

Diagnosis of Gastrointestinal symptoms and follow-up of Schönlein-Henoch purpura in adults

János Novák



Third Department of Internal Medicine, University of Debrecen Medical and Health Science Center (DEOEC), Debrecen, Hungary

INTRODUCTION

Heberden (1802) and Willan (1808) were the first to publish about the alteration which caused rash and oedematose arthritic pain. The disease, as an entity was written down and described by *Lucas Schönlein* (1832), he gave it the name 'Peliosis rheumatica'. Fourty years later one of his students, *Eduard Henoch* (1887) pointed out that in the case of this syndrome there was a connection between the dermatological manifestations and the gastrointestinal (GI) and renal symptoms. *Osler* (1914) suggested that anaphylaxis might have a role in the etiology of the syndrome, which was named 'anaphylactoid purpura' by *Frank* (1915) and *Glanzmann* (1916).

Since then 80 years have passed and numerous publications and studies were carried out in order to throw light on the exact etiopathology of the syndrom, here we have to mention the names of *Ruiter, Gougerot, Blum, Winklemann, Ditto, Wilkinson, Ramsay and Fry*. In the Hungarian medical literature, in 1974, *Boga* published an article about SHP in adults, he covered 6 cases. Me and my colleagues published articles covering more cases (n=73). In these we primarily dealt with extracutan digestive organic alterations and gastrointestinal haemorrhage, which are the symptoms of the abdominal type of the disease (so called Henoch purpura).

Exact epidemiologic data are not available, so we are not aware of the geographical and demographic characteristics.

The Schönlein-Henoch purpura is a well-known and defined pathography, it is a vasculitis, a non thrombocytopenic haemorrhagic syndrome, its origin is unknown but it is probably postinfectious. Circulating immune complexes containing IgA and C3 form deposits in the mucosus membranes of the skin, the stomatic-intestinal canal and joints, in the wall of the arterioles, teh capillary vesels and the venules and in the basal membranes of the renal glomeruli. In the subendothelic deposits polimerized immunoglobulin A1 (pIgA1), actived complements (C3,C5), fibrin/fibrinogen were found, and there are no IgG and IgM, wich are typical of Schönlein-Henoch purpura. It can be detected by immunfluorescence studies also in the vasculitis if the skin, the kidney and the intestines. Nobody managed to prove that

there was exogen antigene binding in the circulating complexes. According to some authors IgA plays a main part in the pathogenesis of SHP. IgA is divided into two subclasses: IgA1 and IgA2. In the vasculitis of SHP mostly pIgA1 is present, but the exact reason for this is unknown. The manifestations occur first on the skin symmetrically on the lower and upper limbs and on the abdomen wall. These manifestations are non thrombocytopenic, non traumatic, palpable purpuras, often associated with painful lesions in the joints, and renal, gastrointestinal, rarely pulmonary, cardiac symptoms.

The classification of systemic vasculitis is not a simple task, there are some possibilities to choose from also at the moment. The acceptance of the diagnostic criteria suggested on the consensual conference in Chapel Hill in 1990 made the diagnosis of the disease much more simpler and useful around the world. In 1992 Michel suggested a change in the diagnostic criteria referring to adults, this criterion system is the more often used now, and also we used this while working.

In the etiology of the disease numerous reasons may play a great part, among these primarily the antigens of microorganisms can be pointed out, although the exact causes are not clarified. The disease might emerge in a polyetiologic way, and eventually many agents may have great deal to do with it.

Gasbarini drew up that *Helicobacter pylori* infection can be possibly a cause of the emergence of Schönlein-Henoch purpura.

According to the facts written down previously I set the gastroenterologic analysis and treatment of the adulthood SHP patients as an aim, I mainly studied the importance of the endoscopic examinations and of the *Helicobacter pylori* in the development and in the course of the disease.

The purpose of this thesis was to:

1. Analyse the characteristics of patients suffering from adulthood SHP treated and cared between 1985 and 2001 in Békés County area, according to their gender, age and organ manifestations of the disease. Calculate the approximate incidence of the disease.

