

Elwira Kołodziejczyk, Karolina Wejnarska, Grzegorz Oracz

AUTOIMMUNE PANCREATITIS IN THE PAEDIATRIC POPULATION – REVIEW OF THE LITERATURE AND OWN EXPERIENCE

AUTOIMMUNOLOGICZNE ZAPALENIE TRZUSTKI U DZIECI – PRZEGLĄD PIŚMIENNICTWA I DOŚWIADCZENIE WŁASNE

Department of Gastroenterology, Hepatology, Feeding Disorders and Pediatrics,
The Children's Memorial Health Institute, Warsaw, Poland

Abstract

Autoimmune pancreatitis (AIP) is a rare chronic inflammatory autoimmune disease of the pancreas. It is estimated that it accounts for 2–6% of all the chronic pancreatitis (CP) cases in adult patients. AIP is usually characterized by sudden onset. The presenting symptoms most commonly include painless jaundice, general weakness and loss of weight. Imaging studies often reveal diffuse enlargement or focal changes of pancreatic parenchyma. In view of the clinical manifestation and changes documented on imaging, AIP should be considered after performing a differential diagnosis from pancreatic cancer or inflammatory masses in the course of CP. According to the International Consensus Diagnostic Criteria (ICDC), AIP can be sub-classified into two subtypes. Type 1 AIP is seen mainly in adult patients and characterized by elevated serum IgG4 levels and its association with IgG4-related disease at extrapancreatic sites (eg, sialadenitis, retroperitoneal fibrosis). Type 2 AIP presents in younger individuals. Serological abnormalities are usually absent. There are no systemic manifestations, except for inflammatory bowel disease. Histologically, type 1 AIP is characterized by massive infiltration by IgG4-positive plasma cells, whereas type 2 AIP shows neutrophilic infiltration with granulocytic epithelial lesions (GELs). Both types rapidly respond to steroid therapy. AIP is extremely rare in children. So far about twenty-five case reports of paediatric AIP were described. Due to such an infrequent occurrence, paediatricians may be unfamiliar with the optimal diagnostic and management strategies of this condition.

Key words: autoimmune pancreatitis, children, AIP type 1, AIP type 2

Streszczenie

Autoimmunologiczne zapalenie trzustki (AIP – autoimmune pancreatitis) to stosunkowo rzadkie schorzenie o podłożu autoimmunologicznym z obecnością charakterystycznych zmian biochemicznych, histologicznych i morfologicznych. Ocenia się, że stanowi ono od 2 do 6% przypadków przewlekłego zapalenia trzustki (PZT) u osób dorosłych. Początek choroby zwykle jest nagły. Często występuje żółtaczka oraz ogólne osłabienie. W badaniach obrazowych uwidacznia się uogólnione powiększenie trzustki lub zmiana ogniskowa w miąższu trzustki. Obraz kliniczny oraz zmiany stwierdzane w badaniach obrazowych powodują, że AIP należy w pierwszej kolejności różnicować z rakiem trzustki, a w następnej kolejności z guzem zapalnym w przebiegu PZT. Według najnowszej klasyfikacji wyróżnia się 2 typy choroby. Typ 1 dotyczy głównie osób starszych. Charakteryzuje się podwyższonym stężeniem IgG4 w surowicy oraz współwystępowaniem pozatrzustkowych manifestacji choroby (np. zapaleniem ślinianek, włóknieniem zaotrzewnowym). Typ 2 występuje u osób młodszych, w tym u dzieci. Markery serologiczne (IgG4, autoprzeciwciała) są zwykle nieobecne, chorobie towarzyszą często nieswoiste zapalenia jelit. W badaniu histopatologicznym, typ 1 AIP charakteryzuje się masywnym naciekiem IgG4-dodatnich komórek plazmatycznych, podczas gdy w typie 2 stwierdza się obecność neutrofilowego

nacieku w nabłonku oraz w świetle przewodów (granulocytic epithelial lesions – GELs). AIP bardzo rzadko jest rozpoznawane u dzieci. Do tej pory zarówno w literaturze krajowej, jak i światowej opisano około 25 pojedynczych przypadków tej choroby w populacji pediatrycznej. Sporadyczne występowanie, skąpość danych na temat diagnostyki, ciężkości przebiegu i skuteczności leczenia tej choroby u dzieci sprawiają iż autoimmunologiczne zapalenie trzustki pozostaje dużym wyzwaniem w praktyce lekarza pediatry.

