THANZ
Thrombosis & Haemostasis society of Australia and New Zealand

Scientific Workshop

Perth Convention & Exhibition Centre
Trade Display: Meeting Rooms 2 & 3
Workshop: Meeting Room 1
Perth

19th October 2019

Thrombosis and Haemostasis Society of Australia and New Zealand.

Previously named the Australasian Society of Thrombosis and Haemostasis (ASTH) is now the Thrombosis and Haemostasis Society of Australia and New Zealand (THANZ) was established in 1994. The Society represents approximately 200 clinicians, scientists and other health professionals committed to promoting and fostering the acquisition, exchange and diffusion of knowledge and ideas relating to normal and abnormal haemostasis. The Society serves as a forum for bringing together a broad array of disciplines, which relate to bleeding, thrombosis and cognate fields.

The THANZ Mission Statement

Promote excellence in clinical care for people with clotting and bleeding disorders.
To lead education and training of scientists and clinicians in the field;
To foster innovation through research, discovery and clinical trials;
To advocate and develop policies that improve health outcomes.

Membership Privileges

Newsletters (three per year)
Notices, booklets, flyers or brochures of interest to members
Copies of Media releases and letters to members from Council
Invitations to Society presentations and seminars
Attendance to Annual General Meetings
Nomination for a Council position
THANZ Medal competition eligibility at ASM (45 years of age and under)
Voting rights at Council elections, AGMs and subcommittee meetings
Access to the ASTH Website and Member's only area, including Discussion group
Discounted registration fees for ASM and Scientific Workshops

Further information
THANZ Secretariat
Email: info@thanz.org.au

8:00 am Registration and Tea/Coffee

9:00 am Welcome: Grace Gilmore (Chair)

9:10 am Joseph Rigano (Melbourne ï Australia)
Evaluation of DOAC Stop to eliminate the interference of DOAC on Thrombophilia assays.

9:30 am Nora Lee (Melbourne ï Australia)
The BIOTEL risk model is a strong predictor for thromboembolism in lung and GI cancer ï further insights from corroborative studies with global assays and selected biomarkers

9.50 am Freda Passam (Sydney ï Australia)
Use of Microfluidic Chips in Thrombosis and Haemostasis.

10.10 am Lisa Kaminskis (Perth ï Australia)

Case Study ï The Big M

10:30 am ***MORNING TEA and TRADE***

11:00 am Dianne Lovelock (QLD ï Australia)
Acquired Haemophilia A-A laboratory perspective.

11:20 am John Balendra (Perth- Australia)
ñCan I have some Prothrombinex, Please?ò

11:40 am Paul Zerafa (QLD ï Australia)
Acquired Factor XIII inhibitor causing a significant bleeding phenotype

12:00 pm Poster Session

12:15 pm ***LUNCH and TRADE***

1:15 pm Prof Midori Shima (Naraï Japan)
The Impact of Clot Wave Analysis for Diagnosis and Treatment of Haemophilia

2:15 pm Liane Khoo (Sydney ï Australia)
Short and Long Acting FVIII

2:35 pm Stephanie PÕNg (Perth - Australia)
The Use of ROTEM and CAT to monitor new therapies.

3:00 pm ***AFTERNOON TEA and TRADE***

3:30 pm Matthew Linden (Perth ï Australia)
Platelet Function in non-severe burns injury.

3:50 pm Yusra Harahsheh (Perth- Australia)
Utility of OpenArray platform for determining differences in microRNA expression profiles in critically ill coagulopathic patients with and without onset of thromboembolism

4:10 pm Jennifer Curnow (Sydney ï Australia)
Thrombosis and Haemostasis in Acute Care Unit of Study, University of Sydney

4.25pm Closing Remarks/ Poster Prize

4:30 pm SUNDOWNDER and TRADE (until 5:30 pm)
Sponsored by THANZ

THANZ would like to thank the following sponsors who have made the 2019 THANZ Workshop possible

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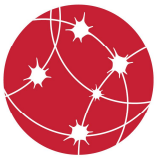

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Evaluation of DOAC Stop to eliminate the interference of DOAC on Thrombophilia Assays

