causes include hypercalcaemia associated with primary hyperparathyroidism caused by the adenoma of parathyroidoma, hyperlipidemia, or aminoaciduria. In 25 % of paediatric patients the cause of pancreatitis is unknown. In these cases recurring attacks are described in as much as 28 % (*3*).

One of the main clinical symptoms, similarly to adults, is nausea, vomiting, and pain in the abdomen located in the epigastrium or the central part of the abdomen, sometimes radiating into the back (*4*), muscular tension in the upper part of the

abdomen, tachycardia, and frequently fever. Laboratory tests reveal an increased number of leucocytes, increased haematocrit as the result of haemoconcentration due to the loss of liquid, in 15 % of patients there is hypocalcaemia, and in up to 25 % of the patients there is hyperglycaemia during the acute attack (*3*).

The sensitivity of amylase in the serum is lower in paediatric patients, compared to adults; according to some authors it can be absent in up to 40 % of cases (*5*), nonetheless, its increase to triple values is considered significant. The values of serum lipases are usually increased as well but the elevation has a low correlation with the extent of pancreatitis.

If no objective cause of the illness is identified or if there are no complications requiring surgery, the treatment is conservative, consisting of complete parenteral nutrition with the release of gastric juices with a nasogastric probe, sufficient substitution of liquid and ions, analgetic therapy due to the Oddi's sphincter spasm, and coverage of the nutrition needs with parenteral supply. Antibiotics are not a first-choice treatment; they should be limited to cases where there is a suspicion of infection or an increased number of leucocytes and CRP, and the patient is feverish. (*3*) The treatment of the illness is strictly individual, sometimes in paediatric patients the condition improves quickly but there are also cases of difficult recovery with the necessity of surgical revision. Besides the sonographic check-up, computer tomography and magnetic resonance, endoscopic retrograde cholangiopancreatography is used in paediatric patients to identify the possible pathology in the area of the biliary tract and pancreatic duct. We emphasise this type of examination as one of the possible causes of the illness listed in the casuistics. Bleeding into the retroperitoneum or duodenum occurs more frequently in children in relation to a blunt abdominal trauma (*7*). Haematoma of the duodenum wall in this case is a relatively rare diagnosis and there is not sufficient experience with the accurate diagnostics and therapy (*8*). Spontaneous intramural duodenum haematoma caused by pancreatitis is rare in adults and is mostly induced by anticoagulation treatment (*9*).

Other sources present the rupture of a pancreatoduodenal artery aneurysm as the cause for duodenum haematoma (*10*). The origin of intramural haematoma of the duodenum wall can relate to the biopsy of the intestine walls during histological examination (*11*). We did not discover that our patient suffered from any metabolic diseases or any other aetiological factors.

The haematoma of the duodenal wall without a traumatic cause is a rare diagnosis in paediatric patients.

MATERIAL AND METHODS

Our casuistics debates a female child born in January 1998. The anamnesis did not identify any hereditary inclination; prior to the first hospitalisation the patient underwent only an adenotomy without complications. Pre-surgery examinations identified a slightly prolonged APTT, the other coagulation status was within physiological limits.

