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CLINICAL REPORT

Henoch-Schönlein Purpura Associated With Solid-organ Malignancies: Three Case Reports and a Literature Review

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Adult Henoch-Schönlein purpura (HSP) is rarely associated with solid-organ malignancies. We describe here three adult patients with HSP diagnosed within 3 months of the diagnosis of associated solid-organ malignancies, including pulmonary, prostate, and renal carcinomas. Two patients had complete remission with a combination of immunosuppressive therapies and treatment of the associated malignancy. The third patient had partial remission with immunosuppressive therapies, but never received treatment for the associated malignancy and did not achieve complete remission before his death 10 months after diagnosis of HSP. These cases suggest that HSP associated with solid-organ malignancies may be resistant to immunosuppressive therapies without treatment of the associated malignancy. Therefore, evaluation for solid-organ malignancies should be considered in adult patients without an identifiable cause of HSP, especially if the disease is not self-limited or does not respond appropriately to treatment. ***Key words: coagulation; cutaneous disease; immunopathology; immunofluorescence; microvascular occlusion syndrome; purpura; vasculitis.***

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We recently described 17 cases of cutaneous small vessel vasculitis (CSVV) associated with solid-organ malignancies (1). In that study, CSVV was equivalent to leukocytoclastic vasculitis or hypersensitivity vasculitis (1), per the inclusion criteria of Fiorentino (2). Similar to CSVV, Henoch-Schönlein purpura (HSP) has also previously been associated with malignancy. However, in contrast to CSVV, HSP has been reported to occur more commonly with solid-organ malignancies than with hematological malignancies (3, 4). In particular, pulmonary, prostatic, and renal carcinomas have been most commonly associated with the development of HSP (4). In total, 47 cases of HSP related to solid-organ malignancy have been reported previously (Table I and Table SI; available from: http://www.medicaljournals.se/acta/content/?doi=10.2340/00015555-1288). We report here 3 additional cases of HSP associated with solid-organ malignancies seen at Mayo Clinic over the previous 13 years.

Table I. *Summary of characteristics of 47 previously reported patients with Henoch-Schönlein purpura associated with solid-organ malignancy*[a]

Characteristic	Value
Age, years, mean	62[b]
Men, *n* (%)	33 (70)
Type of solid-organ malignancy (*n* = 53)[c], *n* (%)	
Lung	14 (26)
Prostate	6 (11)
Kidney	5 (9)
Gastric	4 (8)
Breast	3 (6)
Thyroid	3 (6)
Carcinoid	2 (4)
Maxillary	2 (4)
Cervical	2 (4)
Colon	2 (4)
Epiglottic, Hypopharyngeal	2 (4)
Esophageal	1 (2)
Anal, Rectal	2 (4)
Ovarian, Endometrial	2 (4)
Hepatocellular, Cholangiocarcinoma	2 (4)
Schwannoma	1 (2)
Onset of cutaneous vasculitis in relation to malignancy (*n* = 53)[c], *n* (%)	
Before	14 (26)
Synchronous[d]	19 (36)
After	20 (38)
Response of cutaneous vasculitis to treatment, *n* (%)	
Remission with immunosuppressive agents	3 (6)
Remission with treatment of cancer	9 (19)
Remission with combined treatment	4 (9)
No remission	0
Unknown	31 (66)
Outcome, *n* (%)	
Alive	8 (17)
Deceased	14 (30)
Unknown	25 (53)

[a]Three cases described by Fain et al. (25) were not included because individual patient information was not available.
[b]Two cases had no record of patient age or sex.
[c]Six patients (cases 14, 18, 27, 33, 34, and 47 in Table SI) had 2 separate malignancies associated with HSP.
[d]Occurred within 1 month of each other.

CASE REPORTS

This study was approved by the Mayo Clinic Institutional Review Board.

Case 1. A 47-year-old man presented with palpable purpura of one week's duration involving his lower extremities (Fig. 1A,

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Fig. 1. A. Palpable purpura and several larger, necrotic, purpuric plaques on the bilateral lower extremities. B. Clustered, occasionally coalescent, necrotic plaques with surrounding violaceous erythema along the anterior tibia.

