

Gallbladder and Pancreas in Henoch-Schönlein Purpura: Review of the Literature

*Rossana Helbling, *†Sebastiano A.G. Lava, *†Giacomo D. Simonetti, *Pietro Camozzi, *Mario G. Bianchetti, and ‡Gregorio P. Milani

ABSTRACT

Objective: Involvement of the pancreato-biliary system has been occasionally noted in Henoch-Schönlein purpura. Furthermore, cases of this vasculitis syndrome sometimes develop in the context of a viral hepatitis or after hepatitis vaccination.

Methods: We completed a review of the literature.

Results: Fifty reports published between 1977 and 2015 were retained for the analysis. A pancreato-biliary involvement was recognized in 34 individually well-described patients (♂:♀ = 19:15) with severe abdominal pain: pancreatitis (N = 20), acalculous cholecystitis (N = 11), both pancreatitis and cholecystitis (N = 3). In all of the pancreatitis patients, full recovery occurred (within ≤ 3 weeks in three-fourths of the patients). Cholecystectomy was performed in 8 cholecystitis patients. Seventeen Henoch-Schönlein patients (♂:♀ = 9:8) were associated with a viral liver disease and 4 (♂:♀ = 1:3) with a hepatitis vaccination. The vasculitis syndrome rapidly remitted in the 7 patients accompanying hepatitis A or E, in 2 patients of hepatitis B, and in the 4 patients preceded by a vaccination. Henoch-Schönlein purpura seemed to be serious in 5 patients with chronic hepatitis B and in 3 with chronic hepatitis C.

Conclusions: This analysis indicates that pancreato-biliary involvement is unusual in Henoch-Schönlein purpura. This complication deserves consideration in patients with especially severe abdominal pain. Finally, viral hepatitides and hepatitis vaccinations seem to be rare triggers of Henoch-Schönlein purpura.

Key Words: acalculous cholecystitis, Henoch-Schönlein purpura, hepatitis, kidney disease, pancreatitis, vaccination

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Henoch-Schönlein purpura, also known as immunoglobulin A leukocytoclastic vasculitis, is usually triggered by an infectious disease or, more rarely, a vaccination (1). This condition characteristically presents with palpable purpura concentrated in dependent areas, arthralgia or arthritis, abdominal pain, and kidney disease (1).

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From the *Pediatric Department of Southern Switzerland, Bellinzona, the †Department of Pediatrics, University Children's Hospital, Bern, Switzerland, and the ‡Foundation IRCCS Ca' Granda, Ospedale Maggiore Policlinico, Pediatric Emergency Department, Milan, Italy.

Address correspondence and reprint requests to Mario G. Bianchetti, San Giovanni Hospital, 6500 Bellinzona, Switzerland (e-mail: mario.bianchetti@pediatrician.ch).

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What Is Known

- Case reports document the involvement of bilio-pancreatic system in Henoch-Schönlein purpura.
- Henoch-Schönlein purpura occasionally develops in the context of viral hepatitides.

What Is New

- Bilio-pancreatic involvement is benign and self-remitting in ≈ 3/4 of Henoch-Schönlein patients. Subjects with bilio-pancreatic involvement are older (5–55 years) than those (3–15 years) without.
- Viral hepatitides may be rare triggers of Henoch-Schönlein purpura. In these patients, prognosis of Henoch-Schönlein purpura is determined by the severity of the underlying liver disease.

Pancreatitis and acalculous cholecystitis are unusual but recognized complications of abdominal vasculitides. Unsurprisingly, therefore, involvement of the pancreato-biliary system has been occasionally noted in Henoch-Schönlein purpura. Moreover, cases of this vasculitis sometimes develop either in the context of an acute or chronic viral liver disease or after hepatitis A or B vaccination. Because textbooks and reviews only marginally mention these associations, we reviewed and analyzed the available literature.