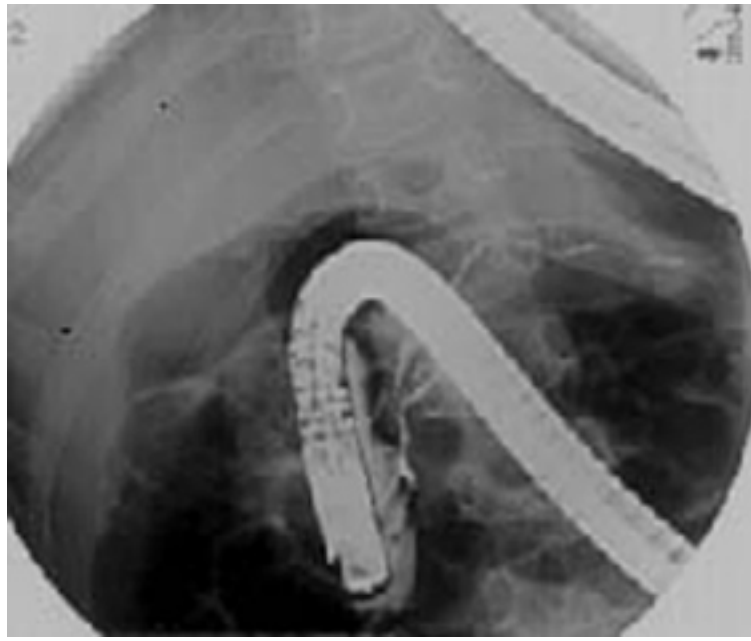

Fig. 1
Endoscopic retrograde cholangiopancreatography with negative findings (June 2002)

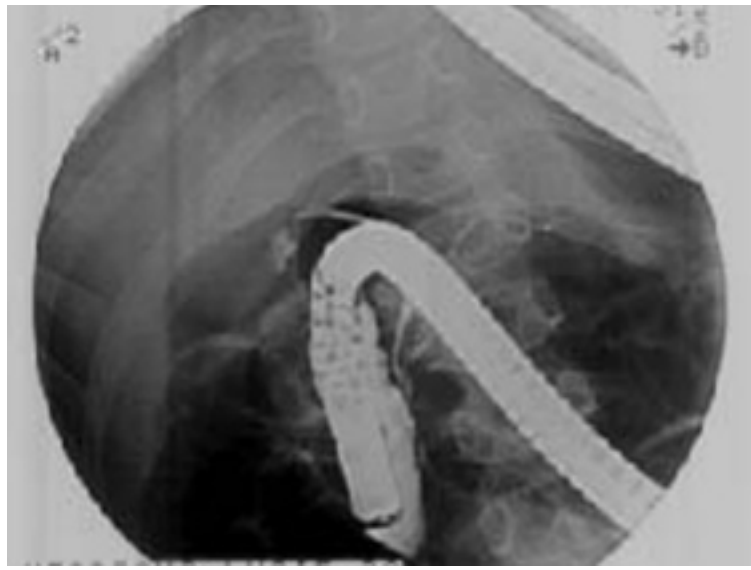

Fig. 2
Endoscopic retrograde cholangiopancreatography with negative findings (June 2002)

The patient was repeatedly admitted to our department with pains in the abdomen and with increased values of serum amylase; the problems usually occurred after a dietary mistake, for example after eating meatloaf or grilled sausage.

For the first time the patient was admitted from 29 March to 2 April 2002. In terms of anamnesis, the pain in the abdomen lasted 6 hours before admission, the patient vomited twice at home and twice in hospital, the pain was localised in the epigastrium. The ultrasound examination showed an enlarged pancreas: capita 16 mm, body 18 mm, cauda 25.2 mm. Laboratory tests were within limits, prolonged APTT (1.3, 1.2). Level F XII 77 %. After one-day tea diet and subsequent gradual introduction of dietary food the condition improved and the patient was released on 2 April with a normal level of serum amylase.

The second admission to our department was from 19 June 2002. Admitted with pain in the epigastrium, vomited twice. Amylase at admission 23, CRP O, Leu 10. Ultrasound examination negative. During hospitalisation ERCP examination carried out with a negative finding (*Figs. 1–2*). Following a conservative therapy the patient was released on 27 June 2002 with a negative clinical finding and a normalised level of serum amylase. Administration of Pancreolan 3x1.

A third admission took place from 19 August 2002, when the patient came with pain in the epigastrium, without vomiting. Amylase in serum at admission 14.12, at release 3.48, maximum Leu during hospitalisation 8.8, CRP 0. Ultrasound identified a diffusion enlarged pancreas echogenicity, the thickness in the body was 15–16 mm, without dilatation of ductus pancreaticus, the pancreas surrounding without free liquid. Following a conservation treatment the patient was released on 26 August 2002, recommended Pancreolan and pancreatitic diet.