2. Show that the clinical course of childhood SHP differs from that of presenting in adulthood. Compare together with paediatrician colleagues the relative incidence of organ manifestations and prognosis of adulthood and childhood SHP.

3. Examine in detail the GI symptoms and changes during two periods, between 1985 -1999 in 62 patients and between 1999-2001 in 11 patients (altogether

73 patients) of adulthood SHP. Examine (on the base of data of 11 adult SHP patients) the immunological and gastroenterologic connections of this infection.

4. Analyse, whether *H. pylori* has any causal or accidental role in the development of SHP.

PATIENTS AND METODS

During two periods between 1985-1999 and between 1999-2001 we hospitalized and examined altogether 73 patients (62 in the first and 11 in the second period) suffering from Schönlein-Henoch purpura, who had turned to the Dermatological Department, the IIIrd Internal Medical Ward of the Pándy Kálmán Hospital or to the dermatological or gastroenterological consultation in Gyula.

The diagnoses were established according to the criteria set up by the American College of Rheumatology modified by Michel in 1992.

To the examination protocol we accepted patients older than 18 years, who complied with the diagnostic criteria, and who gave their consents, independently from their co-patients.

I did not accept those patients who had symptoms which did not comply with the ARC diagnostic criteria, or who did not have a complete documentation, or who did not give their consents. Once the acute phase was over first we controlled the examined patients every 3 months, then every year by physical and laboratory tests. To diagnose and treat GI haemorrhages we used the algorithm which was created by me.

Work-up data

I used the case-histories written during the treatment on the ward or during ambulatory treatment, which were on my disposal for the processing and for the collection of data. The processing was carried out on one hand from prospective and on the other hand from retrospective data. Anamnesis, physical examination, electrocardiogram, laboratory and radiological examinations and focus searching, if it was needed other instrumental examinations were carried out on every patient. By

21.9% of the patients (16 patient) skin biopsy was carried out. Each patient underwent panendoscopy (Olympus CV120) with 2-2 biopsies taken from the corpus, antral and duodenal mucosa and 1 from the antral and 1 from the corpus mucosa for a JATROX[®] or CLO test.

We also investigated the types of inflammations of duodenal and gastric mucosa, and the presence of infection with Hp. The histological sample was coloured with hematoxylin-eosine or modified Giemsa and very rare with Warthin-Starry. Bacterial infection was graded mild, medium and severe by the histologist.

On the anamnestic data none of the patients got eradication therapy because of Hp infection.

Before the endoscopy of the lower intestines we applied cathartics with laxative effect (X-prep solution or pulv. Macrogolum 1540). In each case we strived to carry out total colonoscopy, in some cases we made an examination with double contrast medium of the colon (irrigoscopy) or of the small intestines, and abdominal ultrasound, more rarely abdominal CT or MRI was carried out. The blood test of the stool was made in every patient.

Medicament induction or malignant disease was not confirmed among the causes of HSP, none of the patients had got vaccination in the previous 10 years.

Laboratory methods

Laboratory investigations were carried out at the central laboratory of Pándy Kálmán hospital. Immunological investigations were done at the Regional Laboratory of Immunology of the 3rd Department of Internal Medicine of DEOEC. During the routine investigations complete blood count, sedimentation (acc. to Westergreen (We), liver and kidney function tests were carried out, hepatitis A-B-C virus markers were determined and a complete urine analysis and cultivation of bacteria taken from the pharynx, sputum, urine and stool was carried out.

