Quick reference & rarities

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A summary of everything including rarer stuff! Originally collated by Miss Amy Robinson.
Last updated: May 2023

Adjuvants in one page

EBCTG 2012: 100K woman, 123 trials. Reduced mortality – Anthracyclines (over CMF) = 6.5% reduction, + T further 4.6% (independent of size/grade/nodes/ER). Dose-dense regimens- 2.4%. Most benefit = luminal-B (PR<20%, Ki67hi). Rx breast cancer death in <50yrs and >50yrs is reduced by 38% and 20%. cPR + OS= GeparTRIO trial was the first, NeoBC Meta-analysis, BrightNESS (DFS), Yau et al looking at residual cancer burden – correlates with survival.

Her2 disease

Massive shift in survival for these patients – remember HER2 = 31= NSABP-31 (N=0), N9831 (included N0) = 40% DFS, 37% OS! RR CHF 5.1% reduced LVF, RR1.83 but reversible. Dual therapy superior to monotherapy = CLEOPATRA 19% rx reduction (metastatic) people will dual for a hotty like that! Dual in NAC = NeoSPHERE (pCR 45.8% vs 29%) and safe (APHINITY, TRYPHAENA, BERENICE), Drop anthracycline = TRAIN2 pCR 67% vs 68%! TDM-1 if not complete response = KATHERINE = Kadcyla = herceptin emtansine (Delivers a cytoxix =DM1 via hitch on traztuzamab). New – DYNASTY TRIALS – DERUXTECAN 50%! Metastatic setting. These are a TOP1 "payload". HER2CLIMB 2020 = tecatanib added to Traztuzumab and Capecitabine in Mets 3rd line.

Triple Negative

Give Carboplatin in NAC = CALBG40603, GepartSIXTO, BrighTNESS (veliparib no benefit). add PD1 Ab – Pembroluzimab = Keynote 522 + t. Upgrade to Capecitabine if not complete pCR = CREATE X. Target PARP- in BRCA mutations = OlympiaA = Olaparib (NB can also use in ER+ BRCA mutation metastatic disease).

ER+ disease

- NATO, EBCTG 2011 – Reduced recurrence by ½ in first 5 years then by 1/3. 30% reduction in breast cancer mortality 15 yrs.
- AI better than Tamox = BIG-1-98/ATAC/EBCTG2015 meta-analysis = ARR breast cancer mortality 2.1%.
- Extend it = aTTom/ATLAS and MA17 (AI after 5 years of tamoxifen). no good evidence for AI for 10 years. Absolute benefit in OS of 2.8%. Most patients get SE in 1st yr.
- Risk stratify for chemo using Oncotype DX = SWOG and Tailor X = No if <11 RS, yes if >25 RS. Spare chemo in N0 post-menopausal intermediate RS score on Oncotype = RxPONDER interim results– still not clear in pre-meonpausal.
- High risk = CDK4/6- = MONARCH-E ***N1 needs additionally G3 or >5cm or >20%ki67.
- OFS – SOFT/TEXT studies –Tamox, OFS + tamox, OFS + exemetasine DFS = 67.7 to 78.7 to 83.4%. Progression on therapy = try SERD fulvesterant or oral = elecestrant = EMERALD. Pi3k mutation – apelesib= SOLAR3. Can target PARP in BRCA2 ER+.

ABBREVIATIONS
F= Fluorouracil, E= Epirubicin (also doxorubicin, both anthracycline), C= Cyclophosphamide, T= Taxane e.g. Doxetaxel/paclitaxel. Action: S phase = F = anti-metabolite. E = Topoisomerase Inhibitors – G2 arrest. T = Microtubules in M phase by reducing tubulin crucial for metaphase to anaphase C (and platinumsnb) = Alkylating agent so attached to alkyl group on DNA and breaks X-linking throughout the cycle. NB: Platinums are alkylating but instead of breaking DNA X linking they form covalent metal adducts with DNA.

Screening and Interval Cancers

Forrest report 1985 (UK trial and Swedish trials) resulted in screening in UK from 1988, estimated 30% benefit. Protagonist = Cochrane review 2011 – 15% benefit only, 30% overdiagnosis! Marmot review in response reported– 1300 lives/yr saved. 20% benefit. Recall rate is 4 per 100, 1 in 100 will have cancer. So must inform patients of this before participating. Meta-analysis in 2018 JNCI assessed impact of tomosynthesis – it will reduce biopsies. But not routine in screening so doesn't yet impact on recall anxiety. Interval cancers are a specific issue. Some figures: About 18k BS detected cancers in the UK/yr. About 6k interval, 80% of which are not seen on prior films. Interval includes any cancer within 40 months of prior screen. This is a formal process. Issues: Duty of Candour to patients if missed (20%), Audit and Quality assurance as per the Screening Quality Assurance Service, planning future services and research. It may be beneficial; for the patient to understand and for duty of candour we should inform them (there is a toolkit specifically for this purpose and disclosure of audit guidance for clinicians). This is also an educational opportunity if it was missed. Finally, for monitoring the sensitivity of screening and inform practice. The database is the Screening History Information Management System. 2 independent reviewers will classify the prior mammo as satisfactory, satisfactory with learning points (e.g. subtle changes that in hindsight can be seen but was not unreasonable to miss) or unsatisfactory. The latter must be reported in a screening incident form! It isn't just the radiologists then, it is the whole process e.g did the MDT make the wrong decision etc.

