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SKIN MANIFESTATIONS A CLUE FOR OCCULT GASTROINTESTINAL DISEASE

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ABSTRACT The gastrointestinal GI and dermal systems are closely related in origin. Skin symptoms are often found as part of many GI illnesses. This overview includes disorders involving the skin as well as the intestines. GI malignancies, hereditary nonpolyposis colorectal cancers "CRCs" as well as inflammatory bowel disease "IBD" is examined with a genetic emphasis, diagnostic and histological findings.

Skin problems typically linked to GI include:

- Pyoderma gangrenosum: intestinal illness inflammatory.
- Hepatitis C infection of lichen planus and porphyria dermal tarda.
- Diffuse diabetes-hyperpigmentation (bronze diabetes) in persons with diabetes:
- Erythema nodosum: intestinal illness inflammatory, sarcoidosis, and other infections.
- Eruptive xanthomas: high serum xanthomas.

KEY WORDS : Systemic Sclerosis (SSc), Diffuse Systemic Sclerosis (dcSSc) GIT (Gastrointestinal Tract), Limited Systemic Sclerosis (lcSSc)

Introduction

Due to the embryonic origins of the gastrointestinal system and the skin, dermatological signs and symptoms may precede or follow gastrointestinal disease. Dermatological symptoms are common in gastrointestinal diseases. Clinicians can detect occult disease in the GI tract if they have a thorough understanding of the cutaneous gastrointestinal connection. The goal of this review was to investigate this link by describing illnesses that affect both the GI system and the skin. [1]

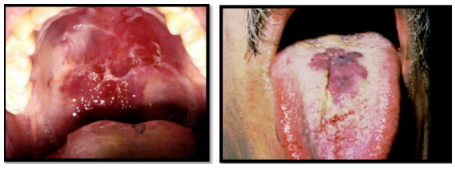
A variety of cutaneous conditions have been linked to underlying gastrointestinal cancers [8]. Cancer cells can penetrate the skin, indicating metastatic dissemination from a GI malignancy like Sister Mary Joseph's nodule. However, some skin lesions are linked to the existence of cancer beneath the surface, but they lack malignant cells and are referred to as paraneoplastic dermatological disorders. The link between cutaneous paraneoplastic diseases and gastrointestinal malignancy morphologic characteristics.

1. Cutaneous Manifestation of Oral and Pharyngeal Diseases :

A. Kaposi sarcoma

Kaposi sarcoma (KS) is defined as a vascular neoplasm of endothelial and lymphatic cells affecting several visceral organs, as well as the skin. The disease has four forms: (1) classical epidemics; (2) epidemics, (3) endemics; and (4). [7][9][10]. The skin lesions of KS – mainly classical and epidemic – begin as red or pink macules which become more and more popular as they grow. Classic KS lesions are more common on the face and trunk of the lower limbs and KS lesions are common. [11] The GI tract can occur on every level, although the small intestine, stomach, and esophagus are the most frequently affected areas (in order). Oral lesions are most likely to affect the hard palate, gingiva, and tongue in order of frequency (see figure below).

Fig-1: Oral Kaposi sarcoma in a patient with AIDS. Note the characteristic purple hemorrhagic papules coalescing into an irregular plaque"



2. Cutaneous manifestation of Esophageal diseases:

A. "Plummer-Vinson syndrome Patterson-Brown-Kelly syndrome"

Signs of Plummer-Vinson syndrome include mucocutaneous signs of koilonychia and iron deficiency anemia and clinical complaint of dysphagia, as well as early teeth, cheilosis, tongue atrophy, and Angular Stomatitis. [12],[13] Although this disease has become less frequent due to earlier iron deficiency diagnoses and the ready availability of iron supplementation.

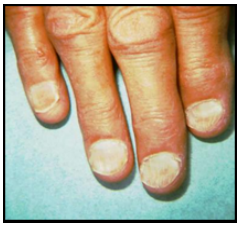


Fig-2:Koilonychia. Note the double concavity (longitudinal and transverse) of the nails.

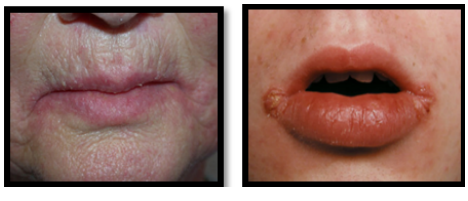


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Fig-3: Angular cheilitis in females a- due to Plummer-Vinson syndrome B- due to chrons disease

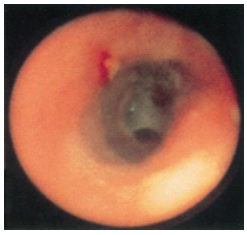


Fig-4: Postcricoid web in a patient with Plummer-Vinson syndrome. Note the 2 small openings within the web at the 2- and 6-o'clock positions, representing a significantly compromised proximal esophageal lumen."

