Treatment of Immuno-blistering Diseases in Elderly: A Two-edged Sword

Speaker: Dr Li Wing Man
Supervisor: Dr Luk Ka Hay James
Geriatric team
Department of Medicine
Queen Mary Hospital
CSP  M / 76

• Ex-smoker

• Premorbid state
  – Walked with stick
  – ADL partially dependent

• Social history
  – OAHR
  – Good family support (wife, 1 son & 1 daughter)
Past medical history

- Hypertension
- Diabetes Mellitis
  - Last HBA1c 8.2%
  - Started on insulin 2 yrs before
- Chronic renal impairment
  - ? DMN
  - Baseline Cr 200-250

*Pt was followed up by Geriatric clinic*
Current Medications

- Amlodipine 10 mg daily
- Perindopril 8 mg daily
- Prazosin 0.5 mg bd
- Protaphane HM 36 units om / 12 units pm

* No addition of new medication in the past two years
History of present illness

• Severe generalized itchiness x 6 months
• Urticarial lesions over limbs x 2 months
• Blisters formations
  – Initially over feet, forearms and hands
  – Apparent since 2 weeks ago
  – Generalized later to affect trunk
• No oral / eye / genital ulcer
Physical examination

- Afebrile
- BP 180 / 60
- Hstix 11.5
- Multiple **tense, intact blisters** over 4 limbs
- Few areas of **erosions** on distal limbs
- **No mucosal lesion**
- Scattered **urticaria-like plaques** on limbs
Investigations

- **WCC** 17.5 x 10⁹/dL  
- **Neu** 9.5 x 10⁹/dL  
- **Eos** 6.2 x 10⁹/dL  
- **Hb** 8.9 x 10⁹/dL  
- **Plt** 156 x 10⁹/dL  
- **Cr** 289 umol/L  
- **LFT** normal  
- **ANA** –ve  
- **CEA, AFP, PSA** unremarkable
Investigations

• Anti-skin Antibodies (ASA): Positive

• ASA pattern: Dermo-epidermal junction (DEJ)

• Skin biopsy (Rt thigh)
  - Subepidermal clefting
  - Abundant eosinophils in the blisters
  - Immunoflorescent studies showed linear deposition of IgG, C3 along at dermo-epidermal junction (basement membrane)
Progress

- Diagnosis: Bullous Pemphigoid
  * Positive direct and indirect IF

- Started on super-potent topical steroid
  (Dermovate®; clobetasol propionate 0.05%)

- Minocycline 100mg BD as adjuvant Tx

- Daily wound dressing
Progress

- No new blister formation in the next week
- Erosions showed satisfactory healing
- Patient was discharged 3 weeks later
- Maintenance Tx: Topical steroid + Minocycline
- Daily wound dressing at OAH recommended
CSP   M / 76

• Readmitted 2 months later

• Worsened skin condition
  – Generalized blisters formation
  – Extensive erosions all over
  – No mucosal lesion

• Dermatology team consulted
  – Started on oral prednisolone 30 mg daily
    (0.5mg/kg/d)
  – Minocycline, Dermovate® continued
Progress

• Transferred convalescence hospital
  – Active rehabilitation; daily wound dressing

• Uncontrolled skin condition
  – New blisters formation despite P30
  – ↑Prednisolone 45 mg daily (0.75mg/kg/d)

• Stabilized; blisters gradually resolved

• Azathioprine as steroid-sparing agent

• ↓Prednisolone 10 mg daily upon d/c
CSP  M / 76

- Adm x  **Symptomatic anemia  3 /12 later**

**Investigations**

- CBC  Hb  $5.8 \times 10^9$/dL  L
  - WCC  $2.1 \times 10^9$/dL  L  ANC  $0.8 \times 10^9$/dL  L
  - Plt  $58 \times 10^9$/dL  L
- Retic  $0.1 \times 10^9$/dL  L
- Serum Fe  normal
- Stool x OB  -ve x 2
Progress