METHODS

Between February and May 2015, we performed a computer-based search with no date limits of the terms (Henoch OR Schönlein OR Henoch-Schönlein OR Schönlein-Henoch OR anaphylactoid purpura OR rheumatoid purpura OR rheumatoid angiitis OR vasculitis) AND (liver OR hepatic OR biliary OR pancreatic OR hepatitis OR vaccination) in the search engine PubMed. In addition, we used our personal files and the bibliography of each identified study. We applied the principles established by the Economic and Social Research Council guidance on the conduct of narrative synthesis and on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses statement (2). For the final analysis, we selected peer-reviewed reports published as full-length article or letter, which include Henoch-Schönlein patients with pancreas, gallbladder, or liver involvement. Cases of this vasculitis syndrome associated with a viral liver disease or preceded by a vaccination against hepatitis A or B infection were also selected. We exclusively retained well-documented original cases presenting subjects of both

sexes and all ages irrespective of follow-up duration, which had been published in Dutch, English, French, German, Italian, Portuguese, or Spanish. When >1 article reported on the same patient, only the more comprehensive article was included and referenced.

The diagnosis of Henoch-Schönlein purpura, pancreatitis, and cholecystitis established in the original publications, which were often supported by figures depicting skin lesions and imaging studies, was reviewed using recognized criteria. The diagnosis of Henoch-Schönlein purpura was based on the European League Against Rheumatism/Paediatric Rheumatology International Trials Organization/Paediatric Rheumatology European Society criteria (3). The latter include palpable purpura together with at least 1 of the following findings: abdominal pain, leukocytoclastic vasculitis with immunoglobulin A (IgA) deposits, acute arthritis, or arthralgia in any joint and renal involvement as evidenced by pathological urinalysis, with or without a biopsy disclosing mesangial IgA deposits (3). The diagnosis of pancreatitis was made in patients with abdominal pain and amylase or lipase level at least twice the upper limit of normal in the absence of alcohol abuse, gallstones, or prescription of drugs implicated as causing pancreatitis (4). The diagnosis of acalculous cholecystitis was made in patients with right upper quadrant abdominal pain and imaging studies disclosing a significant (≥ 4 mm) gallbladder wall thickening (with or without fluid collection around the gallbladder) in the absence of alternative diagnoses such as calculous cholecystitis (5). The diagnosis of hepatitis A, B, or C was made using standard laboratory techniques. Henoch-Schönlein cases associated with infectious mononucleosis, primary biliary cirrhosis or alcohol abuse, as well as with documented causes of pancreatitis or cholecystitis, were excluded.

From each report dealing with Henoch-Schönlein purpura and pancreato-biliary involvement or viral liver disease or vaccination, we excerpted data on sex, age, presence of abdominal pain preceding the purpura, aminotransferase levels, kidney disease, course, and management. The data were extracted by 2 investigators (R.H. and S.A.G.L.) independently and a consensus was reached on all items. Disagreements were resolved through discussion or adjudicated by a third author (G.P.M.).

The kidney disease was classified as absent in patients with normal urinalysis, mild in patients with hematuria and urine protein/creatinine ratio < 200 g/mol, moderate in patients with hematuria and protein/creatinine ratio > 200 g/mol, and severe in patients with protein/creatinine ratio > 200 g/mol, hypoalbuminemia (< 25 g/L), and pitting edema (6).

Results are given either as frequency or as median and interquartile range. The Fisher exact test was used to compare dichotomous variables and the Mann-Whitney-Wilcoxon rank-sum test to compare continuous variables. Statistical significance was assigned at $P < 0.05$.

RESULTS

Search Results

A total of 50 peer-reviewed scientific reports (7–57) published between 1977 and 2015 in English (N=41), French (N=4), German (N=2), Spanish (N=2), and Italian (N=1) were retained for the final analysis (Fig. 1). They had been reported from the following continents: Asia (N=22), Europe (N=16), North America (N=8), Africa (N=3), and Australia (N=1). The reports included 34 well-documented Henoch-