Serological investigations included the examining of anti-Helicobacter Pylori IgG and IgA antibody titres with the ELISA method (Biochem Immunosystem Italia S.P.A.). Besides, the antibodies were determined in the following ways: a: endomysium antibodies (EMA), liver-kidney microsome (LKM), mitochondrium (AMA), smooth muscle (SMA), gastric parietal cell antibodies (PCA) (BioSystems S.A. Spain), and also antinuclear factor (in Hep-2 cells), anti-neutrophil cytoplasmic antibodies (ANCA) wer determined with indirect immunofluorescent technique. b:

Gliadin (Diagnosticum Rt), cardiolipin and β 2-glycoprotein I (β 2GPI) (Calbiochem, UNILAB Rt.) and TNF- α (Becton-Dickinson, USA) antibodies were determined with the ELISA method. In the serum protein investigations the levels of anti-Streptococcus Dnase B, IgA, IgG, IgM, complement 3 (C3), complement 4 (C4), C-reactive protein (CPR) and immunocomplex (IC) were determined by nephelometry (Behring Nephelometer Systems). The total complement (CH50) was determined by hemolytic essay. AMA, LKM, PCA, gliadin, EMA, β 2GPI) showed pathogenic positivity. Anti Streptococcus DNase B was pathologically higher in one acute case and in one remittent case, both of which proved Hp positive.

Statistical analysis

In the statistical analysis we calculated average and standard deviation (average \pm SD). For the statistical evaluation of the divergences we used a Student test because of the low number of patients. The value of $p < 0.05$ was accepted as a significant deviance. The data were processed with Microsoft excel 7.0 or SYNSTAT 10 „descriptive statistic” programe.

RESULTS

1. THE DESCRIPTION OF THE ADULTHOOD SHP PATIENTS:

(division according to age, gender, incidence and organ manifestation)

According to the data of the anamneses from the 73 patients only 4 (5.4%) had previously SHP, and we cured or treated nobody steadily because of chronic SHP. 20 (27.3%) patient suffered from chronic internal organic diseases: hypertension, 17 (23.3%), chronic cardiac diseases, 11 (15%), chronic pulmonary disease, 3 (4.1%), diabetes, 3 (4.1%). Certainly there were superimpositions between the diseases, but substantially the internal organic disease of each patient was in balance with the treatment. The patients had been applying their medical treatments for years before.

We processed the statistic data of 73 adulthood SHP patient in the division of age, gender and organ manifestations. The number of the female patients

investigated was 41% (n:30), and that of the male patients was nearly 59% (n:43). The mean age was 60.5 ± 15 years (extremities: 30-86). There was no substantial difference in the age-group of the males and females. The typical dermatological manifestations developed in every patient. Biopsy was carried out in 16 (21.9%) patients, and in 13 cases the presence of leucocytoclastic vasculitis was proved. Renal manifestation occurred in 17 (23.3%) patients. More female were concerned than male. We noticed microscopic haematuria in 15, and non significant proteinuria in 5 cases. 27.3% of the patients had a symptom related to articular involvement. Arthralgia occurred in 20 patients, nearly proportionally in both genders. 14 patients proved to have monoarthritic lesions, while in 6 cases we diagnosed polyarthritis.

We found different lesions in the GI tract in 21 (28.7%) patients. It might be remarkable that abdominal manifestation occurred three times more often in males than in females. (Table 1.)

Table 1: Statistical data of adult patients suffering in Schönlein-Henoch purpura (1984-2001)

	Patiens n (%)	Female n(%)	Male n (%)
<i>Patiens number</i>	73	30 (41,1%)	43 (58,9%)
Age (year; average \pm SD)	60,5 \pm 15	63,3 \pm 10	58,7 \pm 15
<i>Organ manifestations</i>			
Skin	73 (100%)	30	43
Ren	17 (23,3%)	10	7
Joint	20 (27,3%)	11	9
Gastrointestinal tract	21 (28,7%)	5	16

2. THE COMPARISON OF THE ORGANIC MANIFESTATIONS OF CHILDHOOD AND ADULTHOOD SHP

The documentation of 56 children suffering from childhood SHP was processed. Their mean age was 6.62 ± 3.4 years (extremities: 2-15 years), there were 34 girls (60.7%) and 22 boys (39.3%). Like in adults also in children every patient had the typical dermatologic manifestations. Renal manifestation developed in 9 patients, which means that it was less frequent than in adults (n: 12) (16% vs.