The next admission took place from 9 May 2003 with pains in the epigastrium and mesogastrium. Laboratory tests identified amylase values at admission 26.2, Leu 18, CRP O, with gradual improvement following conservative therapy. At admission, the ultrasound examination identified that the pancreas was difficult to view due to pneumatosis, a 24 x 27 mm formation suspected in the left adrenal gland area. A review ultrasound examination on 12 May was negative; due to the negative finding the patient was released from the hospital and invited for a check-up in one month, diet, Pancreolan. Ultrasound examinations were carried out on 23 and 27 May concentrating at pancreas and the left adrenal gland area; both sonographic findings were negative.

On 22 January 2004 the patient was admitted again with pain in the abdomen and vomiting three times. Condition after the antibiotics treatment of tonsillitis one week before admission. No trauma in the abdomen was identified through questioning. Amylase in serum at admission 2.3, max. 4.9. Ultrasound examination on 22 January identified a dilated duodenum ansa around the submerged capita of the pancreas (capita and body 11.2 mm, cauda 18 mm). In the area between the pancreas capita and the gall bladder a solid focus of 47 x 43 x 70 mm was identified with a central share of liquid. This diagnosis was confirmed by a CT examination describing a globular cystoid formation with the content of higher densities 40–50 HU, size 37 x 39 x 70 mm (*Figs. 3–4*).

Part of the duodenum is compressed and dilated before the formation, the formation is likely to cause a light obstruction in the papilla localisation, the pancreatic tract is more spacious. The gland itself is not enlarged, of normal structure and density. A passage was made through the gastrointestinal tract: dilatation of the duodenum bulb and the proximal part of DI, a significant duodenum antiperistaltic and duodenogastric reflux. A remarkable right-side impression about 58 mm long in the descending part of the duodenum with stagnation of the contrast substance above the spot. RES: stenosis D II conditioned by a pathological focus of extramural aetiology (*Fig. 5*).

Operation on 29 January 2004: Attempt at pre-operational endoscopy: due to stenotic process it was impossible to penetrate into the duodenum. Laparoscopy: in the sub-hepatic area on the duodenum a solid cylindrical formation identified, passing to the next duodenum section. Conversion: after mobilisation according to Kocher a solid formation found in the duodenum (*Fig. 6*) wall in areas D II and D III, thrombus vacuumed and coagulum evacuated (histology) after discission following a puncture and histological sampling. A broader discission was carried out, a papilla located 40 mm from the discission in the proximal direction was checked. Transverse suture of the duodenum. Further post-operation condition was without complications. On 9 February 2004 the patient was released from the hospital with a healed operation wound; the amylase values were normal.

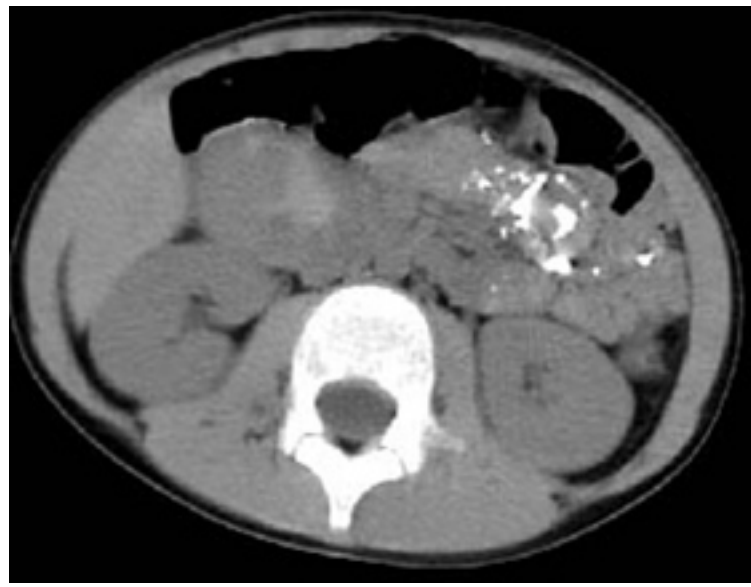

Fig. 3
CT scan – preoperative investigation showing solid formation of size 37x39x70 mm