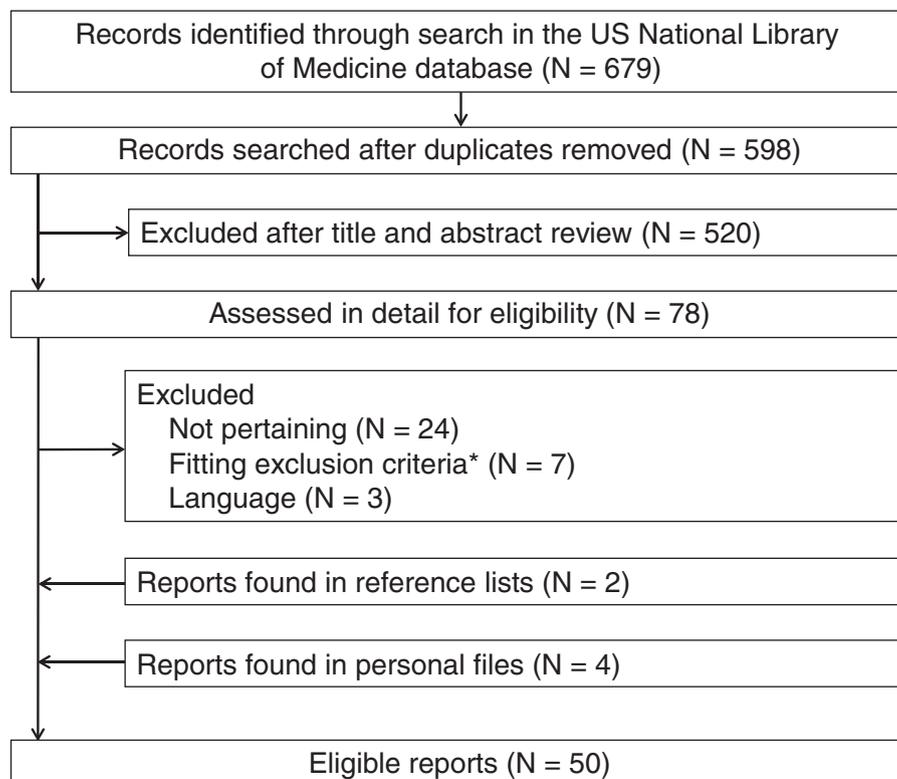


FIGURE 1. Pancreas, gallbladder, and liver in Henoch-Schönlein purpura. Flowchart of the literature search process. *Cases associated with infectious mononucleosis (N=1), primary biliary cirrhosis (N=1), or alcohol abuse (N=2); same case reported in >1 article (N=2); poorly described patients (N=1).

TABLE 1. Characteristics of 34 Henoch-Schönlein patients with bilio-pancreatic involvement

	All	Pancreatitis	Cholecystitis	Pancreatitis + cholecystitis
N	34	20	11	3
Age, y	15 (7–43)	17 (8–47)	8 (5–35)	6, 8, 30*
Sex, ♂: ♀	19: 15	11: 9	6: 5	2: 1
Abdominal pain precedes purpura, N (%)	14 (41)	11 (55)	2 (18)	1 (33)
Aminotransferases elevated, N (%)	11 (32)	5 (25)	4 (36)	2 (67)
Kidney disease, N (%)	17 (50)	10 (50)	5 (45)	2 (67)
Mild	8	3	4	1
Moderate	3	3	0	0
Severe	6	4	1	1

Results are given as either frequency or median and interquartile range (7–38).

* Individual values.

Schönlein cases associated with pancreato-biliary involvement (7–38), 17 patients associated with a viral liver disease (39–52) and 4 preceded (53–56) by a hepatitis vaccination. Using the aforementioned inclusion criteria, we always confirmed the diagnosis of Henoch-Schönlein purpura, pancreatitis, or cholecystitis established in the original publications.

Pancreato-Biliary Involvement

Pancreatitis (7–25) was recognized in 20, acalculous cholecystitis (26–35) in 11, and both pancreatitis and cholecystitis (36–38) in 3 individually well-described patients. A kidney disease was noted in 17 of the 34 patients (Table 1). Patients with kidney involvement were significantly older by 21 years than those without (28 [16–52] vs 7 [5–12] years, $P < 0.01$). Abdominal pain, which was associated with arthralgia or arthritis in 8 patients, preceded the appearance of purpura by ≤ 2 weeks in 14 of the 34 Henoch-Schönlein patients with pancreato-biliary involvement.

Pancreatitis

In addition to the increased amylase or lipase tests, the pancreas appeared diffusely enlarged and hypoechoic on abdominal ultrasound in 8 of the 20 pancreatitis patients. Peripancreatic fluid and bowel wall thickening were noted in 7 and in 5 patients, respectively. Finally, cardiac failure (19) and acute respiratory distress syndrome (11) were observed in each 1 patient. Symptomatic and supportive management was provided in all patients. Steroids were given in 7 patients. In all of the patients, full recovery occurred (within ≤ 3 weeks in 3/4 of the patients). It is true, however, that in 2 patients the clinical remission was observed 3 to 4 months after the diagnosis.