19.3%). In 8 patients we detected microscopic haematuria, and in one case macroscopic haematuria. The patient was taken care of, and is now in permanent remission. We did not find marked proteinuria among the patients. The number of the alterations in joints was more than twice as much as in adults (55% vs. 22.6%). 20 (64.5) children had dominantly polyarthritic lesions, while among adults monoarthritic manifestations were more typical (n:10, 71.2%). GI manifestations occurred more often than in adults (35.7% vs. 24.2%), but these proved to be more benign than in the older age-groups. Massive haemorrhage in the lower tract was noticed once, during the endoscopic examination from the sample from the purpura, the histologist detected vasculitis.

3. NEWER DIAGNOSTIC METHODS , WHICH ARE TURNING OUT MORE EFFICIENT IN THE DIAGNOSIS OF SHP PATIENTS' ABDOMINAL MANIFESTATIONS

3.1 Abdominal manifestations in adult Schönlein-Henoch purpuric patients

From the beginning of 1985 till the end of 1999 I examined from the altogether 62 adult SHP patients 15, who had abdominal purpura. In the 24.2% of all the adult patients abdominal manifestations emerged. Abdominal pain occurred in 86.6% of the patients having abdominal purpura, mainly it was a periumbilical, colic pain, usually 3-4 days after the development of the skin manifestations. In the two third of the digestive manifestations haemorrhage appeared in the GI tract, most frequently it was an occult haemorrhage. In our cases it mounted up to the total haemorrhage's 53.3%. Vomiting, nausea occurred in 40% of the cases. 20-30% of the patients had diarrhoea. Once chirurgic intervention was needed, because of peritonitis and socketed organic perforation. We noticed peritoneal excitation two times, but the examinations carried out did not show perforation or lesions that could have indicated laparotomy. Both patients recovered thanks to the conservative therapy, the cause of the manifestations proved to be pseudoperitonitis. (Table 2)

Table 2: Comparing abdominal manifestations of own adult Schönlein-Henoch purpuric patients data with literary data

Clinical data	Own data(n:15) n(%)	Literary data %
Abdominal manifestations	15/62 (24,2)	29–69
Abdominal pain	13/15 (86,6)	~80
Melaena	3/15 (20)	~50
Haematochezia	2/15 (13,3)	?
Occult bleeding	8/15 (53,3)	~25
Emesis	6/15 (40)	~40
Diarrhoea	3/15 (20)	~25
Peritonitis	1/15 (6,6)	?
Pseudoperitonitis	2 (13,3)	?
Pancreatitis	0 (0)	?
Intestinal perforation	1/15 (6,6)	?
Invagination	0 (0)	?

3.2 The role of endoscopy in the diagnosis of abdominal purpura

Between 1985 and 1999 we carried out endoscopic examinations in altogether 26 adult SHP patients. In 21 cases upper panendoscopy, while in 18 cases lower endoscopic examination was also carried out. We found abdominal manifestations in 28.7% of all the adult SHP patients (n:73). 13 patients suffered from abdominal pain, and 13 had colorectal haemorrhage, from which 3 had massive malaena and 8 had occult haemorrhage. In the oralcavity, oesophagus and ventricle we did not find purpuriform lesions during the endoscopies, while in the deep duodenum we found it in 3 cases. We detected oesophagitis in the upper GI tract once in the 26 patients we examined (Savary-Miller I., etc). In the stomach according to the endoscopic picture we found altogether 18 gastritis, in 14 cases erosion, erythema, focal haemorrhage. In 2 patient we saw ulcer in the antrum, 5 patients proved to be infected by Hp.

In the small intestine in 5 cases duodenitis, in 8 erosion, petechia, erythema and in 3 patients purpuriform lesions were found. Ulcer in the duodenum did not occur. Histologic diagnosis: acute, non-specific gastritis with focal haemorrhage: 12, chronic, non-specific gastritis: 8, duodenitis showing signs of mild activity: in 5 cases, in 1 case vasculitis, in 2 cases the presence of ulcer, in other 2 cases intact mucosa was proved. Malignant lesion was not found in the patients.