B. Epidermolysis bullosa"

Rare, heritage-based disorders that produce fragile skin are known as epidermolysis bullosa (EB). [14] In these patients, blisters tend to form at locations of minor damage, where the symptoms start at birth or early childhood. The condition, according to the skin layer, is clinically categorized into three principal kinds where blister forms 1) (low lamina densa), 2) (Lucida lamina laminas) and 3) (simplex laminate dystrophic; (intraepidermal). Mutations in the dermo-epidermal junction and basement membrane genes have been involved in various kinds of EB. (Lower picture). [14]

Constipation and gastroesophageal reflux are the primary GI symptoms in EB simplex (GOR). Patients in junctional EB may complain of a lack of thriving enteropathy and protein loss (PLE). Most patients with recessive dystrophic EB have had gastroesophageal reflux and dysphagia, and they generally describe severe oesophageal severity.

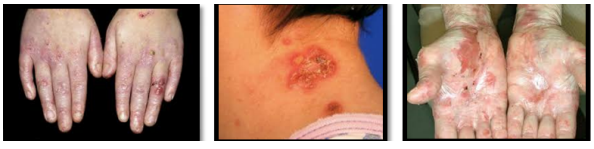


Fig- 5: Three cases of Epidermolysis bullosa"

A. Systemic sclerosis (scleroderma)

Systemic sclerosis (SSc) depicts multisystem disease causing the skin, blood vessels, lungs, heart, kidneys, and gastrointestinal tract to transform fibrotically. [15],[16] SSc is split into five different forms: diffuse systemic sclerosis, (dcSSc), limited systemic sclerosis, transitory scleroderma, and malignant scleroderma. DcSSc and lcSSc are the main forms. In lcSSc, the tightness of the skin is limited to the fingers, hand, and forearm, which lie distal from the elbows, and with the skin of the feet or without a tightening of the legs, which were dubbed CREST (calcinosis, rainforest phenomena, esophagus dysmotility, sclerodactyly, and telangiectasia). DocSSc. The internal involvement in both dcSSc and lcSSc is related, although patients with dcSSc are more likely to experience substantial clinical impairment (see the image below).

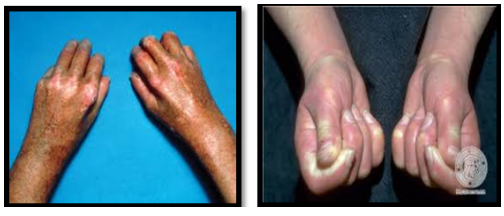


Fig – 6: Skin changes of scleroderma usually begin with an early phase of skin edema, which" "presents as swollen fingers and hands, also known as the"'"puffy hand sign".

The GI tract in SSc is the internal organ most frequently involved.[17] Oesophagus, mid duodenum, jejunum, and large intestine are the most usually affected sites. Esophageal symptoms include early fulfillment, reflux esophagitis, epigastric and discomfort dysphagia, which are regarded as the most prevalent symptoms.

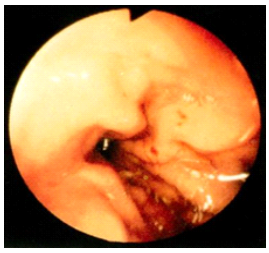


Fig – 7: Telangiectases in the gastric mucosa of a patient with Osler-Weber-Rendu syndrome. The lesions can be seen most prominently at the 2 o'clock position proximally and the 3 o'clock position distally."