• Given PC transfusion
• Pt refused BM examination
• **Hematology team consulted**
  – Thiopurine methyltransferase (TPMT) level checked → 6.7 U (LOW)
  – Pancytopenia most likely due to marrow suppression by azathioprine
  – **Azathioprine stopped**
• Monotherapy with prednisolone 10mg daily
• Stable skin condition; FU at Dermat clinic
• **Major flare** of bullous pemphigoid 1/12 later
  – Painful, tense blisters all over
  – Extensive erosion affecting 30% BSA

• Patient’s son expressed **difficulty in dressing / wound care** (by OAH staffs / family)

• Dermatology team consulted
  – ↑Prednisolone 30 mg daily
Progress

• Skin condition gradually improved

• Poor diabetic control
  – H’stix: 14.1 – 26.0
  – ↑Actrapid HM 14 u om / 16 u noon
  – Kept Mixtard 30 HM 14 u before dinner

• Persistent suboptimal control requiring assessment by Endocrine team

• Changed to basal bolus regime with Actrapid HM tds, Glargine Nocte
Progress

• Skin swab: **MRSA, Pseudomonas aeruginosa**

• **Microbiology & Dermatology team consulted**
  – No sign of active infection
  – Not for systemic antibiotic
  – Regular disinfection by KMnO4 1:8000

• **Convalescent care**

• ↓*Prednisolone 20mg daily* upon d/c
**CSP  M / 76**

- c/o Lt knee pain and admitted 1/12 later
- High fever ++
- Chills and rigor
- P/E: Lt knee effusion ++ increased warmth
- Left knee aspiration by Orthopedic team
- Joint fluid: **Cell count > 100,000**
  - Culture: MRSA
- Blood C/ST: **MRSA**
Progress

- **Diagnosis:** Septic arthritis; MRSA septicemia
- Started on vancomycin
- **Emergency operation**
  - Arthroscopy & arthrostomy performed
- **Kept in-patient care at Orthopedic ward**
  - Persistent low grade fever x 1 mth post-op
  - CRP on rising trend
Progress

- **Gallium scan** arranged
  - mild increased uptake over cervical spine
  - ? infective spondylitis at C4/5

- **Microbiology team**
  - Planned for prolonged Tx with vancomycin

- **Dermatology team**
  - Decrease P20 $\rightarrow$ P10 (active sepsis)
Progress

- Progressively worsened limb power bilaterally
- Subacute deterioration over one week
  - UL / LL power decreased from 4/5 to 0/5
- Brisk jerks; equivocal plantar reflex

Urgent MRI cervical spine
- Spondylodiskitis at C4/5
- Epidural collections with cord compression
- Subligamentous prevertebral inflammatory collection at C1/2 to C6/7 level
Progress

Emergency operation
- Anterior and posterior spinal fusion performed
- Operative findings: Septic spondylitis

Partial neurological recovery
- Limbs power 2-3/5

CRP / ESR on improving trend
- Active rehabilitation by PT / OT
Progress

• Suboptimal control of bullous pemphigoid since post-operatively
• Scattered large blisters on limbs and trunk
• **Microbiology / Dermatology team consulted**
  – Further escalation of steroid not favorable
  – IVIG as adjuvant therapy / facilitate steroid tailing
CSP  M / 76

- Given a course of IVIG
  - Total dose: 2 gm/kg
  - Administered in 4 days (0.5gm/kg/d)
- Skin stabilized in 3 weeks’ time
- ↓Prednisolone 5mg daily in one month
- Now under active rehabilitation at convalescence hospital
Bullous Pemphigoid

Review
Bullous pemphigoid

• Most common autoimmune blistering disease
• Predominantly affects the elderly
• Age of onset > 65 years
• Annual incidence: 6-7 per million
• Relative risk:
  – 300-fold increased risk in pts > 90 yo (c.f. 60 yo or younger)
Pathogenesis

- Immune-mediated disease (cellular + humoral)
- 2 principle antigens
  - Adhesive protein at basement membrane
  - Dermo-epidermal junction (hemi-desmosomes)
  - BP 180 (BPAg2) & BP 230 (BPAg1)
- Most patients have circulating Ab against BP 180
- BP 180 / BPAg2 → Type XVII collagen
- Autoreactive T-cell response to BP 180 & BP 230
  → B cell response in producing autoantibodies
Pathophysiology