3.3 The diagnostical importance of traditional or 'big particle' biopsies from the mucosa of the colon

The vasculitises of the gastrointestinal tract develop in the small vessels of the submucosa, so during endoscopical sampling superficial mucosa biopsy is usually insufficient to detect vasculitises. In such cases the histologist writes in their description acute non-specific inflammation, or maybe focal haemorrhage. In 6 of the 11 patients who underwent colonoscopy superficial biopsy was made, in these cases vasculitis was not proved. From the purpuras in the colon of other 5 patients the sampling was carried out with 'big particle' biopsy, in 3 of these vasculitis was found. In these cases the hitologist wrote down the parcial fibrinoid necrosis of the small vessels, with the infiltration of mononuclear and polymorpfonuclear leukocytes and the extravasation of the erythrocytes.

4. ELEVATED LEVEL OF HELICOBACTER PYLORI ANTIBODIES IN HENoch-SCHÖNLEIN PURPURA

Between 1995 and 2000 we had 11 HSP PATIENTS aged 64 ± 10 years (extreme values: 36-81), out of them 5 were female and 6 were male. It is worth noting the old age of the patients as HSP mainly occurs in childhood (30). The 5 patients in the acute phase had characteristic palpable skin purpura, 3 patients had gastrointestinal symptoms. The panendoscopic examination of the 5 patients revealed 4 cases of gross change in the stomach or in the duodenum. The 6 patients in the remittent phase had no more skin symptoms. 3 patients had abdominal pains, the endoscopy revealed lesions in the upper tract in 4 patients. None of them developed renal failure during the period. (Table 3)

During the routine laboratory investigations the acute patients showed a significantly higher leukocyte count than those in remission (12.7 ± 1.05 vs. 7.62 ± 1.9 G/L) ($p < 0.00171$). One patient showed explicit anaemia (94g/l) in the acute phase, in this case gastrointestinal haemorrhage was confirmed. The acute patients did not show a significantly higher We.value (83.4 ± 8.8 vs. 23.5 ± 8.8 mm/h) ($p = ns$) than remittent patients. Hepatitis infection was not confirmed. In the cultivation of bacteria one patient in Group 2 had urinary infection (*Acinobacter calcoaceticus*). (Table 4)

Table 3. Skin symptoms, abdominal pains and the occurrence of Helicobacter Pylori infections in adult HSP patients

HSP Subsets	Sex	Age	Skin symptoms	Abdominal pain	Urease test (Jatrox-Hp®)	Endoscopic diagnosis	Histology for Hp	Hp serology (IgA,IgG)
Acute phase (n=5)	female	81	+	-	-	Gastritis	Negative	+
	male	38	+	+	+	Gastric ulcer	Positive	+
	male	70	+	-	-	Sine morbo	Negative	+
	male	53	+	+	+	Gastritis	Positive	+
	male	54	+	+	+	Duodenitis	Negative	+
Remittent phase (n=6)	female	69	-	-	-	Gastritis	Negative	+
	male	74	-	+	-	Gastritis	Negative	+
	male	65	-	+	+	Gastritis	Positive	+
	female	81	-	+	-	Gastritis	Negative	+
	female	53	-	-	-	Sine morbo	Negative	+
	female	65	-	-	-	Duodenitis	Negative	-

Table 4.:The level of anti Helicobacter Pylori antibodies (IgG-IgA), serum IgA, TNF- α and C-reactive protein in patients with acute and remittent HSP.