Słowa kluczowe: autoimmunologiczne zapalenie trzustki, dzieci, AIP typ 1, AIP typ 2

DEV PERIOD MED. 2016;XX,4:279-286

INTRODUCTION

Autoimmune pancreatitis (AIP) is a rare, usually adult-onset, chronic inflammatory autoimmune disease of the pancreas, which is extremely uncommon in children. It is suggested that the concept of AIP was for the first time reported more than 50 years ago, when Sarles et al. [1] described pancreatitis associated with hypergammaglobulinemia with probable autoimmunity as a pathogenetic mechanism. However, wider appreciation of AIP came only in the 1990s in the form of case reports of a "mass" lesion, usually mimicking pancreatic adenocarcinoma. The term autoimmune pancreatitis was originally introduced by Yoshida et al [2] in 1995. In 2002 the Japan Pancreas Society published the diagnostic criteria of AIP based on a combination of findings in imaging, laboratory testing, and histological analysis [3]. AIP likely accounts for a significant proportion of cases previously classified as idiopathic pancreatitis [4]. In 2011, the International Association of Pancreatology proposed International Consensus Diagnostic Criteria (ICDC), which are composed of five cardinal features, i.e.: imaging, serology, other organ involvement, histology, and response to steroid therapy, categorized as type 1 or 2 AIP findings depending on the diagnostic reliability [5].

EPIDEMIOLOGY

It is estimated that AIP accounts for 2-6% of all chronic pancreatitis (CP) cases in adult patients [6, 7]. To our knowledge, in the literature there are only a few case reports or small case series of children with AIP, mainly because of the very low incidence of autoimmune pancreatitis in the paediatric population [8-15]. Due to the infrequent occurrence of the condition, paediatricans may be unfamiliar with optimal diagnostic and management strategies of this condition.

DEFINITION

As defined by the ICDC, autoimmune pancreatitis is a distinct form of pancreatitis clinically characterized by frequent presentation with obstructive jaundice with or without a pancreatic mass, histologically by a lymphoplasmacytic infiltrate and fibrosis and therapeutically by a dramatic response to steroids [5].

SIGNS AND SYMPTOMS

AIP is usually characterized by sudden onset. The presenting symptoms are variable and most commonly include painless jaundice and general weakness. Abdominal pain, if any, is mild, whereas the episodes of acute pancreatitis are rarely the first manifestation of the disease [5, 6, 16]. Imaging studies often reveal diffuse enlargement or focal changes of pancreatic parenchyma. In view of the clinical manifestation and changes documented on imaging, AIP should first be considered after carrying out a differential diagnosis from pancreatic cancer, followed by analyzing inflammatory masses in the course of CP [6, 8, 11, 17, 18]. Although autoimmune pancreatitis is less common than pancreatic cancer in adults, failure to consider this disease in making a differential diagnosis can lead to unnecessary medical procedures or pancreatic resection, whereas it often completely resolves with steroid treatment [16].

AIP can also be asymptomatic, with the abnormalities present only in laboratory tests and diagnostic imaging [6, 8, 17, 18].

AIP TYPE 1 AND 2

According to the most recent classification, AIP can be sub-classified into two subtypes, 1 and 2 [5]. The diagnosis of type 1 and type 2 AIP can be definitive or probable, and in some cases, the distinction between the subtypes may not be possible (AIP not otherwise specified). Type 1 AIP, seen mainly in adult patients, is characterized by elevated serum IgG4 levels and association with IgG4-related disease at extrapancreatic sites (eg, sialadenitis, retroperitoneal fibrosis). Type 2 AIP presents in younger individuals, the serological abnormalities are usually absent, there are no systemic manifestations except for possible association with inflammatory bowel disease. Histologically, type 1 AIP is characterized by massive infiltration by IgG4-positive plasma cells, whereas type 2 AIP shows neutrophilic infiltration with granulocytic epithelial lesions (GELs) and few or no IgG4-positive plasma cells [5, 19] (tab. I).

Based on the current state-of-the-art and international consensus for diagnostic criteria, it seems that imaging studies and response to steroids can not distinguish type 1 from type 2. Moreover, the absence of serological markers or the lack of extrapancreatic sites in patients

Table I. Specific features of autoimmune pancreatitis type 1 and 2 [3, 5, 9, 10].