• Binding of autoantibodies (IgG) to target Ag
• Cascade of immune events
  – complement activation
  – recruitment of polymorphs (Eos + Neu)
  – liberation of chemokines and lytic proteases
• Destruction of the hemi-desmosomes
• Subepidermal blister formation
BP - Clinical features

• Polymorphic cutaneous features
  1. Pre-bullous phase
  2. Bullous phase
  3. Erosions
  4. Secondary infection
  5. Post-inflammatory changes
1. Pre-bullous phase

- Prodromal phase
- Intractable *pruritus*
- Edematous, *urtcarial* lesions
- Non-specific eczematous lesions sometimes

**Distinguishing features**

- Arcal distribution
- Persistence of lesion
2. Bullous phase

- Urticarial plaques $\rightarrow$ Vesicles $\rightarrow$ Bullae

- Tense blisters
  - Up to 1-4 cm in diameter
  - Overlying urticarial / inflammatory base
  - Contain clear fluid
  - Persist for days, leaving eroded or crusted areas
2. Bullous phase

- Acral, symmetrical distribution
- Mucosal involvement
  - 10 - 30% of patients
  - Most common in oral cavity
  - Mucosae of the eyes, nose, pharynx, esophagus and ano-genital region are more rarely affected

* Cicatricial Pemphgoid
  - Variant of BP
  - Predominantly affecting mucosa (eye, oral cavity) with prominent scarring and scalp involvement
  - Principal Ag: BP 180
Pre-bullous / Urticarial lesions

- Intact / Tense blisters
- Tense (roof = whole epidermis)
- Clear-fluid-containing
- Ruptures to form open wound

→ Intractable pruritus
→ Precedes blisters by wks to mths
→ Persistent (c.f. true urticaria)
Bullous Pemphigoid

3. **Erosions**
   - Take weeks to heal
   - Infection-prone (large open wound)
   - Scarring: damage at dermoepidermal junction

4. **Secondary infection**
   - Staph. aureus, Gp A Strep.
   - Chronic: **MRSA**, Pseudomonas aeruginosa
Bullous Pemphigoid

5. Post-inflammatory changes

• Scarring
  – DEJ affected
  – Loss of hair follicles / skin appendages
  – Absent sweating / heat-intolerarance

• Dyspigmentation
  – Cribiform hyper- and hypo-pigmentation
BP – lesions of different stages coexist

- Pre-bullous urticarial lesions
- Tense blister rest on urticarial base
- Scarring & hyperpigmentation
- Erosion – Open wound
BP – Post-inflammatory changes

- Hyper- / Hypo-pigmentation in cribiform configuration
- Extensive scarring & dyspigmentation
Peripheral Eosinophilia

• Up to 50% of pts with BP; can reach >10 x 10⁹
• Eos count NOT correlates with ds activity

* Highish Eos count in elderly
  – Bullous pemphigoid
  – Scabies infestation
  – Drug eruption
  – Others: Parasitic infection / Hoghkin’s lymphoma / Vasculitis / Hypereosinophilic syndrome
BP - Diagnosis

- Age
- Typical clinical features
- Compatible histologic features
- Peripheral eosinophilia

- **Gold standard of diagnosis**
  - Positive findings of BOTH *direct* and *indirect* immunofluorescence (IF) microscopy study
BP – Skin biopsy (Direct IF)

- Performed on formed blister
- **FRESH** specimen should be sent (Not in formalin)

**Histopathological examinations**

1. H&E stain
   - Subepidermal clefting
   - Predominant Eos infiltrate within blister

2. Direct Immunofluorescence studies (DIF)
   - DIRECT visualization of IgG / C3 at the dermo-epidermal junction (BMZ) within the biopsy specimen
BP – Serology (Indirect IF)