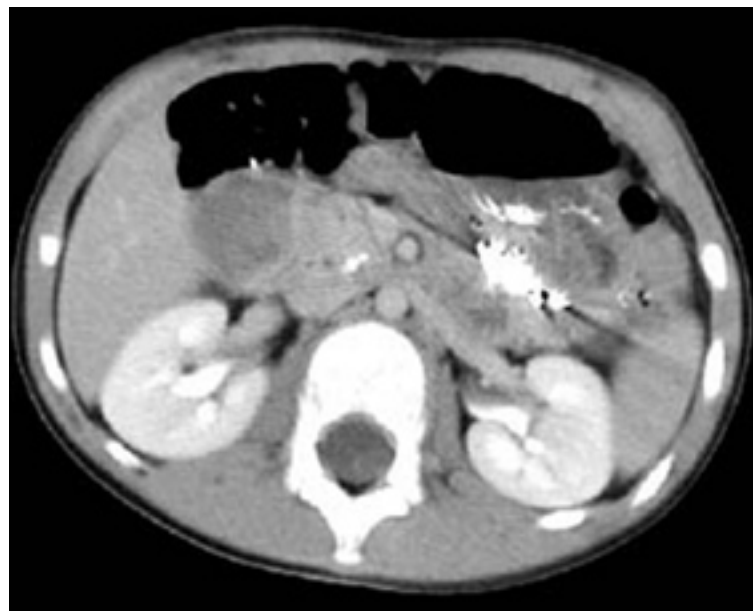

Fig. 4
CT scan – preoperative investigation

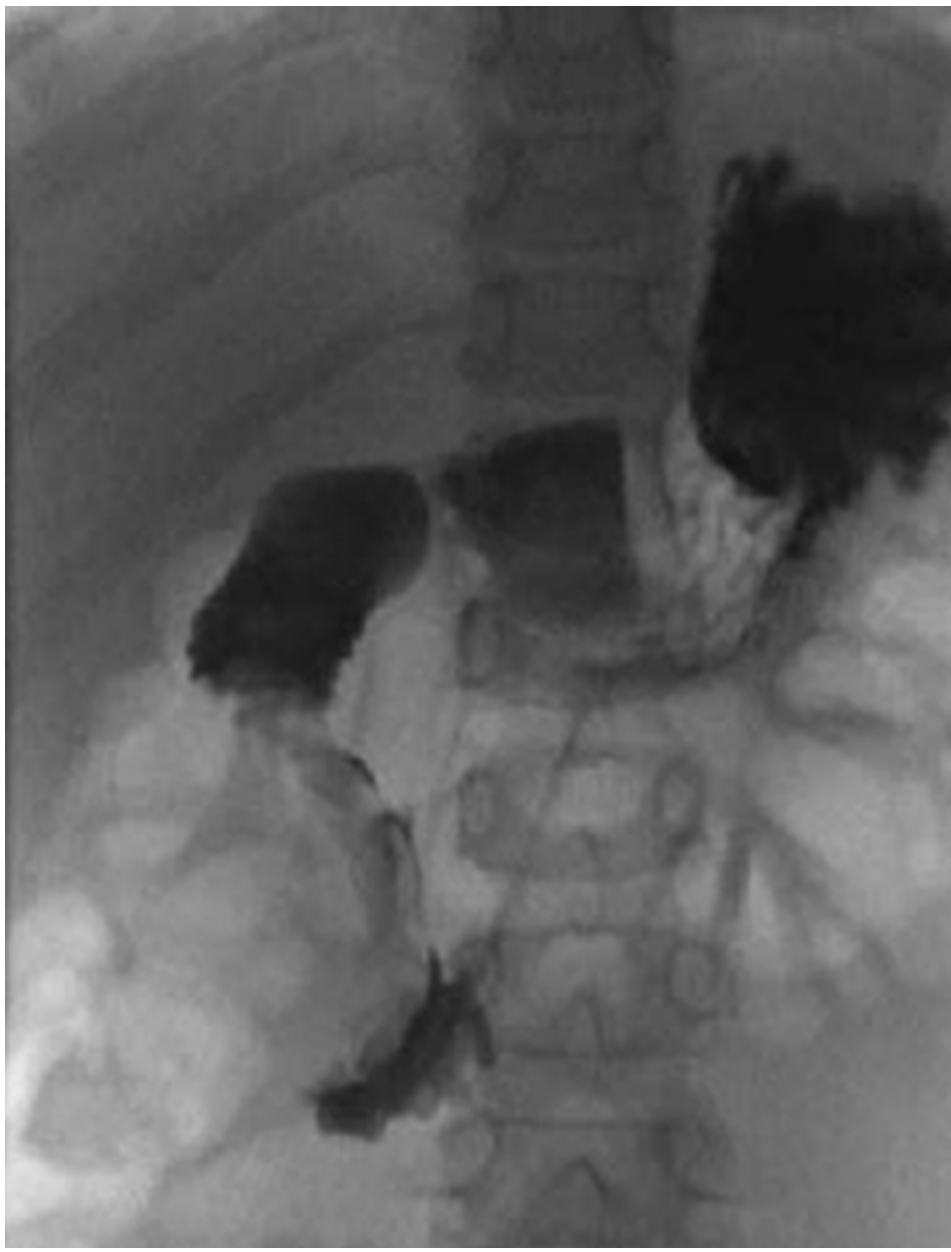

Fig. 5
x-ray passage – preoperative investigation with duodenal compression and stenosis