	AIP type 1	AIP type 2
Imaging of pancreatic parenchyma and duct	1 Diffuse enlargement with delayed enhancement of pancreas (less typical segmental/focal enlargement) 2 Long (>1/3 length of the main pancreatic duct) or multiple strictures without marked upstream dilatation of main pancreatic duct	1 Diffuse enlargement with delayed enhancement of pancreas (less typical segmental/focal enlargement) 2 Long (>1/3 length of the main pancreatic duct) or multiple strictures without marked upstream dilatation of main pancreatic duct
Serology	IgG4, 2 x upper limit of normal value	Often absence of serological abnormalities
Other organ involvement	Segmental/multiple proximal or proximal and distal bile duct stricture Symmetrically enlarged salivary/lachrymal glands Retroperitoneal fibrosis Radiological evidence of renal involvement described in association with AIP	Clinically diagnosed inflammatory bowel disease
Pancreatic Histology	1 Periductal lymphoplasmacytic infiltrate without granulocytic infiltration 2 Obliterative phlebitis 3 Storiform fibrosis 4 Abundant (>10 cells/HPF) IgG4-positive cells	1 Granulocytic infiltration of duct wall (GEL) with or without granulocytic acinar inflammation 2 Absent or scant (0-10 cells/HPF) IgG4-positive cells
Response to steroid therapy	Rapid radiologically demonstrable resolution or marked improvement in pancreatic/extrapancreatic manifestations	Rapid radiologically demonstrable resolution or marked improvement in pancreatic/extrapancreatic manifestations

with AIP does not necessarily imply the diagnosis of type 2, as type 1 can also be seronegative and without systemic manifestations. Using ICDC criteria AIP type 1 can be diagnosed without histology, but type 2 requires an adequate histolopathology result to make a definitive diagnosis.

TREATMENT

A steroid therapy involves the use of prednisone 0.6 to 1 mg/kg with reassessment of imaging after 2 weeks of treatment [5]. Some AIP patients relapse during maintenance therapy or after stopping steroid therapy. They should be re-treated with a high-dose steroid therapy. Recent reports suggest that immunomodulators (azathioprine, 6-mercaptopurine and mycophenolate mofetil), as well biological agents (the antibody to CD20, rituximab) may have a role in maintaining remission in relapsing type 1 AIP [20-21].

REPORT ON CHILDREN WITH AJD PUBLISHED IN THE LITERATURE

Both in the national, as well as in the world literature, there is no data about the incidence of autoimmune pancreatitis in children. Currently there are only sporadic case reports of AIP in children, which suggests that diagnosing the disease may be more difficult than in adults. To our knowledge, so far about twenty-five case reports of paediatric AIP were described [8-15, 22-26] (tab. II).

Similarly to adult patients, the most common clinical presentation of paediatric AIP was a diffuse pancreatic enlargement or pancreatic head mass described in imaging studies with a good steroid response. However, some other features appear to be distinct. AIP type 1 occurs mainly in adults. In paediatric patients the predominant type appears to be AIP 2, which is the disorder not associated with IgG4. IgG4 level and anti-tissue antibodies are helpful in making the diagnosis of AIP when they are elevated. In a study by Oracz et al., the presence of anti-tissue antibodies was detected in 75 out of 129 (58%) children and a the clinically significant increase in IgG4 concentration was revealed in 24 out of 68 (35.3%) patients who were examined for this parameter [9]. However, on the faith of IAP criteria, a suspicion of AIP was finally raised only in 6 and the diagnosis was definitely confirmed in 2 cases, based on clinical improvement observed after corticosteroid therapy. Perhaps the high percentage of patients with

Table II. Reported paediatric cases of AIP.