	Anti Hp IgG	Anti Hp IgA	Serum IgA	TNF- α	CRP
Group 1(n=5)	86,04 \pm 32 *	1,96 \pm 0,58	5,54 \pm 1,08*	58,88 \pm 18,2***	45,30 \pm 22,7*
Group 2(n=6)	32,51 \pm 23,2	3,09 \pm 1,78*	3,49 \pm 1,14	27,30 \pm 5,2	8,08 \pm 4,9
Total (n=11)	57,80 \pm 32,9	2,58 \pm 1,27	4,43 \pm 1,42	42,10 \pm 20,1	25,01 \pm 13,7
Normal value	0-15 U/ml	0-0,9 ratio	0,7-4 g/l	0-42 pg/ml	0-5 mg/l

Notes :* significant difference between the acute and the remittent phases ($p < 0,05$),
 **($p < 0,01$),
 *** ($p < 0,001$)

The most significant differences between acute HSP (Group 1) and remittent (Group 2) patients were found in the levels of anti-Hp IgG-IgA, serum IgA, CRP and TNF- α . In acute patients the anti-Hp IgG titres were significantly higher than in those in remission (86.04 \pm 32U/ml vs. 32.5 \pm 23.2U/ml) ($p < 0.0483$). On the other hand, the levels of anti-Hp IgA in remittent patients were The values of circulating IgG were in both groups in the normal range. The total complement level was increased slightly only in one case, in the case of an acute patient. (102CH50/ml). None of the further antibody investigations (C3-C4, IC, ANCA, ANF, SMA, AMA, LKM, PCA, gliadin, EMA, β 2GPI) showed pathogenic positivity. Anti Streptococcus DNase B was pathologically higher in one acute case and in one remittent case, both of which proved Hp positive.

DISCUSSION

The description of the adult SHP patients

In Hungary, in the Békés County area according to our data the incidence of SHP is higher (11.20/1 million), than, what we find out from the data of the medical literature, in the countries which are in a better socioeconomical position. The 41% (n:30) of the SHP patients is female, the number of male patients is 59% (n:43). The mean age is 60.5 \pm 15 years (extremities: 30-86 years) It is interesting that in adults the dominance of males is one and a half times greater than females, while the disease developed three times more often in girls. In adults the arthritic

manifestations were decreased significantly (88% vs. 22.5%), the GI manifestations dropped from 94% to 19%, the renal manifestations lessened from 80% to 19.3% and the chronic renal diseases from 30% to 2%. It is prominently important, that the changing of renal manifestations into chronic forms decreased the most significantly. In my opinion the cause of the decreasing tendency in the serious organic lesions are the improving medical treatment, the chance for a faster and more exact diagnosis and the modern and more efficient opportunities in therapy.

The comparison of the organic manifestations of childhood and adulthood SHP

In children the symptoms are similar to the symptoms of the adults, although the prognosis and the course of the disease might be milder. In the adult cases arthritic and GI manifestations occur more often, but in most cases renal complications with serious outcomes are less frequent than in children. Concerning the results we have to draw the attention of the pediatrician society to the importance of the qualification of pediatricians able to use endoscopes, since this disease occurs rather often during the childhood and carrying out endoscopic diagnosis is expedient and essential in these cases.

Newer diagnostic methods, which are turning out more efficient in the diagnosis of SHP patients' abdominal manifestations

The abdominal manifestations of the adult SHP patients

From the abdominal manifestations in our cases abdominal pain occurred in most patients, we detected it in 86.6% of them. In the two third of the digestive manifestations haemorrhage occurred in the GI tract. It was usually occult haemorrhage, it counted up for 53.3% of all the haemorrhages, massive malaena was not rare either, but we did not find any haematemesis. We could use the algorithm of examination and treatment, which was recommended by us, very efficiently, according to what is previously written down all are patients got to remission and we did not have to operate anybody.

Judging by my experience I drew the conclusion that the seriousness of the extensive lesions on the skin does not reflect the seriousness of the manifestations in the parenchymatous organs, but the explicit parenchymatous manifestations influence the prognosis of the syndrome.