Colonic telangiectasia is prevalent and can lead to open bleeding that can lead to anemia. In fecal incontinence, neuropathy plays a role. In scleroderma, the esophagus followed by the small intestines is the most prevalent part of the GI tract during SSc. [18] SSc liver and bile involvement is comparatively rare, although in SSc patients it is more prevalent for primary bile cirrhosis (PBC). [18]

B. "Pemphigus Vulgaris"

Pemphigus Vulgaris (PV) has a primary effect on the skin and the mucosa, an autoimmune blistering illness. Mucosal regions, such as oral mucosa, anus, cervix, and conjunctiva are typically affected. In particular, the GI tract, which includes GI hemorrhage, is associated with gastrointestinal impact[19] (see the image below). [20]

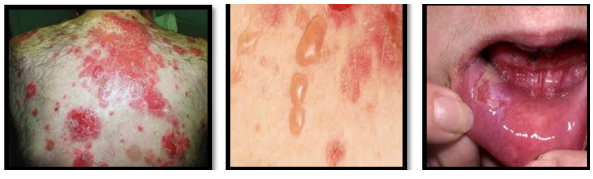


Fig – 8: Pemphigus Vulgaris in skin and oral mucosa

C. "Acrokeratosis neoplastic (Bazex syndrome)"

A psoriasis-like skin eruption mainly appears near the ends of the distal cord, but can also include the nose and the helixes of the ears. (Lower picture).

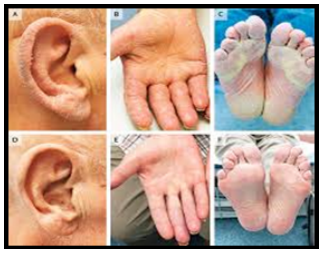


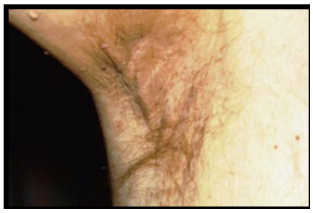
Fig- 9: (a,b) Skin findings on presentation: Hyperkeratosis and desquamation in a symmetrical acral distribution. (c,d) Improvement of skin lesions after one cycle of chemotherapy"

3. "Cutaneous manifestation of gastric diseases"

A. Acanthosis nigricans

Acanthosis nigricans is a very common discovery in the skin, most often in the neck, axillae, and groin, that are brown-to-black, smooth, and velvety plaques (see the image below). A rare but crucial finding accompanies the presentation of acanthosis palmaris or tripe Palms that emerge on the palmar surfaces of your hands as conspicuous skin marks together with a widespread velvet-paddled surface.

Fig- 10: Acanthosis nigricans (AN) in a patient with pancreatic cancer.



Tumor products can affect the skin by activating insulin-like growth factors or skin receptors. Alpha transforming tumor cell growth factors may play a role in malignant acanthosis in Nigricans utilizing the receptors of skin epidermal growth factor. [21] For acanthosis nigricans, in particular in patients without a clear predisposing factor, the possibility of intra-abdominal malignants needs to be addressed.[22]

Adenocarcinoma (85 percent), which included gastric carcinoma of 60 percent, comprises malignancies related to acanthosis nigricans. Other sites of cancer mentioned are the lung, bladder, endometrial, bile duct, kidneys, thyroid, breast, liver.

B. Sign of Leser-Trélat

The indication of Leser-Trélat is marked by numerous Seborry keratosis (rough, well-marked, verrucous, 'stuck-on' plaques), which expand fast in size and number (see the picture below). [23]

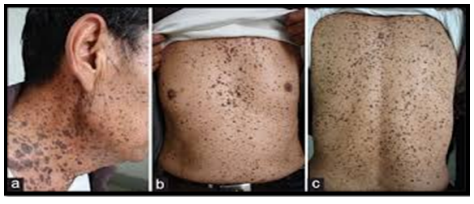


Fig- 11: The sign of Leser-Trélatin patient with hepatic carcinoma

FCF is a rare disease. FCF is a rare disease. The quick appearance of several verrucous papules and nodules that look quite much like viral warts [24] These lesions typically begin first on the back of the hands and wrists and later extend to other body regions. PFC is most usually connected but may also be related to other malignancies with gastric adenocarcinoma. [25]

C. Florid cutaneous papillomatosis (Schwartz-Burgess syndrome)

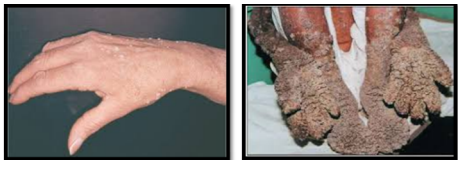


Fig- 12: Florid cutaneous papillomatosis

D. Sister Mary Joseph's nodule

Sister Mary Joseph was the first to observe the presence of a periumbilical nodule as a head nursery and expert operating aid to William Mayo, MD (see the image below). [26],[27] The cutaneous discovery is a firm nodule with a red or purple color that is metastasized by the initial tumor. The cutaneous finding Of 90% are adenocarcinoma, the most usually observed primary malignant adenocarcinoma being stomach or ovarian. The pancreas and bowel are other main locations mentioned.