Patient No.	Age/Sex	Serum IgG4 elevated	Autoimmune antibodies	Imaging studies	Histopathology	Treatment	**Diagnosis**
1. (13)	10/M	No	Negative	US – enlarged pancreas and 2 cystic structures in the cephalic pancreas. MRCP – globular, slightly heterogeneous pancreas and dense peripancreatic fat associated with moniliform multiple stenoses of the Wirsung duct and dilatation of the main and intrahepatic biliary tract	-	Response to steroids; no relapse	AIP, non-specified
2. (25)	7/M	No	-	CT – a 3 cm mass in the head of the pancreas; MRCP - 2.2 x 3.3 x 4.3 cm isointense mass in the head of the pancreas causing obstruction of the common bile duct and partial obstruction of the pancreatic duct	Duct-centric pancreatitis, also referred to as type II autoimmune pancreatitis or idiopathic duct destructive pancreatitis.	Response to steroids; ERCP – stent placement; no relapse	AIP, non specified
3. (26)	8/F	Yes - 320 mg/dl		MRCP – a small volume pancreas and residual lowgrade inflammation throughout with emphasis on the body and tail; the pancreatic duct was not seen.	-	Response to steroids; no relapse	AIP 1
4. (15)	14/F	No	Negative	CT – enlargement of the head of the pancreas; MRCP – enlarged main pancreatic duct in the body of the pancreas with its narrowing in the distal portion; PD in the head of the pancreas was not seen	-	Respond to steroids, two episodes of relapse, both respond to steroids	AIP, non specified
5. (12)	9/-	No	-	MRCP – extrahepatic and intrahepatic ductal dilation suggestive of a biliary stricture	Features consistent with AIP; GELs positive	Respond to steroides	AIP 2

Table II. Cont.

6. (12)	9/-	No	-	MRCP – extrahepatic and intrahepatic ductal dilation suggestive of a biliary stricture	Features consistent with AIP; GELs positive	Respond to steroides	AIP 2
7. (12)	14/-	Yes - 274 mg/dl	-	MRCP – features of pancreatitis	Features consistent with AIP; GELs positive	No steroids prescribed	AIP 2
8. (12)	16/-	No	-	MRCP – features of pancreatitis	Features consistent with AIP; GELs positive	Respond to steroides	AIP 2
9. (12)	17/-	No	-	MRCP – no abnormalities	Features consistent with AIP	Respond to steroides	AIP 2
10. (12)	17/-	No	-	MRCP – features of pancreatitis	Likely AIP; GELs positive	No steroids prescribed	AIP 2
11. (12)	18/-	No	-	MRCP –no abnormalities ; EUS - characteristic features of AIP	Nondiagnostic biopsy	No steroids prescribed	AIP 2
12. (14)	11/M	No	Negative	US, MRI – pancreatic head mass; MRCP – diffuse irregular narrowing of main pancreatic duct, narrow intrapancreatic portion of common bile duct	Extensive pancreatic tissue destruction with marked periductal fibrosis and lymphoplasmacytic infiltration	-	AIP, non-specified
13. (11)	10/M	No		MRI,MRCP – pancreatic head mass with mildly dilated pancreatic duct	Ductocentric lymphoplasmacytic infiltration and fibrosis, GELs positive, IgG4 + plasma cells	Whipple procedure, no relapse	AIP 2
14. (11)	15/M	No		MRI, MRCP – pancreatic head mass, normal pancreatic duct	Ductocentric lymphoplasmacytic inflammation with swirling collagen fibriles and venulitis, IgG4+ plasma cells	Response to steroids, no relapse	AIP 1
15. (11)	11/M	Yes -224 mg/dl		MRI,MRCP – mildly prominent pancreas with irregularity and dilatation of the pancreatic duct		Respond to steroids; 1 relapse, treated with 6-mercaptopurine	AIP 1

Table II. Cont.

16. (8)	16/M	No		US, CT – enlarged head of the pancreas; MRCP – enlarged head of the pancreas, normal pancreatic duct; dilated intra- and extrahepatic biliary tract; the choledochus was not visible, and the cystic duct ended abruptly.	Chronic pancreatitis with interstitial periductal lymphoplasmacytic infiltration	Response to steroids, no relapse	AIP, non specified
17. (24)	10/M			CT – pancreatic head mass, hepatic ductal dilatation, and involvement of the portal vein.	Diagnosed as lymphoplasmacytic sclerosing pancreatitis	Whipple procedure; no relapse	Possible AIP 1
18. (23)	15/F			MRCP – main pancreatic duct stenosis	Ductocentric pancreatitis with neutrophilic infiltration in and around ducts and at the periphery of the lobules GELs positive	Distal pancreatectomy; no relapse	AIP 2
19. (10)	11/M	No	Negative	CT – enlarged pancreas with loss of the cobblestone architecture of the pancreatic surface; ERCP – a tight stricture in the lower bile duct and almost occluded pancreatic duct	Lymphoplasmacytic infiltration, many neutrophils GELs positive Scarce IgG4 +plasma cells	Response to steroids, no relapse	AIP 2
20. (10)	14/F	Yes (slightly)- 190 mg/dL	Slightly positive myeloperoxidase -anti-neutrophilic cytoplasmic antibody; other negative	CT – a mass-like enlargement of the pancreatic head with a tight stricture of the lower bile duct and calibre irregularity of the pancreatic duct.	Lymphoplasmacytic infiltration, periductal and lobular neutrophilic infiltration, GELs positive No IgG4 plasma cells	No steroids prescribed	AIP 2