The role of endoscopy in the diagnosis of abdominal purpura

On the basis of the facts mentioned to choose widespread diagnostics is important, as there are no specific examinations for this disease. In the case of

abdominal manifestations endoscopic examination methods and complementary radiological techniques become conspicuous. According to my results gastrointestinal endoscopy might be described as 'gold standard' in GI haemorrhagic cases and also in connection with other lesions of the gastrointestinal tract, SHP. I can point out that on the endoscopic pictures of the upper GI tract along with the inflamed mucosa, erythema, oedema, punctiform petachiae, erosions, in more serious cases ulcer (soliters or multiplexes) can be found, but typical purpuriform lesions can be noticed only in the descending part of the duodenum. It is important to know that the alterations in the duodenum have a close relation with SHP. On the mucosa of the colon 'spectacular' purpuras can be seen very often, they can develop in the whole colon and in the terminal ileum, they are often segmentary, but can also be diffuse, their shape varies from circular to bizarre shapes. In the case of abdominal purpura, especially if there is haemorrhage, deep duodenoscopy and total colonoscopy are indicated in order to find the exact place of the blood-source.

The diagnostical importance of traditional or 'big particle' biopsies from the mucosa of the colon

Our results show that after carrying out 'big particle' biopsy primarily from the purpuras of the colon there is a bigger chance to detect leukocytoclastic vasculitis in the more profound regions of the submucosa.

Elevated level of Helicobacter pylori antibodies in Henoch-Schönlein purpura

Based on our investigations of our 11 patients, serological investigation revealed 10 cases of Helicobacter Pylori infection.

Our results indicate a firm parallel between HSP and Hp in elderly adult patients. One reason for this is that 6 of the 11 patients had abdominal symptoms and the panendoscope confirmed 9 cases of gastric lesions. On the other hand, 10 patients of the 11 showed a high increase of Anti H.pylori IgG/IgA. This association raises the question whether Hp can have an etiological role in the pathogenesis of HSP. In our view it may have a role in the complexity or aggravation of HSP symptoms, but in triggering or developing the disease it can only be a possible but not sufficient factor. However as a risk factor it may contribute to sustaining the acute symptoms of HSP. We presume that during the acute HSP-characteristic immune

processes the already existing Anti H.pylori immune reaction is increased and this may be caused by anti-idiotypic antibodies which are similar to Hp antigens and increase the development of Anti Helicobacter Pylori antibodies on the basis of antigenic mimicry. On the other hand, the existing Hp. may get intensified in the damaged gastric mucosa because of the weak immune system and reduced antibacterial protection of the patient, which, in turn may activate HSP in the manner of a *vicious circle*. Our own results also suggest this, since acute HSP patients had a significantly higher level of Anti-H.PyloriIgG than those in remission, whereas in the latter group Hp could turn chronic in line with the increase of Anti-H.pylori IgA. This, however, does not intensify the symptoms.

Summary

1/ Analysing the specific features of 73 adult patients suffering from SHP, it could be found that there were no differences in the characteristics of clinical symptoms, as compared with those known from international literature. Compared to the data obtained in the seventies, a significant increase in average age could be stated (45 v. 60,5 years). The ratio between genders did not change. In Békés county the approximate annual incidence -taking into consideration the data of the Central Statistical Office – is around 11, 20 cases/ 1 million population. The frequency of grave extracutaneous organ manifestations has significantly decreased : articular manifestations (80%versus22,5%), gastrointestinal (GI) symptoms (94% v.19%), renal manifestations (80%v.19,5%) and the occurrence of chr.renal insufficiency were also decreased (30%v.2%).

2/ Through this work it is demonstrated that in Békés County the articular manifestations of childhood SHP are two fold more frequent, than those in adulthood (55 v. 24,2%), and mainly the polyarticular localization prevails. Often are observed the symptoms of GI tract (35,7 v.24,2%), however the clinical course of these is significantly more favourable. Of the more frequently occurring upper GI symptoms the incidence rate of vomiting is higher, than that of GI bleeding.

3/ On the base of the investigations performed, it has been proved that in abdominal purpura the lower and upper endoscopy assures topographic and descriptive diagnosis while during the procedure histological sampling and microsurgical intervention are also possible facilitating by this the establishment of an accurate diagnosis and differential diagnosis. In this work there were been characterized and described - in Hungary for the first time- the endoscopic view of the SHP lesions presenting in the upper and lower GI tract.