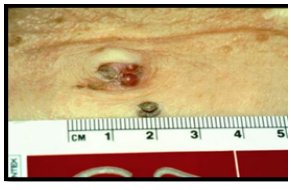


Fig- 13: Sister Mary Joseph nodule in a patient with gastric carcinoma. Note the shiny, reddish, telangiectatic group of papules in the umbilicus.

D. Osler-Weber-Rendu syndrome (hereditary hemorrhagic telangiectasia)

This autosomal dominant syndrome is characterized by vascular dilations of the epidermis, oral, nasal, and GI mucosa, with a rate of 1-case per 10.000 population (see the image below). [28],[29] Three of the four diagnosis criteria are used: epistaxis, telangiectasis, visceral lesions, and suitable family history. [30]

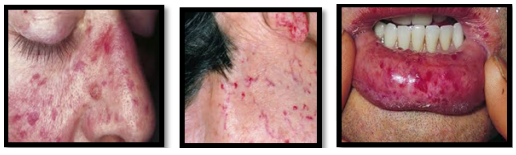


Fig- 14: Three cases of Osler-Weber-Rendu syndrome

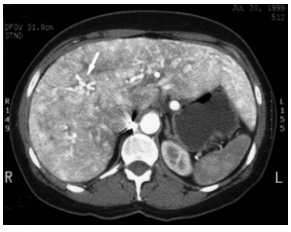


Fig- 15: Arteriovenous malformations as seen on CT scan in a patient with Osler-Weber-Rendu syndrome. The patches of hyperdensity within the liver are the result of previous embolization procedures.

The dermatological examination reveals several macular or popular, sharply demarcation-displayed telangiectasies, 1 to 3 mm in various places, including the face, lips, palate, tongue. [31] Vascular telangiectasia spontaneous bleeding within the GI tube. [32] The occurrence of arteriovenous malformations (AVMs) in the pulmonary, brain, and liver may be another GI symptom. Large AVMs that can develop within the liver lead to a significant creation of shunt.

E. E-Helicobacter pylori (H. pylori)

Bacteria discovered in the stomach Helicobacter pylori (H. pylori) are caused by the majority of peptic ulcers. [33]. [34] In a wide range of diseases not connected to the gastrointestinal tract, H. pylori were involved. The skin is a case in point and some experts have proposed that the treatment be associated with diseases [35],[36]:

- "Chronic spontaneous urticaria"
- "Rosacea – H. pylori can increase levels of nitrous oxide in the blood or tissue contributing to the flushing and erythema (redness) of rosacea".
- "Psoriasis – H. pylori may be one of the organisms capable of triggering the inflammatory response in psoriasis".
- "Sjögren syndrome – H. pylori may induce an autoimmune reaction to the skin and glands causing Sjögren syndrome".
- "Henoch-Schönlein purpura"
- "Alopecia areata"
- "Sweet disease"
- "Systemic sclerosis"
- "Atopic dermatitis"
- "Behçet disease"
- "Generalized pruritus (itch)"
- "Nodular prurigo"
- "Immune thrombocytopenic purpura"
- "Lichen planus"
- "Aphthous ulceration".

4. Cutaneous manifestations of the Liver and pancreatic diseases

A. Telangiectases
B. Spider telangiectasis
C. Palmar erythema
D. Terry's nail
E. Wilson disease: E. Wilson disease: a copper metabolism autosomal recessionary disorder. Wilson's signs include: Kayser-Fleisher iris ring, Shining and blue lunula (half-moons) on the nails Pigmentation
F. Hemochromatosis

The iron overload disorder causing excessive deposition in several organ systems is hemochromatosis. Hemochromatosis [37],[38] The inherited type of glycosuria (diabetes), bronze skin pigments, and cirrhosis was initially described in the late nineteenth century as the classic trio. Skin hyperpigmentation is mainly the dermatologic symptom of the disease. It has a characteristically gray metallic or bronze-brown color, but can be extended to areas of the scars or the front, neck, arm extensor surfs, and genitalia. This discoloration is mostly diffuse. About 20% of patients also show buccal mucosal or conjunctiva pigmentation. Skin shrinkage, ichthyosis, partial hair loss (most typically in a pubic region), and koilonychia are also characteristic symptoms in hemochromatosis. [39], Hepatomegaly, which is detected in over 950 percent of symptomatic patients, is the most prevalent GI discovery in hemochromatosis. Although functional liver deficiency was clinically found (see image below). Cirrhosis development. [39]

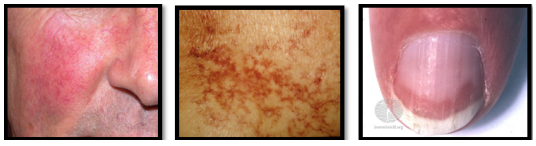


Fig-16: Facial redness, Telangiectasia and Terry nail as Cutaneous effects of liver disease.