- Anti-skin antibodies (ASA)
- To detect the presence of auto-Ab in pt’s serum
- Methods
  - Blood taken from pts
  - Incubated with substrates i.e. monkey oesophagus
  - Substrate specimen examined histologically
  - Demonstrate the presence of IgG deposition at the DEJ of the surrogate specimen

* 2 patterns in +ve ASA
  - DEJ (dermoeipidermal junction) → Bullous pemphigoid
  - ICS (epidermal intercellular substance) → pemphigus
Indirect IF

- ASA: Sensitivity 70-80%; Specificity 60-70%

Enzyme-linked immunosorbent assay (ELISA)

- Emerged as additional diagnostic technique
- Higher sensitivity & specificity
- Identify specific antibodies implicated
  - BP180 / BP230
  - Distinguish other immuno-blistering ds with auto-Ab against DEJ molecules i.e. Epidermolysis Bullosa Acquisita (Ab against Type 7 collagen), Paraneoplastic pemphigus (Ab against plakins)

- Cost / Availability
Our patients...

- Delayed presentation
  - Carer unaware / neglect non-specific symptoms
    - Family / OAH staffs / Attending doctors
    - Pre-bullous phase
      → intractable pruritus alone
      → urticarial eruption
      → confusion with ezema / scabies infestation
Our patients...

• **Refusal** of diagnostic skin biopsy
  – Direct IF - Gold standard for diagnosis
  – **Under-utilized**
    • Regards as too invasive by family
    • Doctors not familiar with procedure
  – **Technical error** – Sent in formalin

* Diagnosis by typical S/S + compatible indirect IF acceptable
** Skin biopsy advocated in atypical cases
BP - Management

• Goal of management
  – Retard disease progression
  – Facilitate wound healing (minimize scarring)
  – Prevention of infection (cause of mortality)
  – Symptomatic relief (esp. pruritus)

* Quick and efficient therapy is important to prevent long term complications like scarring and infection
Special considerations in Geriatric patients

1. Diagnostics
   - Neglect / unawareness of symptom (pre-bullous phase)
   - Reluctance of confirmatory procedures (direct IF)

2. Topical treatment
   - Labour-intensive (carers in OAH / family)
   - Right amount
   - Rt location (BOTH blisters & erosions)

3. Systemic immunosuppression
   - ↓Dosage (impaired metabolism / excretion)
   - ↑Infective risks
   - ↑Metabolic complications (e.g. osteoporosis, electrolytes disturbance)
Special considerations in Geriatric patients

4. Wound care
   - Polymorphic natures (dry / wet / infected)
   - Bed-bound state / Hygiene - colonization
   - Cross-infection: OAH residents / staffs / admissions

5. Nutrition
   - Hypoalbuminemia
   - Dehydration from extensive raw areas

6. Social issues / Carer stress
   - Natural course - frequent flare
   - ? Admission
   - ? Contagious
BP - Evidence-based Treatments

• A recent Cochrane review of the 7 RCTs concluded the following:

(1) very potent topical steroids are effective and safe treatments for BP

(2) their use in extensive disease may be limited by side effects and practical factors
BP - Evidence-based Treatments

(3) starting doses of prednisolone > 0.75 mg/kg/d **DO NOT** seem to give additional benefit in disease control

(4) the effectiveness of the addition of plasma exchange or azathioprine to corticosteroids has **NOT** been established

(5) combination treatment with **tetracycline** and **nicotinamide** may be useful
Management in BP

• **Questions to be answered:**

  ➢ What is the **optimal route** of administration of corticosteroids and **dose** for newly diagnosed BP?

  ➢ Who should be treated with **oral vs topical** corticosteroids vs **steroid-sparing drugs**?