Table II). Biopsies of representative lesions demonstrated leukocytoclastic vasculitis. Direct immunofluorescence performed on samples of lesional skin revealed deposition of IgA within superficial dermal blood vessels. Urinalysis revealed microhematuria. A diagnosis of probable HSP was made, and treatment was begun with dapsone 25 mg orally twice daily, as well as topical fluocinonide 0.05% cream and tacrolimus 0.1% ointment.

Throughout the next several weeks, the lesions spread to involve his trunk and bilateral upper extremities. An increasing number of large, crusted, necrotic lesions developed on the distal lower extremities (Fig. 1B). Concomitantly, arthritic pain developed in his knees, ankles, and hands, along with abdominal pain, nausea, and diarrhea. In addition, he experienced irritation and a sensation of fullness in his oropharynx. Otolaryngological evaluation determined that he had uvular swelling that was probably associated with HSP. Treatment with prednisone 60 mg orally daily was initiated; he had significant improvement in his rash, abdominal pain, arthritis, and uvular swelling within 2 days of starting oral prednisone. The skin lesions and symptoms continued to improve throughout a 4-week prednisone taper.

The patient underwent computed tomography of the abdomen to evaluate his abdominal pain and diarrhea. A 2-cm, solid, enhancing renal mass was identified. Further evaluation revealed clear cell renal cell carcinoma (stage I, T1aNXM0). He underwent partial nephrectomy approximately 5 weeks after the initial diagnosis of HSP. Immunofluorescent histological analysis of the glomeruli showed mesangial staining for IgA and C3, consistent with IgA nephropathy.

After the partial nephrectomy, the patient's skin lesions and systemic symptoms continued to improve. No new lesions developed. The oral dapsone was tapered 2 months later, and the HSP has shown no signs of recurrence after 28 months of follow-up.

Case 2. A 71-year-old woman was evaluated for palpable purpura on her bilateral lower extremities (Table II). Three months earlier, she had received a diagnosis of squamous cell carcinoma of the lung (stage IIIb, T4N2M0). Treatment of the carcinoma had included bronchoscopy with laser ablation of the tumor and subsequent radiotherapy and chemotherapy with etoposide and cisplatin for nodal metastases. Pertinent positive laboratory values during evaluation of the purpura included an erythrocyte sedimentation rate of 56 mm/h (normal $\leq$ 22 mm/h) and hematuria and proteinuria on urinalysis. Skin biopsy of a lower extremity lesion indicated leukocytoclastic vasculitis,

Table II. *Characteristics of patients with Henoch-Schönlein purpura (HSP) associated with solid-organ malignancy*

Case	Age, years/sex	Presenting features of HSP	Onset of vasculitis in relation to malignancy	Type of solid-organ malignancy	Abnormal laboratory values[a]	Treatment of vasculitis	Treatment of solid-organ malignancy	Response to treatment	Outcome, follow-up[b]	Comment
1[c]	47/M	Purpura, arthritis, abdominal pain, nausea, diarrhea	Synchronous[d]	Renal carcinoma	Mildly increased RF level (negative CCP antibodies), microhematuria	Prednisone, dapsone, fluocinolone 0.05% cream, tacrolimus 0.1% ointment	Surgery	Remission[e]	Alive, 28 months	Uvular swelling believed to be secondary to HSP per otolaryngologist evaluation
2	71/F	Purpura, arthritis	3 months after	Lung carcinoma	Increased ESR, microhematuria, proteinuria	Prednisone	Surgery, chemotherapy, irradiation	Remission[e]	Deceased, 14 months	None
3	68/M	Purpura, bilateral pulmonary infiltrates, arthritis	Synchronous[d]	Prostate carcinoma	Increased ESR and CRP levels, decreased C3 level, microhematuria	Prednisone, HCQ, triamcinolone 0.1% cream	None	Partial remission[f]	Deceased, 10 months	Bilateral pulmonary infiltrates believed to be secondary to HSP per pulmonologist evaluation

[a]Skin biopsy findings showed leukocytoclastic vasculitis in all cases.
[b]From diagnosis of HSP.
[c]Briefly reported in Podjasek et al. (1).
[d]Occurred within 1 month of each other.
[e]After combination of cancer treatment and immunosuppressive therapy.
[f]After treatment of vasculitis with glucocorticoids or other immunosuppressive agent.
CCP: cyclic citrullinated peptide; CRP: C-reactive protein; DIF: direct immunofluorescence; ESR: erythrocyte sedimentation rate; HCQ: hydroxychloroquine; RF: rheumatoid factor.

and direct immunofluorescence demonstrated IgA and C3 deposition within papillary dermal blood vessels. Moreover, on renal biopsy to evaluate the hematuria and proteinuria, immunofluorescent histological analysis showed segmental and diffuse granular mesangial staining for IgA and C3, consistent with IgA nephropathy.