G. Porphyria cutanea tarda

Fig-17: Porphyria cutanea tarda

The most prevalent porphyria occurring in grownups is porphyria cutanea tardia. The reduced effect of protein uroporphyrinogen decarboxylase (UROD), the fifth protein in the heme biosynthesis pathway, is the result of porphyria cutanea tarda. Skin photosensitivity with increased skin delicacy, face hypertrichosis, scarring of the skin, and other sun-exposure areas are the characteristic of cutaneous findings. Screening for hepatitis infection and hemochromatosis should be performed by all patients with porphyria cutanea tarda[40]. Hepatitis C predominance in PCT patients was measured by patients from various countries, and in those patients, the rates were as high as 85 percent. [41]Fig-17: Porphyria cutanea tarda as Cutaneous effects of liver disease

F. Pancreatic fat necrosis

Pancreatic fat bribery shows the association of pancreatic skin knobs. Dermatological injuries may seem easy or severe and start on the legs of the flesh and trunk. [42] The introduction can include lower limited pruritus with associated erythema splotchy. The move to a nodular component is linked to sensitivity. The buttons have been shown to suddenly deplete white-rich exudate, which recovers the injury with hyperpigmentation and the neighboring scar configuration.

The presence on the skin surface of such fluctuating damage was used to identify fundamental pancreatic diseases. [43],[44] The skin emission regularly leads to the comprehension of the need for restoration in the first place. In up to 65% of patients suffering from pancreatic cancer and up to 22% from patients receiving pancreas corruption, pancreas fat corruption occurs. More than half of patients with subcutaneous fat corruption linked with pancreatic cancer have the shape of acinar. [45]

G. Lichen planus

Lichen planus, known to be associated with several liver diseases, is an idiopathic provoking skin seizure and mucus layers. Injuries of the skin are lilac, polygonal, flaccid papules, usually pruritic. They can be located on the skin, even though several places are most usually affected, including the handles, lower legs, shins, lower back, and genitalia. In addition, the mucosal layers are affected as frequently as possible[46] and can be located as they were. The proximity of pterygia and longitudinal margins is also typical for nail dystrophy (see the pictures underneath). Köbner marvel, which happens in the locations of prior damage, shows both cutaneous and verbal injuries.

Lichens planus is not pathogenesis, however, a few inventors have theorized that both essential and chronic hepatitis B and C biliary cirrhosis are associated. [47],[48],[49],[50]

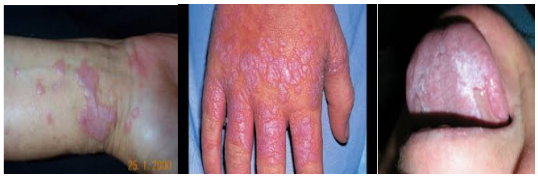


Fig-18: lichen planus as Cutaneous effects of liver disease"

H. Trousseau syndrome

Trousseau syndrome was used for the illustration of several types of infections: classic transitory thrombophlebitis to emboli of the blood vessel, verrucous endocarditis, and any kind of malignant coagulopathy. [51] Inflammatory, red, and painful linear lesions reflecting a vasculitis induced by the formation of a clot are found in migratory thrombophlebitis. The clots develop, dissolve, and then form elsewhere again, moving through the patient's trunk and limbs (see the image below). [51] This may occur in various adenocarcinomas but is strongly emphasized for pancreatic cancer. [52]

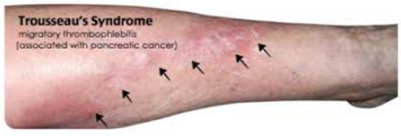


Fig-19: Trousseau syndrome

I. Necrolytic migratory erythema

Necrolytic migration erythema displays erythema patches that rank, disintegrate and hull, as ring-shaped patches. All phases of injury, from patches to bladder, to coverings, can be viewed synchronically when observing an understanding. Injuries may be precarious or agonizing and often spread across the peripheral site, lower-middle rim, flesh, crotch, and lower legs (see the picture below). [53] It is connected with glucagonomas, which secrete pancreatic alpha cells from glucagon. tumors. Necrolytic erythemas, in any case, can also be associated with hepatic infection and malabsorption in the intestines. [54]