HRT

The Million Woman Study: HRT and VTE = 2-4-fold increase but not if gel/transdermal. Endometrial cancer risk if oestrogen (x1.5 only or particularly tibolone at x1.8). OCP much higher risk especially if oestrogen containing confers a 3-6-fold increase in VTE (increase VII, VIII and X).

"If a group of **10k women in their 50s** had never taken HRT, **26 women would still get breast cancer** in a year. If all 10k women had recently taken combined HRT for less than five years, **35 would get breast cancer.** So, in this large group of women, the HRT is linked to **9 extra cases of breast cancer in a year. That is less than one in a thousand women**"

- Type associated with cancer– oestrogen alone best but can only be used post hysterectomy> norethisterone combo worst > didrosterone combo best.
- Age – older and take it raises the risk as already places you at risk.
- Timing – taking for over 5 years raises much more, less than 1yr has hardly any effect.

What about after a cancer diagnosis? Meta-analysis of 4 RCTS and 4k patients Poggio et al. Breast Cancer Research and Treatment (2022) 191:269–275 – suggests that HRT does significantly increase recurrent disease ***HR1.8 (CI went up to 2.8) so fair to say approx. 2-fold rx****. This is shared decision making – provide the info the patient will decide. A trial off is a reasonable suggestion and often a compromise. Quality of life matters! British Menopause Society useful resources and NICE provide guidance on alternatives for each of mood swings, secretory-motor symptoms and vaginal dryness.*

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Breast Cancer Rx

The table from companion: Age >55 (x10), BRCA, Benign disease (e.g. LCIS x8), BIRADS mod-high (x4), FDR <50 (x2), HRT 2.3%/yr, Nulliparity at 30 and menopause at 54 = 3%/yr, weight 1%/kg, drink 7%/drink/day, current OCP = 24%! Breast cancer now generations study following 110k woman over 40yrs= smoking bad, exercise good and 350 new gene combinations. Stress and working pattern no effect. Pre-term and pregnancy complications are related as are levels of Mullerian hormone in pre-menopausal. Million-woman study also will provide a lot of insight similar findings. Per 1k women aged 50-59 - 23 cases, BMI>30 = extra 24 cases!

Breast Cancer Genetics

BRCA1 (60-90%, ovary 40-60%), BRCA2 (45-85%, ovary 10-30%, male 10%, panc 24% (x3pop)) and PALB2 (50%*) tested in in anyone under 60 with TN. NEJM 2021 paper 113k women – additional to 3 tested for: ATM (ER+), CHK2 (ER+, x2* and male x10*) v. high, then BARD1, RAD51C, TP53 (x18*).

Surveillance:

- **Li-Fraumeni** Tp53 50% by 50yrs, **MRI yearly from 20yrs old**. DO NOT XRAY!
- BRCA or PALB2; annual **MRI 30-50yrs** (assess BIRADS can be up to 70). Annual **mammo 40-70**.

All others get **yearly mammo from 40 (or can start at 30)**:

- Cowden's (PTEN) 89% have some sort of breast issue, cancer in 50% Think breast, colon and skin.
- Peutz Jeuger's (STK11 and others) 30-50% rate breast cancer
- CDH1 – loss of e-cadherin so lobular breast (50%) and also diffuse gastric cancers (70%)

Links but not for surveillance:

- Gardiner's (APC) FAP + extraintestinal desmoid + osteomas + thyroid + breast cancers (v rarely)
- HNPCC (MLH1, MSH2, MSH6 and PMS2) not certain this does increase rx but book quotes 2 fold.

Risk reduction discussion: First, what is the risk – moderate or high only eligible for rx reduction.

1. Start with the basics: Exercise, alcohol, weight.
2. MRI surveillance evidence shows benefit in high risk of early detection (MARIBS and MRISC)
3. Tamoxifen only in BRCA2. IBIS 1 (Lancet Oncol 2014, 16 yrs fu of 7k women) = Tamoxifen as chemoprevention only effective for ER+ disease – HR0.64 in this subgroup. Hence do not use in BRCA1. Effect continues, constant 29% annual preventive effect 15 yrs+ after completion of 5 yrs treatment. IBIS-2 (12yrs Fu– AI), 36% reduction again in ER+ disease. NNT = 29.
4. *BSO – very poor evidence and likely the main benefit is from preventing death from the ovarian cancer rather than reducing the risk of breast cancer. Logically the effect on the breast is more important in BRCA2 but the risk of ovarian cancer is highest in BRCA1. Systematic review in 2021 found no good evidence to support the practice – there are very contradictory reports.*
5. RR Mx – key is that this does not increase OS but reduces rx cancer by 95%. Factors to consider in all is the FH eg moderate + considered, proven gene carrier can have, surveillance averse, symmetrising if have had other surgery then yes can have.