  ➢ Are the **steroid-sparing agents** safe & effective?
Systemic Corticosteroid

• The type of steroid, dosage, and duration of therapy varies among clinicians

• The optimal starting dose for oral prednisolone should be no higher than 0.75 mg/kg/d

• The formulation of steroid does not seem to matter, as a study comparing methylprednisolone versus prednisolone found no significant difference
Systemic Corticosteroid

• Long duration / high dose may be required in extensive diseases

• Systemic corticosteroids are responsible for multiple adverse effects, which can be detrimental and potentially fatal in the elderly

• Minimal effective dose should be used
Topical Corticosteroid

- Topical corticosteroids has been evaluated as possible way to avoid systemic corticosteroids in BP

- Landmark study by a French group
  - Pts randomized into topical vs oral steroids
  - Treatment with super-potent topical corticosteroid i.e. clobetasol propionate 0.05% (40 g/d) was associated with improved overall survival ($P = .02$) and fewer severe adverse effects ($P = .006$) than oral prednisone in patients with extensive BP (presence of $> 10$ new blisters/day on 3 consecutive days)
Topical Corticosteroid

• Another prospective, randomized study
  – Multi-center study in France
  – Recruited 312 pts with moderate or extensive BP
  – 1-year follow-up
  – Compared the efficacy and safety of 2 dosings of clobetasol propionate 0.05%
    (1) 40 g/d reduced over 12 months
    (2) 10-30 g/d reduced over 4 months
Topical Corticosteroid

• *No difference* in the rate of BP control or event-free survival occurred between the two treatment groups

• *Strong beneficial effect* of the mild regimen versus the moderate regime, with reduction of mortality rate and serious side effects (e.g. sepsis, hyperglycemia etc)
Topical Corticosteroid

• **Practical limitations**
  – need to apply the cream on almost the entire body!!!
  – the assistance of either a relative or a nurse is often required

• **Possibility of systemic side effects**
  – Augmented absorption into blood stream with open wounds

• **Much higher costs than oral Tx**
Systemic Corticosteroid

• Typical starting doses: 0.5 – 0.75 mg/kg/d
• Initial starting dose depends on the severity of disease and patient comorbidities
• Continue the effective dose until the cessation of active inflammation, new blister formation, and pruritus **for at least two weeks** and **80% of existing blisters** have healed
Systemic Corticosteroid

• Vitamin D, Calcium supp. needed
• Slow tapering of over several months (5mg every 1-2 wk)
• Oral maintenance often needed
  – Prednisolone 5 – 7.5 mg/d
  – Life-long terms
  * No available data in the literature to support / reject use
• Steroid-sparing agents
  – Often NOT required
**Tx response in BP**

- The level of BP180 antibodies (ELISA) in serum usually correlates with the clinical activity of bullous pemphigoid.
- Discontinuation of treatment may be attempted once patients have remained in **complete remission on minimal therapy** (e.g., ≤0.1 mg/kg per day of prednisolone or ≤20 g of clobetasol propionate per week) for at least two months.
Adjuvant Tx - Immunomodulation

Anti-inflammatory agents

• Tetracyclines, macrolides, dapsone
• Efficacy only demonstrated in case series
• Typically considered when a steroid–sparing regimen is required with mild, localized diseases (few new blisters / day)

* In these pts, the risks of immunosuppressants often outweigh the benefits
Adjuvant Tx - Immunomodulation

• Tetracycline group
  – Tetracycline 500 mg QID
  – Doxycycline / Minocycline 100 mg BD
  *Tetracycline – difficult dosing
  **Minocycline - Cost

• Common side effects
  – Photosensitivity (esp. over inflammed skin)
  – Gastrointestinal upset
  – Mild LFT derangement (Bld test recommended)
Adjuvant Tx - Immunomodulation

• Macrolides
  – Erythromycin 500mg QID
  – Clarithromycin 500mg BD

• Dapsone
  – Inhibition of polymorphs (esp Neu, Eos)
  – Sulphur-containing \( \Rightarrow \) drug eruption / DRESS syndrome
  – Check G6PD status
Steroid-sparing Tx

• Several agents have been proposed as adjuvant therapy to corticosteroid in BP as a mean to reduce the cumulative steroid dose

• Despite the beneficial intention, iatrogenic complications such as immnosuppression, susceptibility to infections, and renal or hepatic injury are not uncommon
1. Azathioprine

- Although there have been reports of improvement in disease severity with adjunctive use of azathioprine, evidence from RCTs is lacking.
- **Empirical use** as steroid-sparing agent.
- Dose at 2 mg/kg/d (0.5-2.5 mg/kg/d) typically used.