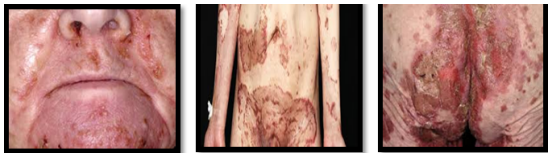


Fig-20: Necrolytic migratory erythema

A. Necrolytic acral erythema

As a psoriasis eruption with a well-circumvented pelvis to rhythm plate, with a thick adhesive scale, necrolytic acral-erythema (NAE) appears. [55] The lesions are related to burning or pruritus and are only contained in the acral. In the majority of instances, NAE is related to infection with hepatitis C,[56]though in people without hepatitis C, some cases have occurred. [57],[58] [58] (see the image below).

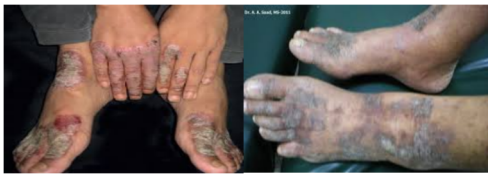


Fig-21: Necrolytic acral erythema

5. Cutaneous manifestation of Small Intestinal diseases:

A. Peutz-Jeghers syndrome

Peutz-Jeghers Syndrome (PJS) is an autosomal prevailing condition with GI polyposis marked by the presence of mucocutaneous hyperpigmentation. Clinical diagnosis involving small bowel polyposis and mucocutaneous melanotic pigmentation, and a family history of PJS needs histological identifiable evidence of intestinal hamartomatous polyps in conjunction with 2 out of 3 additional clinics criteria. Lips (95% of patients) are mostly affected by lesions (59%) and oral mucosa (83%) (see the images below). The palms of the hands, fingers, nose, gingiva, lids, and hard palate are included in other impacted destinations as frequently as possible.

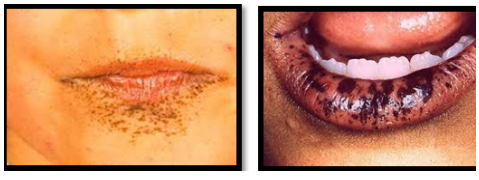


Fig-22: Peutz-Jeghers syndrome

GI PJS symptoms include the proximity of many hamartomatous polyps most frequently occurring in the jejunum (see the image below). The stomach, duodenum, ileum, and colon are further destinations. Side effects can include intussusception stomach pain, dysfunction, rectal prolapse, or impediment. In other words: 2-3% of patients create GI-carcinoma throughout their lives, a repetition that caused a protracted evacuation of all big polyps in patients suffering from the upper endoscopy and the lower endoscopy of the two. [60],[61]

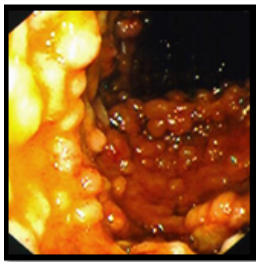


Fig- 23: Multiple large intestinal polyps in a patient with Gardner syndrome.

A. Henoch-Schönlein purpura

This small artery vasculitis shows palpable purpura, arthralgia, stomach symptoms, and glomerulonephritis clinically. [62],[63] Usually in early infancy, the diagnosis can also occur in infancy or adulthood. [64] The illness is believed to be attributable to the immunological complexes mediated throughout the entire body by immunoglobulin A (IgA). The lesions of the skin may occur in groups and some circumstances may be vesicular or ulcerative. Glomerulonephritis with proteinuria, minute blood cell casting, and often red cells form Renal abnormalities in patients with this condition (see the image below). Symptoms of gastric enterology reported in more than 50 percent of Henoch-Schönlein purpura (HSP) patients are colicky abdomen discomfort, diarrhea/constipation, covert bleeding, or even open bleeding, and uncommon intussusception/perforation. [65]