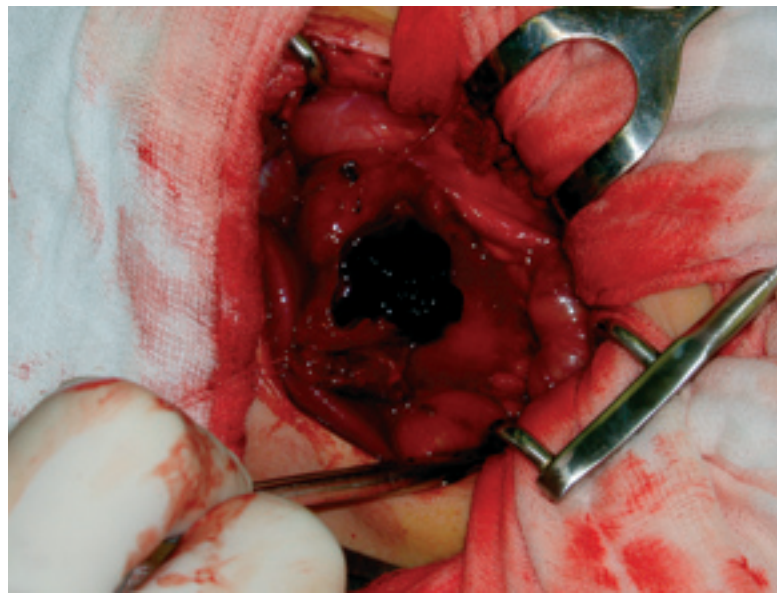

Fig. 6
Intramural blood tumor of the duodenal wall – intraoperative view

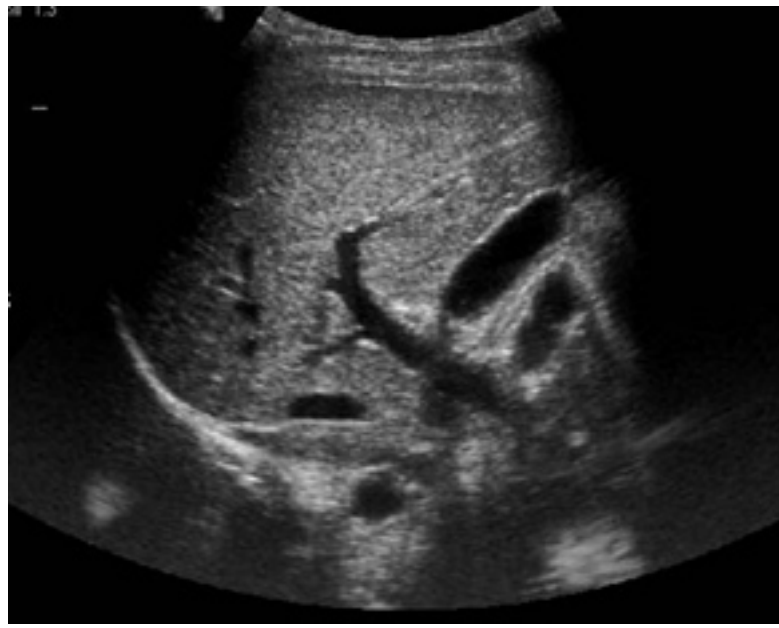

Fig. 7
Ultrasonic investigation – postoperative with negative findings (May 2004)

Conclusion of the histological examination: haemorrhage in muscularis and adventitia of the duodenum wall.

So far the last admission took place on 15 February 2004. The cause was a short history of pain in the abdomen; amylase in serum at admission was 16.39, at release 3.68, CRP O, Leu 12,2. Ultrasound examination of the abdomen was negative in the area of the pancreas capita, in the area of the gland a small amount of liquid was suspected.

The passage through the duodenum shows a discrete narrowing of D II which is freely developing with a free flow of the contrast substance into other sections of the small intestine. After conservative therapy the child was released without any symptoms; diet and Pancreolan were recommended.

On 26 May 2004 the last ultrasound examination took place at the outpatient department with a negative finding *(Fig. 7*). At that time the child was without clinical symptoms.

DISCUSSION

Pancreatitis in childhood and haematoma of the duodenum without a traumatic cause are very rare diagnoses in children; in our case they occurred only in one patient. As regards pancreatitis, it is a disease without any aetiological grounds, where the literature supposes the highest probability of relapse. In the case of the haematoma of the duodenum we considered an association with the ERCP examination but this was carried out 18 months before the diagnosis and a number of check-ups in the meantime were negative. The parents ruled out the possibility of an accident and the clinical finding did not identify any signs of abdominal trauma. The slightly prolonged