CP showing autoimmune markers, both in children and adults, is related to the pathomechanism of CP [9, 27]. Chronic inflammation is associated with the release of autoantigens from pancreatic parenchymal cells. This is reflected by the synthesis of autoantibodies, as in the case of other chronic inflammatory process. Murata et al. described three cases of childhood-onset AIP: 2 boys aged 4 and 16 years, diagnosed with ulcerative colitis and 1 case in a previously healthy 10-year-old boy [23]. All three children presented with abdominal pain associated with elevated pancreatic enzyme levels. Serum IgG4 level was elevated only in the 16-year-old boy. Pancreatic enlargement together with the narrowing of the main pancreatic duct was evident on CT-scans in all 3 patients. As shown in the data presented in Table II, serum IgG4 elevation is rarely seen in children with a final diagnosis of autoimmune pancreatitis. Based on the available case reports it is hard to assess the diagnostic usefulness of autoimmune antibodies in paediatric AIP, because in most cases there is no data about the presence or absence of autoantibodies. The diagnosis of AIP may be delayed or overlooked in children, because serologic markers are often unreliable. The performance of a percutaneous biopsy or brushings obtained from ERCP should be taken into consideration in any child presenting with pancreatitis associated with pancreatic mass. Additionally, the authors from the Mayo clinic recommend the inclusion of EUS-guided pancreatic biopsy in the evaluation of AIP in paediatric patients [12]. Not only is the histopathological examination helpful in confirming the diagnosis of AIP, but it can also distinguish between the types of this condition. Because of the higher frequency of AIP in adults compared to children, diagnosis of AIP in paediatric patients should be based on criteria of ICDC. Future studies of AIP in children are needed to expand our knowledge of this rare entity. However, without an international registry of patients it may be difficult to conduct them, as AIP is so scarce a disease in children.