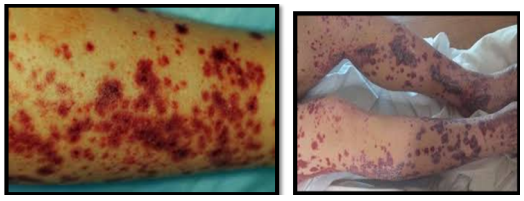


Fig- 24: Henoch-Schönlein purpura

B. Dermatitis herpetiformis

Dermatitis herpetiformis (DH) is a serious skin lesion pruritus, but some are virtually entirely asymptomatic. Pruritus can develop to substantial excoria and subsequent alterations, caused by an atopic dermatitis misdiagnosis (see the images below). [66] 20% of DH patients suffer from clinically open celiac disease, patients suffer from weight loss, diarrhea, bloating, and steatorrhea. Chronic malabsorption difficulties can eventually lead to anemia in iron or folate. [67],[68]

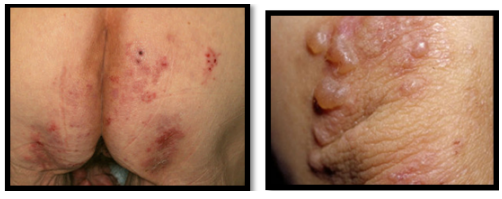


Fig- 25: Dermatitis herpetiformis

C. Blue rubber bleb nevus syndrome

This is a mix of skin vascular defects and GI hemorrhage caused to vascular defects. [69],[70] may be presented in three forms:

- "Non-tender soft nodules that when compressed leave behind a blue empty sac that refills rapidly with blood (blue rubber nipple)"
- "Blue-black punctate tender macular lesions widely distributed on the extremities and trunk"
- "Large hemangiomas (up to 10 cm in diameter) that may interfere with important limb or organ function(see the images below). The GI manifestations of the blue rubber bleb nevus syndrome involve the presence of vascular malformations found most frequently in the small intestine and colon (although lesions have been identified in the mesentery, lung, liver, eye, and CNS.)"

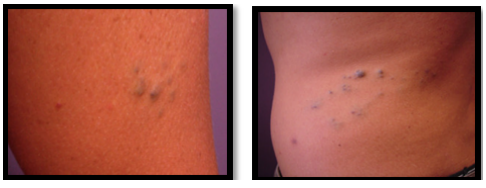


Fig- 26: blue rubber bleb nevus syndrome

6. Cutaneous manifestation of Large Intestinal diseases

A. Familial adenomatous polyposis (Gardner syndrome)

B. Muir-Torre syndrome

Epidermal cyst, osteoma, and adenomatous polyposis are all three of Gardner's syndrome. [71] The cysts are present on the lower limbs, faces, scalps, and upper limbs.

Muir-Torre syndrome (MTS) is a variable expression autosomal

dominant condition characterized by colonic malignant cutaneous lesions. [72] Various skin abnormalities are reported in MTS including adenoma, epithelioma, carcinoma, carcinoma, and keratoacanthomas. [73] In a vast number of patients, visceral cancers occur most commonly in the colon (51 percent of all primary tumors). The larynx, small intestines, stomach, cervix, eyelet, ureter, kidney, and bladder are also attracted by cancer sites.

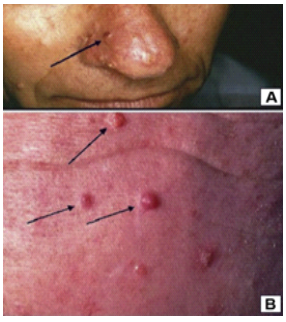


Fig- 27: Muir-Torre Syndrome (MTS) science direct .com

C. Cowden disease (multiple hamartoma syndrome)

Cowden disease is a rare autosomal disease that is characterized by various tissue hamartomas. Dermatology findings include trichilemmoma (that is, skin-colored papules around apertures), acral keratosis, and verbal papillomas. An array of malignancies, breast counting, thyroid, endometrial, cervical, and colon cancer are associated with the virus. Up to 95% of people with Cowden's illness experience colonic polyps. [74] Despite polyps accumulating inside the esophagus, stomach, ball bladder, and intestine, the most prevalent sites of polyposis are the colon and rectum.

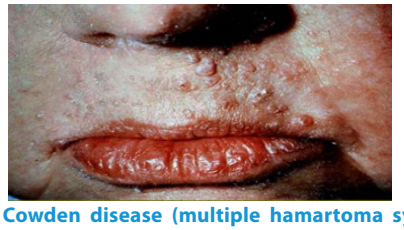


Fig- 28: Cowden disease (multiple hamartoma syndrome) medicines. medscape.com"

D. Bannayan-Riley-Ruvalcaba syndrome

Genodermatosis of the typical three groups of macrocephalous, genital, and bowel polyposis. Vascular abnormalities, lipomatosis, punctured penis lentigines and vulva, face acanthosis, such insults, and multiple acrochordons form part of mucocutaneous findings. There has been noticeable hamartomatous polyposis, with an inclination towards the colonic or rectal location, anywhere along the GI tract. The polyps can be extremely large, leading to intussusception and blockage.