APTT in the haemocoagulation status did not cause any bleeding conditions in the patient and is not likely to be the cause for the haematoma. The literature rarely mentions bleeding into the jejunum wall in adults induced by long-term administration of coumarin preparations (*12*). In the aetiology of the haematoma origin we incline toward the bleeding induced by the pancreatitis described in the literature; however, this theory is rather a hypothesis. The operation revision in our case was required due to the fact that an accurate differential diagnostic of the possible tumour duodenum was not possible and the haematoma obstructed the intestine passage. Repeated attacks of pancreatitis were undoubtedly also caused by the failure to observe the recommended diet.

Acknowledgement

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INTRAMURÁLNÍ HEMATOM DUODENA U DÍTĚTE S OPAKOVANÝMI ATAKAMI PANKREATITIDY

S o u h r n

V naší kasuistice uvádíme případ dítěte, které bylo na naší klinice hospitalizováno opakovaně s atakami pankreatitidy. Během těchto hospitalizací byla provedena řada vyšetření: opakované sonografie, ERCP a CT vždy s negativními nálezy. V lednu 2004 byl bez jakékoliv traumatické anamnézy zjištěn intramurální hematom duodena, který jsme řešili operací. U dítěte kromě lehce vyšších hodnot APTT nebyly rovněž během hospitalizací zjištěny poruchy koagulace. Uvedená diagnóza bez traumatické příčiny je v dětském věku ojedinělá, u dospělých pacientů je popisována například v souvislosti s dlouhodobým užíváním kumarinových preparátů. V závěru se zamýšlíme nad možnými příčinami vzniku onemocnění, zda se jednalo o spontánní krvácení do stěny dvanácterníku při opakovaných atakách pankreatitidy, nebo zda by se mohlo jednat o pozdní následek vyšetření ERCP.

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