**Adverse effects**
- Myelosuppression
- Hepatotoxicity
- Hypersensitivity syndrome
Azathioprine - Safety

• Thiopurine methyltransferase (TPMT) level should be checked prior to the initiation of azathioprine to determine the appropriate dose and minimize risk for myelosuppression.

• TPMT
  – involves in metabolism of Azathioprine
  – Deficiency level → multiply myelosuppression
**Azathioprine - TPMT**

- **Maximum doses in patients...**
  - High TPMT (>19 U) → 2.5 mg/kg/d
  - Medium TPMT (13.7-19 U) → 1.5 mg/kg/d
  - Low TPMT (5-13.7 U) → 0.5 mg/kg /d

- Initiation of Azathioprine should be **extremely cautious** in pts with low TPMT level

- Myelosuppression can also occur even with normal / high TPMT levels (**xanthine oxidase** pathway; no serological test a/v)
Azathioprine - Safety

- Other potential adverse effects include malignancy, gastrointestinal disorders, and infections

- **Serological surveillance**
  - complete blood count, liver / renal function tests
  - every two weeks for the first three months
  - every two to three months thereafter
2. Mycophenolate mofetil (MMF)

- **Evidence??**
  - The only randomized trial of MMF in BP was an unblinded randomized trial of 73 patients.
  - Compared treatments with methylprednisolone (0.5 mg/kg/d) plus MMF (2 g/d) to methylprednisolone (0.5 mg/kg/d) plus azathioprine (2 mg/kg/d).
  - The two regimens appeared similarly effective.
MMF in BP

- Typical dosage: **1.5 to 2 g/d** in divided doses
- **Maximal dose of 3 g/d**
- Less marrow / hepatic toxicity compared to azathioprine
- **Common adverse effects**
  - gastrointestinal upset
  - Herpes zoster infection (esp. in elderly)
  *Anti-viral prophylaxis (e.g. acyclovir / valacyclovir) often prescribed*
  **Beware of mental confusion of acyclovir / valacyclovir in elderly esp. with impaired RFT**
3. Methotrexate

- No randomized trials have evaluated the efficacy of methotrexate in bullous pemphigoid

- Case reports and uncontrolled studies suggest that methotrexate may be beneficial when used alone or in combination with topical corticosteroid or systemic glucocorticoid therapy
MTx in BP

- Typical dosage: **5 - 20 mg/wk**
- In **elderly** pts, a **low dose (5 mg/wk)** is used as initial therapy and slowly titrated upward

- **Folic acid** supp. to reduce the risk for hepatic toxicity (daily or weekly dose; to be administered 3-4 days after MTx in case for weekly dosing)

- **Adverse effects**
  - hepatotoxicity
  - pulmonary fibrosis
4. Immunoglobulin (IVIG)

- No head-to-head comparison to demonstrate superior efficacy over systemic corticosteroid
- Conclusive evidence from case series to show that IVIg is an effective treatment in BP patients refractory to steroid therapy
- **Therapeutic role:** neutralizing the pathogenic auto-anti-skin antibodies
- **Common dosage:** 2 – 3 g/kg in divided doses over 3-4 days
**IVIg in BP**

- **Safe in general; no infective complication**

- **Potential adverse effects**
  - Anaphylaxis
    - Rare; in case of selective IgA deficiency (<1/700)
    - Check Ig pattern in elective cases (decreases IgA)
  - Allergic reaction (pruritus, skin eruption)
  - Fluid overload in **elderly** (lower dose, slower infusion rates)
  - Renal tubular toxicity (rare)
  - Aseptic meningitis (rare)
**IVIg in BP**

- Administered in monthly course (2-3g/kg)
- No guideline / evidence to suggest duration of treatment
- Usually 6 courses with gradual spacing of Tx
- Single course maybe used as salvage Tx
- Cost +++ (HK $600/3g vial)
  - Average cost $20000 – 30000 per course
5. Other treatment modalities