It was drawn the attention to the typical purpuriform lesions presenting primarily in the descending part of the duodenum, and to the fact that these duodenal changes in close connection with the existence of SHP. Every effort has to be made to perform total colonoscopy and survey the terminal ileum. In the case of abdominal purpura , especially when bleeding also occurs deep, duodenoscopy and total colonoscopy is justified.

On the base of our investigations we were the first in the literature who called the attention to the possibility of a new investigating method the so called „big particule biopsy” by which the leukocytoclastic vasculitis was successfully detected not only in resected intestinal specimens but also in in vivo conditions from deeper submucosal layers of the intestine .

4/ On eleven adult patients was investigated that the link between SHP and HP infection whether is causal or accidental. According to the results obtained our standpoint is, that the HP infection could have a role in the modulation or aggravation of SHP symptoms, however in the triggering and formation of the SHP, it could be one of the possible but in itself not sufficiently adequate pathogenetic factor. As a risk factor, however , could contribute to the maintaining of SHP's acute symptoms.

List of publications, abstracts and lectures

1. Publications related to the theme:

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6. Novák J., Márki-Zay J., Csiki Z., Sebesi J., Takáts A. und Sipka S.: Die Schönlein-Henoch Purpura bei Erwachsenen Z. Gastroenterol ;2001;39:775-782 IF:0,803
7. Novák J., Csiki Z., Sebesi J., Takáts A., Demeter P. és Sipka S.: Helicobacter pylori ellenes antitestszint emelkedés Schönlein-Henoch purpurában. Orv. Hetil. 2003;144:6,7-11.
8. Novák J., Szekanecz Z., Csiki Z., Sebesi J., Takáts A., Bene L., Demeter P. és Sipka S.: Elevated level of Helicobacter pylori antibodies in Schönlein-Henoch purpura. Autoimmunology(közlésre elküldve) IF:folyamatban

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MGT. 44. Nagygyűlés, Balatonaliga 2001 Június 04-08.

Curriculum Vitae

PERSONAL PARTICULARS

FAMILY NAME: DR NOVÁK

CHRISTIAN NAME: JÁNOS

DATE OF BIRTH: 25 SEPTEMBER 1959

PLACE OF BIRTH: KÉTEGYHÁZA

CITIZESHIP: HUNGARIAN

PERMANENT ADDRESS: GYULA 5700

ERNYŐ STR 9/A

HUNGARY

TELEPHONE: (HOME) 36 66 361-008 Mo: +36 30 9950 319

(HOSPITAL) 36 66 361 833/2265 Fax:36 66 468-379

MARITAL STATUS: MARRIED, FATHER OF TWO

QUALIFICATION: SPECIALIST OF INTERNAL MEDICINE AND GASTROENTEROLOGY

PRESENT POSITION: HEAD PHYSICIAN OF 3rd DEPT. OF INTERNAL MEDICINE

PLACE OF WORK: PÁNDY KÁLMÁN COUNTY HOSPITAL, 3rd DEPARTMENT OF INTERNAL MEDICINE/GASTROENTEROLOGY
H-GYULA 5700, SEMMELWEIS STR.1

EDUCATION:

I took my school-leaving exams at academic grammar school in 1978. In that year I was admitted to the Szentgyörgyi Albert University of Medicine in Szeged. I got my degree as a doctor in 1984. I began working 1984 . First 5 years I studied endocrinology and diabetology and in 1989 I got my degree as a specialist of internal medicine. From 1989 I have been working in the Department of Gastroenterology as a specialist of gastroenterology , I got my degree specialist of gastroenterology in 1992. I have had 18 years of experience of internal medicine and gastroenterology. In present I am employed as head physician of 3rd dept. of Internal Medicine. I can speak two languages ,English and Rumanian and a little in Italian. I obtained a certificate of intermediate level in English and advanced level from Rumanian . I am member of Hungarian Society of Gastroenterology and Internal Medicine, member of medical section of South Great-Plane Academy. 2002 I took GCP (PRA International) exam. My plan in the future (maybe in this year), to get my degree of PhD.

DATE: GYULA, 2003-06-17

SIGNATURE