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The Weird and the Wonderful in two pages

A bit of basics first: Remember there is stromal tissue then the ducts and lobules. Highly specialised apocrine gland. Duct and lobule lined by epithelial cells – Basement membrane then both basal and myoepithelial cells then the luminal cells. **Luminal cells** in the breast normally stain for cytokeratin 8/18/**19**. Basal cancers lack these CKs but express **CK5/CK6,** CK14 or CK17, EGFR, SMA (myoepithelial), P-cadherin, p63 or c-kit.

Angiosarcoma

Rare (0.2% of all breast tumours) presents as radiation induced (RIAS) around 7-10 yrs on average but ranges 1yr-26yrs! Purple nodules on the breast. May have mass. Histology – macro – highly infiltrative, layering of endothelial cells, atypia. Micro - high mitosis +ve for **CD31**, **CD34**, ERG, **VEGF** and factor **VIII**. Very poor evidence as rare – meta-analysis in 2014 EJC of 74 articles and just 222 patients! Royal Marsden 15yrs - 49 cases! Need CT staging. Rarely to LNs so SNB only. Prognosis = size. Surgery is the mainstay of treatment – not chemo sensitive and can't give Radiotx if RIAS. So, aim for R0 margin. Relapse very high and usually within 2yrs at least half recur. 5yr survival 30-50% depending on stage.

Granulomatous Disease of the Breast

Rare 2.4/100k. Idiopathic. mc in Asian, Hispanic, and Arabic. Theories re pathology: autoimmune trigger 2nd to epithelial cell damage, lobular disruption during lactation (also raised in prolactin excess e.g. anti-psychotics), associated with korneybacterium (kroppenstedi, but also TB), autoimmune – associated with erythema nodosum/arthritis. Present with painful mass, erythema, may get fistula or abscess. Radiology mimic cancer. HISTOPATH diagnosis = **non-necrotizing granuloma** + infiltrate of **multi-nucleated giant** cells, epithelioid histiocytes, lymphocytes, and plasma cells.

Differentials = **R/o inflam cancer**, other granulomatous diseases: TB, sarcoidosis* of breast, Wegner's. Histoplasmosis, Actinomycosis, foreign body reaction, fat necrosis, IgG4 mastitis. *Fever, arthritis, or erythema nodosum = Lofgren's syndrome. This is a sterile condition and even with corneybacterium antibiotics won't penetrate. Best evidence supports steroids – trial into topical. Otherwise, prednisolone. Methotrexate also used. Can offer surgery. May recur with both tactics.

Diabetic mastopathy= lymphocytic mastopathy= sclerosing lymphocytic mastitis

Pre/peri-menopausal DMI. Masses often suspicious of a cancer – irregular, hypoechoic, post acoustic shadow etc. Treat conservatively once ruled out a cancer. So, need histology: triad LEK=

1. **Lymphocytic** inflammation
2. **Epithelioid myofibro**blasts
3. **Keloid** type **fibrosis**

Fibromatosis of the breast = extra-abdominal desmoid disease

Monoclonal stromal benign tumour. ESMO 2017 guidance on desmoids. They do not metastasise but are locally aggressive and often recur. **Association with APC/Gardners syndrome (also prior trauma and siliconomas) so must check for this**. So, in the long case if they have polyps/colon cancer and hard mass – r/o cancer as get breast cancers too but if benign think fibromatosis. Staining on IHC = **B-catenin**, SMA, Ki67, CD34. Molecular genetics diagnosis is useful as some Beta-catenin mutations confer higher risk of recurrence and response to mAB. Difficult to diagnose! Therapeutic options include excision with 1cm margin (tending to veer away from this as high-rate recurrence 9-15%, with over 1/3rd requiring re-exision), radiotherapy, tamoxifen and NSAIDs (not enough evidence to recommend but can try) and imatinib/sorafenib, NOTCH2- in trials.

BEWARE there is a condition = **Fibromatosis-like MBC** (may be associated with sclerosing lesions e.g papillary lesions or radial scars). This entity is one of the rare breast lesions in which a diagnosis of malignancy may rely solely on the demonstration of **CK expression on IHC** despite the absence of definite morphological features of malignancy. So, seems like fibromatosis but with CK +ve think this!

BIA-ALCL

Guidance paper from UK in JPRAS 2021. How common? In UK had 68 cases by 2020. Suspect if a new seroma or mass (meantime 8-10yrs). So about 0.1% experience late onset peri-implant swelling BIA-ALCL makes up 10% of these 0.1% so 0.01%. Some have lymphadenopathy and 15% associated mass. Ask about hx of malignancy e.g. melanoma, B symptoms (up to 9% will have) and take a FH – link to Li-Frau and BRCA! Common things are common so lymphadenopathy can be benign- silicon (snow-storm), malignancy – breast, lymphoma, sarcoma and mets e.g melanoma.