Cholecystitis

Cholecystectomy (open surgery, $N=6$; laparoscopic surgery, $N=2$) was performed in 8 of the 11 patients affected with cholecystitis. The intraoperative diagnosis of gallbladder perforation with biliary peritonitis was made in 1 patient (34). Supplemental bile duct stenosis leading to severe secondary cholestatic liver disease was noted in 1 further patient (31). Finally, cholecystitis was associated with intussusception in 1 patient (35).

Cholecystitis and Pancreatitis

None of the 3 patients with concurrent involvement of gallbladder and pancreas underwent surgery. In 1 of these patients,

a ureteric obstruction was also observed (36). The bilio-pancreatic involvement resolved after ≤ 10 days in 2 and approximately after 4 months in the patient with ureteric obstruction (steroids had been given in this patient).

BIOPSY STUDIES

A skin biopsy, performed in 12 patients with pancreatitis, in 3 with cholecystitis, and in 2 with both cholecystitis and pancreatitis, showed a leukocytoclastic vasculitis with IgA deposits. A kidney biopsy, performed in 7 patients with pancreatitis, revealed mesangial IgA deposits. Histopathology of gallbladder, performed in 7 patients with 2, revealed a leukocytoclastic vasculitis in 4 patients and the distinctive signs of cholecystitis in the remaining 3 patients.

Henoch-Schönlein Purpura Associated With a Viral Liver Disease or Preceded by Hepatitis Vaccination

The literature included also 17 Henoch-Schönlein cases associated with an acute or chronic viral liver disease (39–52) and 4 preceded (53–56) by a hepatitis vaccination (Table 2).

Henoch-Schönlein purpura spontaneously resolved within ≤ 4 weeks in the 7 cases accompanying hepatitis A or E and in the 4 cases preceded by vaccination against hepatitis A or B. No kidney disease was observed in these 11 patients.

Henoch-Schönlein purpura spontaneously resolved without any kidney disease in 5 of 7 hepatitis B patients. These patients also rapidly and spontaneously recovered from the liver disease. A severe respectively a moderate kidney disease occurred in each 1 of the 2 remaining patients, who were affected with a chronic

TABLE 2. Cases of Henoch-Schönlein syndrome associated (40–57) with a viral liver disease ($N=17$) or preceded by hepatitis A or B vaccination ($N=4$)

Trigger	N	Age*	Sex (♂: ♀)
Viral liver disease			
Hepatitis B	7	3.0, 6, 8, 13, 23, 32, 40	2: 5
Hepatitis A	6	2.7, 8, 10, 10, 11, 13	4: 2
Hepatitis C	3	50, 63, 56	3: 0
Hepatitis E	1	6	0: 1
Vaccination			
Hepatitis A	2	1.6, 24	1:1
Hepatitis B	2	21, 28	0:2

* Individual values.

hepatitis B (no follow-up information is available for these patients).

In the 3 patients with chronic hepatitis C (2 of them complicated by liver cirrhosis), Henoch-Schönlein purpura presented in each 1 patient without, with mild or with moderate kidney disease. The long-term prognosis was predicted by the underlying liver disease.

DISCUSSION

The present review indicates that in Henoch-Schönlein purpura there is rarely a clinically relevant pancreato-biliary involvement. Furthermore, this condition is sometimes triggered by a viral liver disease or preceded by a vaccination against hepatitis A or B.

It is assumed that the prognosis of Henoch-Schönlein purpura is essentially determined by the severity of the accompanying kidney disease (1,6). This impression is supported by the present analysis: in $\approx 3/4$ of the Henoch-Schönlein patients with pancreato-biliary involvement the disease was benign and self-remittent.

Ninety percent of Henoch-Schönlein cases occur between the ages of 3 and 15 years (1), cases with pancreato-biliary involvement between the ages of 5 and 55 years. Recommended management of pancreatitis includes proper fluid management, pain relief, and nutritional support (4), that of acalculous cholecystitis surgery and antibiotics (5). We speculate that steroids as well deserve consideration in cholecystitis or pancreatitis that develops in the context of a vasculitis syndrome. All the more so because in Henoch-Schönlein purpura steroids effectively treat abdominal pain, which mostly results from bowel wall swelling and bleeding.

In a retrospective case series from the Republic of China including 225 apparently unselected children with Henoch-Schönlein (57), imaging studies disclosed abnormalities of the biliary system in 14 (6%) of them: gallbladder wall thickening ≥ 4 mm (N = 7), sludge (N = 5), gallbladder dilatation (N = 1), and bile duct dilatation (N = 1). These data, which deserve further confirmation, point to the possible existence, in some Henoch-Schönlein patients, of gallbladder abnormalities that may predispose to acalculous cholecystitis.