• Cyclosporin, Cyclophosphamide
  – NO conclusive evidence on beneficial role
    (i) Improving outcome (survival, ds-free duration) in combination tx with steroid
    (ii) Quicker reduction of steroid dose
  – Associated renal toxicity / hypertension in CycA; Hemorrhagic cystitis / subfertility in CPH

• Plasmapheresis, Rituximab (anti-CD20)
  – experimental uses in refractory cases in individual reports and small series
BP – Geriatric patients

PRACTICAL TIPS
BP – Social aspect

• Carer stress
  – Effort wound care
  – Frequent flare of disease
  – Decision on admission
  – Worry of contagious disease

• Thorough education
  – Preferrably provided by geriatrician / specialty nurse
  – Natural course of BP – flares, infection-prone
BP – Care at OAH / Home

• Drug compliance

• Daily wound care
  – To open wounds until re-epithelization
  – KMnO4 (1:8000) as soak; @ 5 – 10 mins; 2x/day
  – KMnO4 $\rightarrow$ dilution (pink color)
    $\rightarrow$ Purple: too concentrated (irritated / stain)
  – Alternatives: Hibitane / Chlorhexidine

• Awareness of signs of infection
  – Increased amount / purulence of discharge
  – Fever / Decreased GC
BP – Wound care

• Polymorphic natures
  – Open wound / Raw areas
    • Weeping + Discharge +
    • KMnO4 useful → anti-septic, hasten ‘drying-up’ of wounds
    • Light dress >> occlusive dress (infection-prone)
    • Change dressing every at least once a day
    • Optional
      – Silver impregnated dressing → prevent infection
      – Algae-containing dressing → excessively weeping wound
  – Re-epithelized, dry wound
    • Moisture-rich dressing: Vaseline gauze, Aquacel®
    • Eschar should be chimmed (Stoma nurse)
BP – Wound care

– **Infected wound**
  - Topical antibiotic – fusidin, bacitrcin mupiricin
  - Systemic antibiotic
    - Skin flora / Staph. Aureus
      » Cloxacillin, amoxicillin-clavulunate, cefuroxime
  - Chronic wound
    » Anti-pseudomonal antibiotics
    » Colonization vs true infection (signs)

– **Scarring**
  - Heat dissipation (loss of sweat glands)
  - Liberal use of emollient (↓ elasticity)

– **Pain control**
Our role as geriatricians

• **CGAT**
  
  – Management of **mild to moderate** cases
    • Localized wounds (confined to limbs)
    • No systemic upset / Non-septic
    • Controllable with topical steroid

  – **Useful prescriptions**
    • Potent topical steroids (LA BD)
      – **Dermovate**®, Diprosone®, Betnovate®
      – Ointment → dry wound; Cream → weepy wound
      – Potency: Ointment > Cream

    • **Diprogenta**® → Betamethasone + gentamycin
      – Problem of resistance in long term use

KM: 24.1.3000 – 10000
Our role as geriatricians

• Admission / Dermatologist referrals
  – Acute flare requiring systemic treatment
  – Uncontrolled disease (>10 new blisters /day)
  – Extensive open wounds / raw areas
    • In-patient dressing care (covalescence hospital when stable)
    • Systemic antibiotic
  – Need for titration of immunosuppressants
  – Need for IVIg
Our role as geriatrician

• Education
  – Disease course
  – Dressing care
  – Adverse effects of treatments
  – Prognosis
    • High chance of sepsis
    • Overall high mortality (all-cause mortality in 1 yr: 30%)
Summary

• BP is the commonest autoimmune blistering disease in elderly
• Patients may present with non-specific pruritus and urticarial lesions at early stage
• Diagnosis is made by positive direct and indirect immunofluoresence studies
• Corticosteroid is the mainstay of treatment in bullous pemphigoid
Summary

• Topical corticosteroid is proven to be as effective as systemic treatment in mild to moderate cases
• Heavy immunosuppression may be required in refractory cases and associated with serious septic complications
• IVIg is a safe, adjuvant therapy to steroid in bullous pemphigoid
Thank you