The main sample is sent as three separate specimens: USS guided aspiration (>50cc):

1. Haematological Malignancy Diagnostic Service (HMDS), 10cc in 2 purple-top EDTA tubes
2. Microbiology, 5-10cc in a white-capped sterile universal container
3. Cytology should receive the entire remaining volume (at least 50cc but can be over 500cc)

   cytospun -stain cells + cytoblocks. Their first job is to find cells (acellular = not BIA-ALCL – only 78% sensitive so if recurs low threshold for repeat).

Markers: **CD45 and CD2/3/4/8 T cells**. Should be **CD30+**, **ALK1 neg** (anaplastic lymphoma) and **B-cell negative e.g. CD20**. Must report to MHRA. Treatment = Tertiary = en-bloc total capsulectomy (4% rx pneumothorax). Prior PET-CT and MRI breast AND BM biopsy to check for involvement. Also, FBC and LDH. Stage II+ get systemic CHOP = cyclosphamide, doxrubacin (hydroxy), (onco)vincristine, prednisolone. Brentuximab vendotin = **antiCD30** available. TNM staging, modified Lugol's Ann Arbor. RadioTx if couldn't excise all. 2016 onwards = implant registry mandated.

Benign disease

Mastalgia

Classify into cyclical, hormonal medication, non-cyclical and non-hormonal and referred musculoskeletal.

- Reassurance not a symptom of cancer and often resolves– 0.4% mastalgia have cancer, 20-30% cyclical resolve (though 60% recur), 50% non-cyclical do resolve
- Supportive bra
- Weight loss and healthy diet including reducing caffeine and fat
- Change medications eg SSRI, OCP, HRT: HRT and breast pain - Womans' Health Initiative trial 16k woman combined HRT vs placebo – mastalgia x3 in cHRT. But subsequent study showed that at lower doses there was no increase.
- NSAIDs topical and oral
- Vitamin E supplements
- Gamma linoleic acid containing natural remedies star oil or evening primrose (EPO) not harmful
- Tamoxifen is not recommended as 1st line but can be used. Meta-analysis 2007 Cochrane –Bromocriptine works as does Danazol. EPO no difference. Tamoxifen achieved a relief 2-fold. Conclude Tamoxifen is associated with least side effects and should be the drug of first choice.

Gynaecomastia

- "I would manage them according to the ABS guidance: History including medication and elicit dx use and check bloods– LFTs, TFTs, Testosterone, bHCG and AFP"
- Under 25 P1/P2 – discharge, Over 25 P2+ image and biopsy
- Treatment – 10mg Tamoxifen, 1mg Anastrozole, Danazol. Surgery not funded.

Fibroadenoma and phylloides

- Aberration of normal development of the intralobular fibroepithelial cells. Fibroblasts and reactive epithelial (biphasic)
- Biopsy if over 25yrs or rapidly growing
- Not funded unless diagnostic excision (?phyllodes/B3+) or cosmesis AND over 4cm
- Phyllodes are difficult to distinguish as also intralobular, biphasic but can have a much more prominent stromal growth, leaf like pattern and borders may be irregular. WHO grades into benign, borderline and malignant phyllodes, according to 4 factors with 3 categories: Tumour margins/infiltrative, mitotic index, stromal cellularity and atypia, and stromal overgrowth.

B3 lesion

Breast MDT Screening working group paper Pinder et al 2018;

- Need 14g core followed by VAE of >4g for all except papilloma + atypia on Core = surgery.
- Distinction with all is the presence or absence of atypia, without atypia most lesions around 6-7% chance of associated malignancy therefore does not need further excision or surveillance

- Flat epithelial atypia as only has a rate of 11% so second line VAE is ok as is for Radial scar with atypia (17%)
- Highest risk lesion is **papilloma with atypia 33%** chance associated cancer and atypical ductal hyperplasia and atypical intraductal proliferation 28% so excise!
- Extra point – add to Sloane atypia project if doing surveillance

SURGERY

BCS surgery safe if give radioTx – NSABP and Milan trials. Radiotherapy reduces LR by 1/3 – in LN- 30 to 10% and LN+ 45% to 15% = EBCTG. In DCIS **B-17** and **EORTC 10853** ½ recurrence. Post Mx RadioTx in high rx = T4 or N2+. Trial = SUPREMO (T3NO/T2+N1 = IIB). Omit RadioTx = PRIME in over 65yrs good prognostic features. BOOST <40yrs, 23.9% to 13.5% LR reduction.

Ptsosis – Normal/Grade A is all glandular tissue and nipple sits above IMF. Grade B = Nipple at IMF a gland below. The both nippla and gland below IMF - Grade C = partially pointing down nipple Grade D= nipple looking at the floor. Glandular distortion sagging below IMF but nipple above IMF = pseudo

The BREAST-Q includes core breast cancer modules with four subscales:

1. satisfaction with breasts 2. psychosocial well-being 3. physical well-being 4. sexual well-being

Late 90s Krishna Clough grading of outcome from BCS:

Type I, asymmetrical breasts with no deformity of the treated breast

Type II, deformity of the treated breast, compatible with partial reconstruction and BCS

Type III, major deformity of the breast, requires mastectomy.