Henoch-Schönlein cases often develop after an upper respiratory infection (1). Further infectious triggers include Epstein-Barr virus, *Helicobacter*, *Legionella*, *Mycoplasma*, Parvovirus, Varicella zoster virus, or *Yersinia* (1). Cases also have been reported following vaccination (1). The present analysis indicates that viral hepatitis and vaccination against hepatitis A or B may be further possible triggers of this vasculitis. The number of reported Henoch-Schönlein patients temporally associated with viral hepatitis or vaccination against hepatitis is limited. It is true, however, that most vasculitides associated with hepatitis or vaccinations are “non-Henoch-Schönlein vasculitides” (58). Henoch-Schönlein purpura that develops after vaccination against hepatitis or in the context of hepatitis, which usually rapidly resolve on their own, is benign and remits soon. On the contrary, cases that develop in the context of chronic hepatitis B and C may be serious. In the latter patients, the long-term prognosis is essentially determined by the severity of the underlying liver disease.

Some limitations of this work should be mentioned. First, it results from the small number of reported patients, often without or with a brief follow-up. Second, diagnostic and therapeutic recommendations arise from authors' opinions. Third, data on liver involvement in patients not triggered by a viral liver disease are inconsistent. Fourth, the relation between viral liver diseases respectively vaccinations against hepatitis A or B and Henoch-Schönlein purpura is so far undemonstrated.

In conclusion, this analysis indicates that pancreato-biliary involvement is unusual in this vasculitis. This complication deserves consideration in patients with especially severe abdominal pain. Testing for lipase and ultrasonic imaging by an examiner aware of the diagnostic suspicion represents, in our opinion, the most reliable diagnostic tools currently available for Henoch-Schönlein patients with suspected pancreato-biliary involvement. Finally, the present analysis demonstrates that viral liver disease and, to a lesser extent, hepatitis vaccinations may be rare triggers of Henoch-Schönlein purpura.

REFERENCES

1. Reid-Adam J. Henoch-Schönlein purpura. *Pediatr Rev* 2014;35:447–9.
2. Moher D, Liberati A, Tetzlaff J, et al. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *Ann Intern Med* 2009;151:264–9.
3. Brogan P, Eleftheriou D, Dillon M. Small vessel vasculitis. *Pediatr Nephrol* 2010;25:1025–35.
4. Carroll JK, Herrick B, Gipson T, et al. Acute pancreatitis: diagnosis, prognosis, and treatment. *Am Fam Physician* 2007;75:1513–20.
5. Barie PS, Eachempati SR. Acute acalculous cholecystitis. *Gastroenterol Clin North Am* 2010;39:343–57.
6. Bucher B, Fiore E, Bernasconi M, et al. Schönlein-Henoch Syndrom des Kindesalters—Gewöhnliche und ungewöhnliche Beschwerden, Komplikationen, Finkelstein-Seidlmayer Variante, und Behandlung. *Ther Umsch* 2008;65:269–77.
7. Garner JA. Acute pancreatitis as a complication of anaphylactoid (Henoch-Schönlein) purpura. *Arch Dis Child* 1977;52:971–2.
8. Puppala AR, Cheng JC, Steinheber FU. Pancreatitis—a rare complication of Schönlein-Henoch purpura. *Am J Gastroenterol* 1978;69:101–4.
9. Branski D, Gross V, Gross-Kieselstein E, et al. Pancreatitis as a complication of Henoch-Schönlein purpura. *J Pediatr Gastroenterol Nutr* 1982;1:275–6.
10. Mateo Alvarez S, Morales Cerdán JM, Bello Nicolau I, et al. Pancreatitis aguda. Una excepcional manifestación inicial de la púrpura de Schönlein-Henoch. *Rev Clin Esp* 1987;180:88–9.
11. Van Bever HP, Van Doorn JW, Demey HE. Adult respiratory distress syndrome associated with *Mycoplasma pneumoniae* infection. *Eur J Pediatr* 1992;151:227–8.
12. El Alami S, Azar C, Bheane M, et al. Acute pancreatitis: a rare initial clinical manifestation of Henoch-Schönlein disease. *Acta Gastroenterol Belg* 1994;57:264–5.
13. Diaz CF. Schönlein-Henoch purpura and pancreatitis. *Dig Dis Sci* 1995;40:750–1.
14. Cheung KM, Mok F, Lam P, et al. Pancreatitis associated with Henoch-Schönlein purpura. *J Paediatr Child Health* 2001;37:311–3.
15. Chen SY, Kong MS. Gastrointestinal manifestations and complications of Henoch-Schönlein purpura. *Chang Gung Med J* 2004;27:175–81.
16. Katopodis K, Katsanos KH, Tsianos EV, et al. Hyperamylasemia as manifestation of gastrointestinal involvement in adult type Henoch-Schönlein purpura. *Ann Gastroenterol* 2004;17:303–5.
17. Sato S, Irisawa A, Shio K, et al. Case of an elderly man with associated Henoch-Schönlein purpura during treatment of acute pancreatitis. *Fukushima J Med Sci* 2006;52:135–42.
18. Soyer T, Egritas O, Atmaca E, et al. Acute pancreatitis: a rare presenting feature of Henoch-Schönlein purpura. *J Paediatr Child Health* 2008;44:152–3.
19. Zaidi M, Singh N, Kamran M, et al. Acute onset of hematuria and proteinuria associated with multiorgan involvement of the heart, liver, pancreas, kidneys, and skin in a patient with Henoch-Schönlein purpura. *Kidney Int* 2008;73:503–8.
20. Cakir M, Dilber B, Kalyoncu M, et al. Acute pancreatitis as an atypical presentation of Henoch-Schönlein purpura and cystic fibrosis. *Turk J Med Sci* 2009;39:809–13.
21. Dinler G, Bek K, Açikgöz Y, et al. Acute pancreatitis as a presenting feature of Henoch-Schönlein purpura. *Turk J Pediatr* 2010;52:191–3.
22. Snoussi M, Frigui M, Lehiani D, et al. Pancréatite aiguë révélatrice d'un purpura rhumatoïde. *Rev Med Interne* 2010;31:S154.