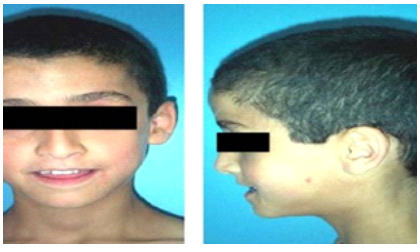


Fig- 29: Bannayan-Riley-Ruvalcaba syndrome researchgate.net"

E. Cronkhite-Canada syndrome

This tetrad has diffuse baldness, nail atrophy, and GI polyposis. This is a diffuse tetrad. [75],[76] Maculations and plaques with hyperpigmentation generally appear on the upper extremities, although can be diffused. Dystrophy of the nail. Nail. The alopecia described with this condition (>95 percent) begins quickly, with a progression from patchy hair loss to final hair loss. [77] The symptoms of GI include diarrhea, substantial loss of weight, and abdomen pain. Hamartomatous polyps are very common in some circumstances, with development into cancer.

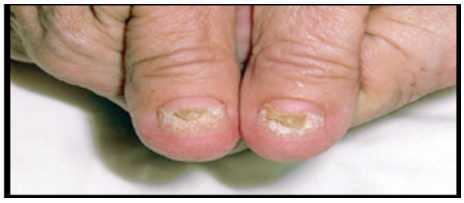


Fig- 30: Cronkhite-Canada syndrome nature.com

Inflammatory bowel disease

Crohn's disease (CD) and ulcerative colitis are the major two illnesses of inflammatory bowel disease (IBD) (UC). The CD is clinically characterized by fever, pain, fatigue, and diarrhea, which can or cannot become bloody. IBD is related to an immense diversity of dermatological abnormalities. The disease can occur from the mouth into the anus in all sections of the GI tract. While UC disease is more common in people of similar age groups with bloody diarrhea and abdomen distension. IBD is quite common with cutaneous symptoms. [80] [79], [80] Four primary etiological groupings can classify the cutaneous findings. The groups have disease-related skin manifestations, reactive skin manifestations, comorbid illnesses, and secondary skin manifestations due to IBD or IBD therapeutic consequences (see Table 3 below).

Table 3. Dermatologic Manifestations of Inflammatory Bowel Disease

"Disease-Specific Cutaneous Manifestations"	"Metastatic CD"
"Reactive Cutaneous Manifestations"	"Erythema nodosum, pyoderma gangrenosum, Sweet syndrome, leukocytoclastic vasculitis, aphthous ulcers, pyostomatitis vegetans"
"Associated Cutaneous Disorders"	"Psoriasis, hidradenitis suppurativa, vitiligo, acquired epidermolysis bullosa"
"Secondary Cutaneous Disorders Due to Complications of IBD or IBD Therapy"	"Acrodermatitis enteropathica (see below), skin cancers, psoriasis"

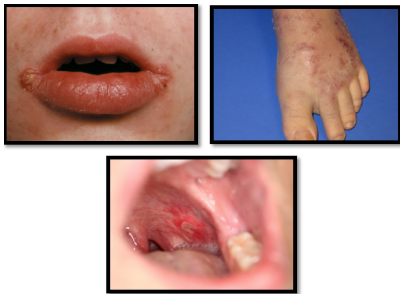


Fig 31: Crohn cheilitis (Angular cheilitis), Metastatic Crohn disease and Oral Crohn disease (Aphthous ulcerate)

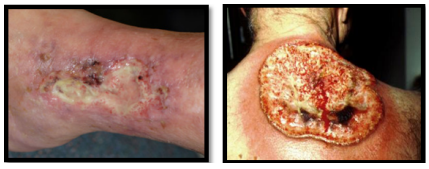


Fig-32 :Pyoderma gangrenosum. Note the rolled-up, edematous, and undermined border with the surrounding halo of bright-red erythema. The base of the ulcer contains a fibrinopurulent exudate."

Conclusion

In many illnesses, the accompanying implication of GI and skin systems is usually noticed. We examined the dermatologist, gastroenterologist, and other disciplines for the early diagnosis and therapy of these individuals in their skin symptoms of GI disease.

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