Therapeutic mammoplasty?

Discussion with patient of gains: TM include fewer radiotherapy-related side effects in large-breasted women and alleviation of allied symptoms associated with macromastia BUT fat necrosis and higher rate of wound dehiscence. Smoking, DM and high BMI increase risks of complications.

TM DOES not delay adjuvant therapy: TeaM study had a subset comparing Mx and TM: Complications lower in TM 21.0%; mastectomy 37.2%; mastectomy and IBR 35.6%. Does not delay adjuvant therapy (agrees with 2014 paper by J Harvey Manchester and Mike Dixon in Edinburgh).

Implant based Reconstruction

Oncoplastic Breast Surgery in the UK – NQIP, TeaM and iBra studies summarised in joint ABS/BAPRAS guidance 2021. Targets (match the NQIP):

Re-operation <10% within 3 months index procedure

Loss < 5% within 3 months index procedure

100% patients receiving full information and collection of satisfaction

What to consent for:

Risks and benefits of mesh assisted implant reconstruction:

Early and when to come back:

Gross swelling or blood in the drain – haematoma

Red breast – can be infection can be reaction – would want to see

Overt signs of infection or wound breakdown

Within 3 mnths (from iBra study):

Re-operation rate 18% **(aim <10%)**

Re-admission 18%

Infection 1 in 4

Implant loss 9% **(locally quote 5%)**

Longer term:

Revisional surgery including capsular contracture (historically 25% but with **ADM 13%**) BUT state evidence not great include effects of radiotx. ADM = BROWSE retrospective UK analysis. Symmetrising surgery if not already undertaking and no guarantee for funding.

BIA-ALCL – any new seroma or systemic symptoms would want to see. 1:24,000

Leak – change in feel/new lump/new skin changes

ADM – synthetic or animal product and is it absorbed, implant material etc. Benefit in reducing capsular contracture and protecting thin skin flap but may cause reaction.

Patient specific consent: IBra: loss 9%, infection 25%, re-admission 18% and re-operation 18%.

High BMI and smoking are associated with increases in all 4 complications

Prior radioTx was associated with infection only - National guidance states increased caps contr and overall a 1.5x rx complications. Longer op-time associated with re-operation rates higher, hence dual team operating. Age, NAC, bilateral surgery, indication for surgery, nipple-sparing, definitive fixed-volume implant, and type of reconstruction not significant. National guidance paper reports Mx weight >600g associated with increased infection. To spare the nipple? Cochrane review 2016 reported very poor evidence. ABS agree that there is not the evidence to say either way. In cancer surgery can take a shave behind nipple.

Oncoplastic techniques that are synonymous

Dohnut = Round block. Central qudrantectomy with a skin flap to replace nipple originally grisotti described, key is that the incision is medial. IMF needs to be 8cm away. If not you can modify the grisotti, taking it more laterally. Equally if you want to combine with a vertical mastopexy, make it straighter. It is just a dermogladular flap random.

Axilla

1-2 SNB +ve – options to discuss are:

1. No treatment Z011 trial and B=04 historically seems safe – or enrol in POSNOC (now closed!) Especially if you have an oncotype patient who is going to get chemo and endocrine.

2. Ax RadioTx – AMAROS = equivalent recurrence yet ½ lymphadenopathy (11% vs 23% at 5yrs)

3. Clearance – Oncologists wanting to upstage to push more radioTx (SUPREMO also) so e.g. if tumour is borderline but the LN would qualify them then may push for this.

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Basic anatomy and physiology

Lat dorsi

3 functions – adduction, internal rotation and extension of arm. NV- thoracodorsal artery from subscapular = 3rd part of axillary artery. Nerve from post cord = solely motor. Veni commitantes x 1.

Origin – iliac crest, thoracolumbar fascia from and spinous process T7-T12, tip scapula. Tendon inserts intertrabecular grove humerus between pec major and teres major.

Pec major and minor

Pec major - **A**dduction, **I**nternal rotation, **E**xtension (sternocostal head = ES) PLUS Flexion (clavicular head). Artery – major – Thoraco-acromial + perforators from internal mammary. Two origins: sternocostal 1-6 and medial clavicle. Inserts into lateral lip of the greater tubercle of humerus.

Pec minor Thoracoacromial a. (pectoral and deltoid branches) (2nd part axillary artery), superior thoracic a. (1st part axillary artery) and lateral thoracic a (2nd part axillary artery) – think makes sense as its deeper so closer to these arteries. Nerves mainly **medial** pectoral then communicating with lateral pectoral via ansa pectoralis overlying on pec minor. Origin = Anterior costal cartilages of ribs 3-5. Insertion: coracoid process of scapula. Mainly a stabilizer of the scapula and accessory in resp. Acts with serratus anterior to allow the full range of scapula movement. Bulky pec minor- TOS.