23. Frigui M, Lehiani D, Koubaa M, et al. Acute pancreatitis as initial manifestation of adult Henoch-Schönlein purpura: report of a case and review of literature. *Eur J Gastroenterol Hepatol* 2011;23:189–92.
24. Nie Y, Li W, Tong Z, et al. Acute pancreatitis associated with Henoch-Schönlein purpura. *Pancreas* 2011;40:315–6.
25. So MW, Kim YJ, Park HS, et al. Acute pancreatitis as a complication of adult Henoch-Schönlein purpura. *J Rheum Dis* 2012;19:43–6.
26. Katz AJ, Gang DL. Case records of the Massachusetts General Hospital. Weekly clinicopathological exercises. Case 14-1980: a five-year-old girl with a skin rash and abdominal pain. *N Engl J Med* 1980;302:853–8.
27. Kumon Y, Hisatake K, Chikamori M, et al. A case of vasculitic cholecystitis associated with Schönlein-Henoch purpura in an adult. *Gastroenterol Jpn* 1988;23:68–72.
28. McCrindle BW, Wood RA, Nussbaum AR. Henoch-Schönlein syndrome. Unusual manifestations with hydrops of the gallbladder. *Clin Pediatr (Phila)* 1988;27:254–6.
29. Pery M, Alon U, Lachter JH, et al. The value of ultrasound in Schönlein-Henoch purpura. *Eur J Pediatr* 1990;150:92–4.
30. de la Peña A, Yuste JR, Beloqui O, et al. Colecistitis alitiásica en el síndrome de Schönlein-Henoch. A propósito de un caso. *Rev Med Univ Navarra* 1995;39:126–9.
31. Viola S, Meyer M, Fabre M, et al. Ischemic necrosis of bile ducts complicating Schönlein-Henoch purpura. *Gastroenterology* 1999;117:211–4.
32. Hoffmann JC, Cremer P, Preiss JC, et al. Gallbladder involvement of Henoch-Schönlein purpura mimicking acute acalculous cholecystitis. *Digestion* 2004;70:45–8.
33. Ryan P. Buttock spots. *Clin Pediatr (Phila)* 2006;45:867–9.
34. Hashimoto A, Matsushita R, Iizuka N, et al. Henoch-Schönlein purpura complicated by perforation of the gallbladder. *Rheumatol Int* 2009;29:441–3.
35. Özkaya AK, Güler E, Çetinkaya A, et al. Henoch-Schönlein purpura complicated by acalculous cholecystitis and intussusception, and following recurrent appendicitis. *Paediatr Int Child Health* 2015 Apr 3. [Epub ahead of print]
36. Bosio M, Ravelli A, Ruperto N, et al. Síndrome di Schönlein-Henoch con severo interessamento multisistemico. *Minerva Pediatr* 1993;45:197–201.
37. Amemoto K, Nagita A, Aoki S, et al. Ultrasonographic gallbladder wall thickening in children with Henoch-Schönlein purpura. *J Pediatr Gastroenterol Nutr* 1994;19:126–8.
38. Lévy-Weil FE, Sigal M, Renard P, et al. Pancréatite aiguë au cours d'un purpura rhumatoïde. À propos de deux cas. *Rev Med Interne* 1997;18:54–8.
39. Maggiore G, Martini A, Grifeo S, et al. Hepatitis B virus infection and Schönlein-Henoch purpura. *Am J Dis Child* 1984;138:681–2.