REFERENCES

1. Sarles H, Sarles JC, Muratore R, *et al.* Chronic inflammatory sclerosis of the pancreas: an autoimmune pancreatic disease? Am J Dig Dis. 1961;6:688-698.
2. Yoshida K, Toki F, Takeuchi T, et al. Chronic pancreatitis caused by an autoimmune abnormality. Proposal of the concept of autoimmune pancreatitis. Dig Dis Sci. 1995;40(7):1561-1568.
3. Members of the Criteria Committee for Autoimmune Pancreatitis of the Japan Pancreas Society. Diagnostic criteria for autoimmune pancreatitis by the Japan Pancreas Society. J Jpn Pan Soc. 2002;17:585-587.
4. Zandieh I, Byrne MF. Autoimmune pancreatitis: a review. World J Gastroenterol. 2007;13:6327-6332.
5. Shimosegawa T, Chari ST, Frulloni L, et al. International consensus diagnostic criteria for autoimmune pancreatitis: guidelines of the International Association of Pancreatology. Pancreas 2011;40(3):352-358.
6. Finkelberg D, Sahani D, Desphande V, Brugge W. Autoimmune pancreatitis. N Engl J Med. 2006; 355: 2670-2678.
7. Braganza J, Lee S, McCloy R, McMahon M. Chronic pancreatitis. Lancet 2011; 377:1184-1197.
8. Blejter J, Weller S, Pace R, et al. Autoimmune pancreatitis: an adolescent case and review of literature. J Pediatr Surg. 2008;43:1368-1372.
9. Oracz G, Cukrowska B, Kierkus J, Ryzko J. Autoimmune markers in children with chronic pancreatitis. Prz Gastroenterol. 2014;9:142-146.
10. Zen Y, Grammatikopoulos T, Hadzic N. Autoimmune pancreatitis in children: insights into the diagnostic challenge. J Pediatr Gastroenterol Nutr 2013; May 2; in press.
11. Friedlander J, Quiros JA, Morgan T, et al. Diagnosis of autoimmune pancreatitis vs neoplasms in children with pancreatic mass and biliary obstruction. Clin Gastroenterol Hepatol. 2012;10:1051-1055.
12. Fujii L, Chari S, El-Youssef M, et al. Pediatric pancreatic EUS-guided trucut biopsy for evaluation of autoimmune pancreatitis. Gastrointest Endosc. 2013;77:824-828.
13. Gargouri L, Ponsot P, Viala J, Belarbi N, Martinez C, Bellaiche M, et al. Recurrent autoimmune pancreatitis in a 10-year-old boy. J Pediatr Gastroenterol Nutr. 2009;48:374-377.
14. Refaat R, Harth M, Proschek P, Lindemayr S, Vogl T. Autoimmune pancreatitis in an 11-year-old boy. Pediatr Radiol. 2009;39:389-392
15. Takase M, Imai T, Nozaki F. Relapsing autoimmune pancreatitis in a 14 year old girl. J Nippon Med Sch. 2010;77:29-34.
16. Greenberger NJ, Blumberg RS, Burakoff R. Autoimmunologiczne zapalenie trzustki. Gastroenterologia, hepatologia i endoskopia. Czelej 2012: 324-325.
17. Park D, Kim M, Chari S. Recent advances in autoimmune pancreatitis. Gut 2009;58:1680-1689.
18. Hart P, Kamisawa T, Bugge W, et al. Long-term outcomes of autoimmune pancreatitis: a multicentre, international analysis. Gut 2013;62:1771-1776.
19. Kamisawa T, Chari ST, Giday SA, et al. Clinical profile of autoimmune pancreatitis and its histological subtypes. Pancreas 2011;40:809-814.
20. Sodikoff JB, Keilin SA, Qiang Cai, et al. Mycophenolate mofetil for maintenance of remission in steroid-dependent autoimmune pancreatitis. World J Gastroenterol. 2012;18:2287-2290.
21. Topazian M, Witzig TE, Smyrk TC, et al. Rituximab therapy for refractory biliary strictures in immunoglobulin G4-associated cholangitis. Clin Gastroenterol Hepatol. 2008;6:364-366.
22. Murata S, Yoden A, Aomatsu T, et al. Three cases of childhood-onset autoimmune pancreatitis. Japanese J Gastroenterol. 2014;111(8):1632-1639.
23. Zamboni G, Luttges J, Capelli P, et al. Histopathological features of diagnostic and clinical relevance in autoimmune pancreatitis: a study on 53 resection specimens and 9 biopsy specimens. Virchows Arch. 2004;445:552-563.
24. Bartholomew SV, Zigman A, Sheppard B. Lymphoplasmacytic sclerosing pancreatitis presenting as a pancreatic head mass in a child: case report and management recommendations. J Pediatr Surg. 2006;41:23-25.
25. Long J, Birken G, Migicovsky B. Autoimmune pancreatitis in a child presenting as a pancreatic mass. J Ped Surg Case Reports. 2015;3:111-113.

26. Galloway DP, Wallihan D, Smith MT, et al. An unusual presentation of pediatric autoimmune pancreatitis. Pancreas 2016;45:e1-2.
27. Uzan NK, Levy P, O'Tolle D. Is idiopathic chronic pancreatitis an autoimmune disease? Clin Gastroenterol Hepatol. 2005;3:903-909.

Author's contributions/Wkład Autorów
According to the order of the Authorship/Według kolejności

Conflicts of interest/Konflikt interesu
The Authors declare no conflict of interest.
Autorzy pracy nie zgłaszają konfliktu interesów.

Received/Nadesłano: 29.06.2016 r.
Accepted/Zaakceptowano: 23.11.2016 r

Published online/Dostępne online

Address for correspondence:
Elwira Kołodziejczyk
Klinika Gastroenterologii, Hepatologii,
Zaburzeń Odżywiania i Pediatrii
Instytut „Pomnik – Centrum Zdrowia Dziecka"
Al. Dzieci Polskich 20, 04-730 Warszawa
tel. (+48 22) 815-73-84
e-mail: elwira.kolodziejczyk@vp.pl