Serratus anterior and long thoracic n. injury

1-8th rib origin and inserts into the anterior surface of scapula (medial border).

- The first 0-15˚ of abduction is produced by the supraspinatus (C5/6 suprascapular n. sup.trunk)
- The middle fibres of the deltoid are responsible for the next 15-90˚ (axillary n post cord)
- Past 90˚, the scapula needs to be rotated to achieve abduction – that is carried out by the trapezius and serratus anterior.

So classic description may not be the **winged scapula** but rather than cannot lift arm beyond 90 e.g. lifting child in the air. Nerve is the long thoracic coming directly off C5/6/7 roots of brachial plexus. What can you do if injury – notice intra-op and complete neurotmesis – repair! Post op – while up to 13% reported deficit on EMG post ANC, **only 2% persist past 1yr** i.e. majority are neuropraxia. So, first line is physiotherapy and OT. Nerve grafting can be tried (e.g sural or medial/lateral antebrachial cutaneous n). If fails, then a transfer of the pec major tendon to the scapula (you keep the clavicular origin but loose the sternal head which switches with the tendon- so loose contribution to arm extension)! If that fails, then Scapulothoracic fusion is a salvage procedure performed to stabilize the scapula on the thorax. It involves the fusion of the medial border of the scapula to the underlying 3-5 ribs.

Axilla

Is a dome shaped region, with a apex and 4 walls. Apex clavicle/scapula/1strib. Borders – SA + Ribs 1-4, lateral humerus coracobrachialis and short head biceps brachii, pec major and minor anterior wall and clavicopectoral fascia, posteriorly the LD/Subscapularis/Teres major and. Recall quadrangular space between T major and minor and posterior to subscapularis divided by long head of triceps (medial border) transmitting the axillary nerve and post circ. humeral artery as they curl posteriorly around the surgical neck of humerus which is the lateral border. Medial to this space is the triangular space teres minor/major/long head where scapular branches pass from the subscapular artery.

Artery – starts as subclavian passes under first rib and ends as exits at the inferior edge of Teres major. Divided by pec minor. 1st part, 1 branch = superior thoracic, 2nd part, 2 branches = thoracoacromial (gives pectoral branches) and lateral pectoral artery (LTAP running along the border of pec minor), 3rd part, 3 branches = ant+post circuflex humeral and SUBSCAPULAR (largest) – Thoracodorsal with branch to Serratus and Lat Dorsi and intercostal perforators.

Local flap and anatomy

TD based pedicles: TD comes off Subscapular artery and first branch = to Serratus anterior. Then divides into two branches – descending vertical and horizontal. These branches give numerous perforators to the skin. Anatomic studies on cadavers have shown that the vertical intramuscular branch provides 2-3 cutaneous perforators.

How do you do a LD:

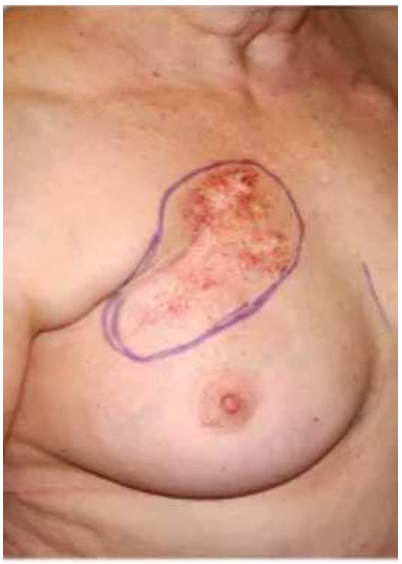
- Mark up – Iliac crest, paraspinal, tip of scapula, anterior axillary line, Breast incision and then the skin flap approx. 6-9cm wide and 20-26cm long ensuring that it comes together
- Pre op brief the team including moving the patient and anticipated steps – e.g. I ask for quiet when I am doing the pedicle
- Give abx, ensure there are flowtrons and the patient is being actively warmed with a warming matt with a bear hugger. Position the patient lateral with stomach support and arm above in trough and pillow between legs.
- Incise down to Scarpa's fascia, define the superior then medial (thoracolumbar fascia) then inferior boundaries of my dissection, and then raise the LD muscle, careful haemostasis, clipping any large perforators.
- Divide the attachment to the scapula and then the teres major so you simply have the thoracodorsal pedicle then tunnel to the anterior chest wall.
- Haemostasis, drain, quilt and 2-layer closure. Dress and then turn the patient to supine.

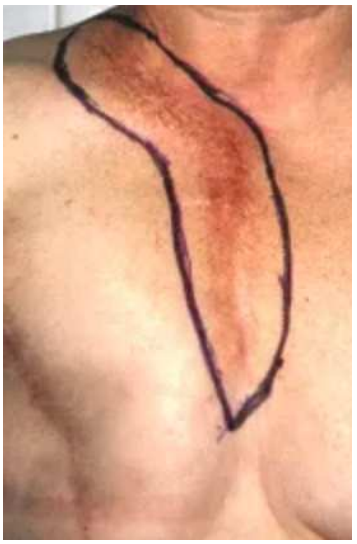
Blood supply of the breast (relevant for TM/reduction) – see pictures!!! Essentially, internal thoracic and 1-4 perforators with 2-3 being most reliable. Lateral thoracic artery and perforators. Posterior and anterior intercostal artery perforators to the medial and lateral breast respectively.