40. Stemerowicz R, Möller B, Lobeck H, et al. Schönlein-Henoch Purpura bei HBsAg-positiver chronischer Hepatitis. *Immun Infekt* 1988;16:12–5.
41. Frankum B, Katelaris CH. Hepatitis C infection and Henoch-Schönlein purpura. *Aust N Z J Med* 1995;25:176.
42. Ogawa M, Makino Y, Ueda S, et al. Rapidly progressive glomerulonephritis in association with Henoch-Schönlein purpura in a patient with advanced liver cirrhosis. *Nephron* 1995;71:365–6.
43. Braun S, Ramaker J, Dippel E, et al. Purpura Schönlein-Henoch assoziiert mit Hepatitis-B-Infektion bei einem HIV-Patienten. *Dtsch Med Wochenschr* 2001;126:103–7.
44. Madison DL, Allen E, Deodhar A, et al. Henoch-Schönlein purpura: a possible complication of hepatitis C related liver cirrhosis. *Ann Rheum Dis* 2002;61:281–2.
45. Islek I, Kalayci AG, Gok F, et al. Henoch-Schönlein purpura associated with hepatitis A infection. *Pediatr Int* 2003;45:114–6.
46. Chemli J, Zouari N, Belkadi A, et al. Hépatite A et purpura rhumatoïde: une association rare. *Arch Pediatr* 2004;11:1202–4.
47. Ergin S, Sanli Erdoğan B, Turgut H, et al. Relapsing Henoch-Schönlein purpura in an adult patient associated with hepatitis B virus infection. *J Dermatol* 2005;32:839–42.
48. Altinkaynak S, Ertekin V, Selimoglu MA. Association of Henoch-Schönlein purpura and hepatitis A. *J Emerg Med* 2006;30:219–20.
49. Shin JI, Lee JS. Hepatitis B virus infection and Henoch-Schönlein purpura. *J Dermatol* 2007;34:156.
50. Mohan N, Karkra S. Henoch-Schönlein purpura as an extra hepatic manifestation of hepatitis A. *Indian Pediatr* 2010;47:448.
51. Thapa R, Biswas B, Mallick D. Henoch-Schönlein purpura triggered by acute hepatitis E virus infection. *J Emerg Med* 2010;39:218–9.
52. Sasan MS, Doghaee MA. Association of Henoch-Schönlein purpura with hepatitis A. *Iran J Pediatr* 2012;22:571–2.
53. Bani-Sadr F, Gueit I, Humbert G. Vasculitis related to hepatitis A vaccination. *Clin Infect Dis* 1996;22:596.
54. Masse I, Descoffres MC. Vasculite d'hypersensibilité après vaccination contre l'hépatite B. *Presse Med* 1998;27:965–6.
55. Chave T, Neal C, Camp R. Henoch-Schönlein purpura following hepatitis B vaccination. *J Dermatol Treat* 2003;14:179–81.
56. Jariwala S, Vernon N, Shliozberg J. Henoch-Schönlein purpura after hepatitis A vaccination. *Ann Allergy Asthma Immunol* 2011;107:180–1.
57. Chao HC, Kong MS, Lin SJ. Hepatobiliary involvement of Henoch-Schönlein purpura in children. *Acta Paediatr Taiwan* 2000;41:63–8.
58. Guillevin L. Infections in vasculitis. *Best Pract Res Clin Rheumatol* 2013;27:19–31.