On the basis of the clinical presentation and biopsy results, HSP was diagnosed. Radiotherapy and chemotherapy for the lung carcinoma were continued. A 3-week course of oral prednisone 60 mg daily led to resolution of the cutaneous lesions. Per recommendations from her nephrologist, she continued prednisone therapy for a total of 6 months for the IgA nephropathy.

Unfortunately, the carcinoma recurred 14 months after the initial diagnosis of carcinoma, although the HSP did not recur at this time. The patient died of metastatic disease 3 months later.

Case 3. A 68-year-old man was evaluated for palpable purpura involving his bilateral lower extremities and buttocks (Table II). He had received a diagnosis of prostatic adenocarcinoma (stage II, T2aNXM0) one week earlier. Pertinent positive laboratory values included an erythrocyte sedimentation rate of 140 mm/h (normal ≤22 mm/h), C-reactive protein value of 19 mg/dl (≤0.8 mg/dl), C3 value of 65 mg/dl (75–175 mg/dl), and microhematuria. Chest radiography showed new, patchy, bilateral, nodular pulmonary infiltrates not seen on a chest radiograph taken one week earlier. These findings were confirmed with computed tomography of the chest. Histopathologic analysis of a representative lesion on the lower extremity showed leukocytoclastic vasculitis; direct immunofluorescence showed deposition of IgA and C3 in papillary dermal blood vessels.

On the basis of the patient's clinical presentation and biopsy results, a diagnosis of HSP was made. Pulmonary consultation was obtained to fully evaluate the changes detected on imaging studies. Transbronchial biopsy of a nodular infiltrate in the right upper lobe revealed benign, reactive changes. No evidence of neoplasia, infection, or hemorrhage was detected via biopsy or bronchoalveolar lavage. The changes were believed to be most likely secondary to pulmonary involvement of the HSP.

Treatment with prednisone 40 mg orally daily was initiated. Both the cutaneous disease and pulmonary infiltrates improved substantially within 2 weeks of initiating prednisone. Attempts to taper prednisone below 30 mg orally daily resulted in flaring of his cutaneous disease. Therefore, hydroxychloroquine 200 mg orally twice daily was initiated as a steroid-sparing agent after 6 months of treatment with prednisone. After the addition of hydroxychloroquine, attempts to taper the prednisone dosage to less than 10 mg orally daily resulted in flaring.

Radiation therapy for his prostate adenocarcinoma was deferred because of concern for the increased likelihood of adverse events associated with radiotherapy while the patient was taking prednisone. Unfortunately, the patient died of unknown causes 10 months after the diagnosis of HSP.

DISCUSSION

IgA vasculitis is diagnosed when patients with cutaneous palpable purpura have either prominent vascular deposition of IgA or vascular deposition of IgA that is dominant with respect to IgG and IgM on direct immunofluorescence testing (27). The classification of IgA vasculitis includes both HSP- and non-HSP-related IgA vasculitis, depending on the presence or absence of characteristic extracutaneous involvement of the joints, gastrointestinal tract, and kidneys. Per the American College of Rheumatology criteria (28), a diagnosis of HSP is 87.1% sensitive and 87.7% specific when at least 2 of the following features are present: palpable purpura, bowel angina, age 20 years or younger at onset, and histological changes of leukocytoclastic vasculitis. HSP typically presents with IgA deposition involving the skin, kidney, and intestines, and vascular IgA deposition is one of the Chapel Hill criteria for diagnosis of HSP (29). Nonetheless, IgA deposition can also be detected in the skin of patients with numerous other conditions, including IgA nephropathy (30), alcoholism (31), and dermatitis herpetiformis (32). Thus, although the finding of IgA deposits is non-specific, combination of this finding with clinical data improves the diagnostic accuracy for HSP (33).