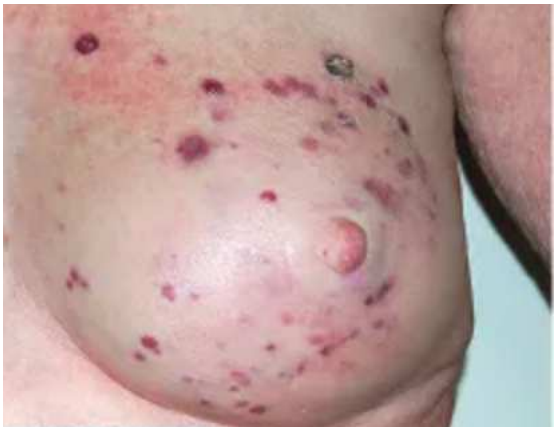
Cases that have come up in FRCS

Radiotherapy Necrosis and Basics of Radiotherapy

In the long case you may get this as a long case with a photo, comparing the below:







Notice the hypopigmentation and the telangiectasia that have become confluent and the retracted tissue in the 1st photo – this is radiation necrosis. In the second notice this is in the field where supra-clavicular radiotherapy has been given. In the 3rd one it is more nodular over the breast – angiosarcoma as discussed above. Remember in anyone with a history of breast cancer to rule of recurrence also!

What is radiotherapy?

Ioinising radiation (any energy sufficient to detach electrons from molecules) causes DNA damage and the release of reactive oxygen species. In the context of cancer, these cells are less able to readily repair DNA and this cumulatively this causes cell death. This can be given in both benign and malignant setting, with curative, adjuvant or palliative intent. It can be delivered as external beam using a linear accelerator, locally such as brachytherapy or SIRT or targeted through conjugates eg iodine (graves or thyroid cancer) or peptide receptor in NETs. The dose absorbed by the tissue is recorded in Grays. Hypofractionation is a term used to describe delivering higher dose over less sessions or dose concentrating.

What factors affect toxicity?

Radiotherapy toxicity is very varied and largely depends on site, dose, method of delivery and patient factors. Patient factors include any state where microvascular injury is predilected, for example smoking, diabetes, vasculopathy and vasculitidis, including SLE. Connective tissue disorders such as scleroderma are a contraindication, as is Li-Fraumeni as you cannot repair the DNA in normal cells so will develop malignancy.

Toxicity can be graded 0 to 4. Based on pigmentation (hypo or hyper), pain, erythema, puritis, de-squamation, tissue oedema and fibrosis.

Grade 1: No pain and all the above will be mild with only mild thickening of the tissue.

Grade 2: Marked changes will be seen with intense puritis, moist desquamation, telangiectasia, erythema++, pain, and the tissue will be moderately oedematous. May have superficial ulceration.

Grade 3: Think grade 3 burn, painful intense erythema, intense pruritus that interfere with daily activities, moist desquamation, ulcerative dermatitis, ++ confluent telangiectasia, ulcerations >2 cm.

Grade 4: general exfoliative ulcerative or bullous dermatitis, full-thickness skin necrosis with spontaneous bleeding.

The issue in cases where there is extensive involvement is going to be that there is tissue loss precluding simple mastectomy so you will need to replace this – eg LD, +/- skin grafting or free flap. Example answers:

"While I would want to rule out a recurrence, this looks like radiation toxicity with radiation necrosis. A key issue is that there is an extensive area affected and therefore this cannot simply be excised and I would need to consider options for tissue replacement, for example a LD. If a recurrence were not found, I would discuss this case in the MDT and involve plastic surgery to consider all the options for the patient. If recurrence were found, this is a different scenario and I would want to CT stage this lady and further treatment would depend on the outcome of that"

"While I would want to rule out a recurrence, this appearance is very worrisome for radiation induced angiosarcoma, which presents most commonly 8 years following radiotherapy. I would want to involve the sarcoma team but there are three key issues here to consider: Firstly, if she is suitable for surgery there is clearly going to be a need for tissue replacement given the extent of resection needed to get a therapeutic resection, yet the 50% recurrence rate and only 50% 5yr survival for these patients complicates eligibility for complex reconstruction such as free flaps. Therefore, her option may need to be a LD. Secondly, the sarcoma team prefer that a dyed biopsy tract is present, so I would not necessarily rush to do a biopsy without speaking with them first. Fortunately, we do have medical photography and online discussion with the team can be done promptly. Thirdly, as these are aggressive this may have already metastasized to the lung and CT staging is needed to confirm or refute this.

Paget's

Picture of a nipple with de-squamation ie ulcer or scabbing.