Most commonly, cases of HSP are associated with infections of mucosal sites, including the gastrointestinal, respiratory, and genitourinary mucosae. Other previously reported causes of IgA vasculitis (both HSP- and non-HSP-related) include IgA paraproteinemia (34), multiple myeloma (35), inflammatory bowel disease (36), ankylosing spondylitis (36), systemic lupus erythematosus (35), Sjögren syndrome (37), rheumatoid arthritis (38), cryoglobulinemia (27), and adverse drug reactions to numerous medications, including calcium-channel blockers (39) and carbamazepine (40).

Furthermore, numerous cases of HSP have been associated with solid-organ malignancies. One previous group suggested that, in adult patients with HSP, the most commonly associated solid-organ malignancies involve the lung and the prostate (4). Another group examined the cause of death of 250 adults with HSP and found that malignancies, most commonly involving the lung, upper respiratory tract, and digestive tract, accounted for 17 of 64 deaths (27%) (41). Finally, a more recent study described 24 cases of malignancy-associated HSP in adults, associated with solid-organ malignancies in 18 cases, cutaneous malignancies in 3 cases, and lymphoproliferative disorders in 3 cases (16). In contrast, only 2 cases of childhood HSP have been associated with solid-organ malignancies, both of which were Wilms tumors (42).

All 3 of our patients had palpable purpura of the lower extremities and granulocytes in blood vessel walls on biopsy, thereby meeting the American College of Rheumatology criteria for the diagnosis of HSP (28). Additionally, each patient had hematuria, arthritis, and vascular deposition of IgA on direct immunofluorescence testing of tissue from representative skin lesions, which provided further support for the diagnosis of HSP (per the Chapel Hill Consensus Conference criteria) (29). Cases 1 and 3 had extracutaneous involvement of the uvula and lung, respectively, both of which are only rarely involved by HSP.

The cutaneous vasculitis was believed to be possibly secondary to the solid-organ malignancy when no known triggering factor (e.g., infection, medication, chemotherapeutic agent, allergic reaction, cryoglobuli-

nemia, or autoimmune connective tissue disease) could be identified as a more likely cause. Although it is difficult to state with certainty, the HSP in patient 2 was not thought to be related to her chemotherapy because her HSP resolved completely with a brief course of oral glucocorticoids and she continued receiving the chemotherapeutic agents without any recurrence of HSP.

Each patient had partial remission of HSP with immunosuppressive therapies, including prednisone, dapsone, hydroxychloroquine, topical glucocorticoids, and topical calcineurin inhibitors. Two patients (cases 1 and 2) also received treatment of their associated malignancy and achieved complete remission of HSP, suggesting that treatment of the associated malignancy may have been necessary to achieve complete remission of HSP. Patient 3 did not receive treatment for his associated prostate adenocarcinoma and never had complete remission of HSP before his death from an unknown cause 10 months after the diagnosis of HSP. In contrast, 9 of 17 patients (53%) with CSVV associated with solid-organ malignancies in our recent study had complete remission of the vasculitis with immunosuppressive therapies alone (1). Although our sample size of three cases is small, our data suggest that HSP associated with solid-organ malignancy may be more resistant to immunosuppressive therapies than malignancy-associated CSVV, and that treatment of the associated malignancy may be required more frequently to achieve complete remission of HSP. Future studies may help elucidate this potential difference in the response to treatment between malignancy-associated HSP and malignancy-associated CSVV.

The precise relationship between HSP and solid-organ malignancies is still not entirely clear. It is possible that abnormal immune complex deposition, perhaps secondary to: (*i*) aberrant production of antibodies and tumor antigens with resultant immune complex deposition within vessel walls, (*ii*) resemblance of tumor antigens to endothelial cell antigens, or (*iii*) decreased clearance of immune complexes, has an important role (43–45). Conversely, treatment of the solid-organ malignancy may induce HSP. Cases of HSP associated with numerous chemotherapeutic agents, including erlotinib (46), cytarabine (47), and anastrozole (48), have been described previously. Finally, subclinical infections involving undetectable bacterial or viral antigens might be causative (49).

Although the exact nature of the relationship between HSP and solid-organ malignancies remains ill defined, we provide further evidence of this important association, especially with malignancies involving the lung, prostate, and kidney. We also suggest that HSP associated with solid-organ malignancies may be resistant to immunosuppressive therapies without treatment of the associated malignancy. Finally, we recommend that evaluation for solid-organ malignancies be considered in adult patients without an identifiable cause for the development of HSP, especially if the disease is not self-limited or does not respond appropriately to treatment.

The authors declare no conflicts of interest.

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