Key here is that it starts on the nipple and involves the nipple, whereas eczema is on the skin/areolar. Pagetoid is simply a term in dermatology that describes cells that are upwards spreading- so the pagetoid cells are in situ ductal cells that spread into the epidermis causing ulceration and crusting. The pagetoid cells are giant cells that have clear cytoplasm and pleomorphic nuclei – google they are obvious! If not mass to biopsy, then can do imprint cytology ie smear the slide in clinic, shave biopsy or a punch of the nipple. Underlying DCIS or Invasive disease present in 95% of patients. They are more commonly ER- and HER2+. Not common so not any RCTs to

inform practice. Treatment – central excision +/- displacement e.g. grissoti or replacement e.g. local flap, mastectomy.

Extra – epidermotropic theory is most widely accepted whereby migration along the basal layer of the ducts is supported by DCIS and keratinocytes acting on the HER2 receptor. Another theory is that this is transformation of keratinocytes (tolk cells)– argument for the 5% that do not have associated DCIS.

Inflammatory breast cancer

This is often used interchangeably with any T4 disease but is a distinct entity (T4d) in which there is lymphatic obstruction in the breast which causes breast lymphoedema – thickened, peu d'orange (fibrotic reaction), erythema. Don't get confused if they show you an ulcerating breast cancer that yes is T4 but is not necessarily inflammatory! Also beware breast mastitis/ abscess, the latter can be peri-puerperal or non-puerperal (lactational or non-lactational). It will cause similar features to an inflammatory cancer and the key is to re-assess them and image again. One difference is in the distribution – mastitis is more commonly SAR and inflammatory cancers are more common in the outer breast. Stage all (BJS 2020 4k patient cohort study = 85% LN+ at presentation). Not much in the way of tumour biology – they can be anything. Advocate for NAC in the MDT in inflammatory breast cancer thought the outcome will be the same as predicted by biology ie hope thay are HER2+ ER-!

Advanced ulcerating breast cancer in old lady

Full assessment including NABCOP frailty index and holistic approach. Often, they are indolent, slow growing ER+ cancers and can respond really well to an aromatase inhibitor so if willing try this then reassess. Can undertake excision for local control under local anaesthetic if need be but not axillary surgery.

Ulcer on the breast, medical ward and doesn't look like a fungating tumour

This has come up for a few folks now. If it looks like a simple ulcer, then say that! Think about factors – pressure damage, arteriopath, diabetic. Examine – is there an underlying mass and exclude malignancy with an USS. Optimise and manage with tissue viability team +/- diabetes team. Debridement can aid – if it is over-granulated you need granulation tissue to heal so to debride it is fine. Maybe not on the ward but could bring them to plastics department outpatient clinic where nurses can also dress this. On the ward it should be monitored as you would any other ulcer. Hopefully they won't but what about dressings: Cochrane review 2017 had 20 categories and concluded that there was no good evidence favouring any. Principles of the dressing are – absorbent and prevent moisture and maceration (especially hydrocolloids), promote healing, prevent infection either via a barrier or with added antimicrobial effects (honey, iodine impregnated and silver). Remember nothing will heal unless you have granulation tissue! Granulation tissue is actually super complex and has a constant balance of proliferation, scarring and remodelling and contracting. It is contractile (myofibroblasts), immunoprotective (monocytes an macrophages present), and a key cell is the endothelial cell- angiogenesis, chemotaxis, remodelling, matrix degradation and remodelling! Keratinocytes are needed for re-epithelialisation. Hence why those on steroids etc do not heal as all these processes are inhibited!

A summary of everything including rarer stuff! Originally collated by Miss Amy Robinson.
Last updated: May 2023

Male Breast Cancer

Don't overcomplicate it – same as women. Need Genetics and treat with Tamoxifen.

Pregnancy

No higher rx recurrence compared with age matched controls and based on the biology.

Aim to do mastectomy as cannot give radiotherapy and cannot breast feed/ be close to the baby

Chemo- aim to wait till second trimester when risks are lower but yes give it. Do not give tamoxifen or Herceptin in pregnancy nor when breast feeding. Less evidence base for taxanes.

JOINT DECISION MAKING!!!

Do not forget that termination is an option in first trimester – involve obstetrics. Green top recommendatiosn by RCOG and 2015 review provides guidance.

Young patient

Do not forget all the usual but also, genetics and fertility considerations if going for chemo. Likely to be TN for NAC – pembroluzimab + carboplatin + paclitaxel then capecitabine if not cPR.

Nipple discharge

Take a full history! Include medications and anything additional taken. Rate of cancer in physiological, puerperal (up to 2 years) and bilateral discharge breast cancer rate is 0% in small cohorts and certainly no more than 3%. Bloody discharge (except puerperal) rate of cancer is at least 11%. Review and offer hadfield's or surveillance USS if imaging normal and does not want excision (PPV Mammo only 46%). ABS "MRI can be considered if USS/mammo normal" so e.g., in a young woman with continued bloody discharge unilateral, normal imaging and wants to breast feed in the future.