Joseph Rigano

1Complex Haemostasis Department, Austin Heath, Heidelberg, Melbourne, Australia

Direct oral anticoagulants (DOACs) are known to interfere with thrombophilia assays such as lupus anticoagulant (LA), antithrombin (AT), protein C, protein S and activated protein C resistance. The impact of DOACs on result interpretation can cause misdiagnosis and clinical consequence. Interruption of anticoagulation for the purpose of thrombophilia testing exposes patients to an increased risk of thrombosis. We aim to evaluate DOAC-Stop® (Haematex Research, Australia) to eliminate the interference of DOACs on thrombophilia assays. 48 DOAC treated patients, 56 LA positive patients, 42 LA positive patients spiked with DOACs and 33 normal controls were enrolled. AT activity, dRVVT screen and confirm, APTT and plasma concentrations of DOACs were assayed using HemosIL® reagents on the ACL TOP CTS 500 analyser. A significant decrease in dabigatran, rivaroxaban and apixaban plasma concentrations was observed in DOAC treated patients following the DOAC-Stop® procedure (251.4 to 2.6 ng/mL (p = 0.004), 223.9 to 4.1 ng/mL (p < 0.0001) and 255.6 to 2.2 ng/mL (p < 0.0001) respectively). Similar results were observed in LA positive patients spiked with dabigatran, rivaroxaban, apixaban and edoxaban (350.1 to 1.3 ng/mL (p = 0.02), 395.4 to 3.5 ng/mL (p = 0.01), 388.3 to 1.7 ng/mL (p = 0.02) and 361.9 to 3.1 ng/mL (p = 0.005) respectively). Prior to the DOAC-Stop® procedure, false positive results for LA assays were observed in all DOAC treated patients. Following the DOAC-Stop® procedure all DOAC treated patientôs results were negative for LA assays. Following the DOAC-Stop® procedure, LA positive patients spiked with DOACs remained positive for all LA assays. A significant overestimation of AT was observed in all direct Xa inhibitor treated and spiked patients following the DOAC-Stop® procedure. There was no significant difference in LA positive and normal control patients following the DOAC-Stop® procedure. The DOAC-Stop® procedure is effective at eliminating DOAC interference on thrombophilia assays to allow the accurate interpretation of results in patients receiving DOAC therapy.

Table 1.

	DOAC Treated LA Negative Patients (n = 48)						DOAC Spiked LA Positive Patients (n = 42)								LA Positive Patients (n = 56)		Normal Controls (n = 33)	
	Dabigatran (n = 12)		Rivaroxaban (n = 23)		Apixaban (n = 13)		Dabigatran (n = 7)		Rivaroxaban (n = 17)		Apixaban (n = 11)		Edoxaban (n = 7)					
	191 ng/mL (22-870)		205 ng/mL (50-454)		212 ng/mL (109-604)		75 ng/mL (45-141)		137 ng/mL (75-259)		222 ng/mL (143-355)		241 ng/mL (138-372)					
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
	DOAC-Stop®		DOAC-Stop®		DOAC-Stop®		DOAC-Stop®		DOAC-Stop®		DOAC-Stop®		DOAC-Stop®		DOAC-Stop®		DOAC-Stop®	
dRVVT Screen (ratio)	2.42	1.09	1.77	1.00	1.41	1.05	4.78	2.38	2.96	1.72	2.65	1.84	3.01	1.49	1.78	1.85	0.96	0.99
	p = 0.001		p < 0.0001		p < 0.0001		p < 0.0001		p < 0.0001		p = 0.005		p = 0.0008		p = 0.14		p = 0.15	
dRVVT Confirm (ratio)	2.34	1.05	1.41	0.97	1.73	1.01	1.97	1.09	1.36	1.05	1.46	1.09	1.94	1.02	1.00	1.01	0.94	0.95
	p = 0.001		p < 0.0001		p < 0.0001		p < 0.0001		p < 0.0001		p = 0.0005		p = 0.0006		p = 0.16		p = 0.19	
dRVVT Screen/Confirm (ratio)	1.04	1.03	1.26	1.03	0.83	0.97	2.42	2.18	2.17	1.65	1.80	1.68	1.56	1.46	1.77	1.82	1.02	1.05
	p = 0.51		p = 0.0004		p = 0.0007		p = 0.01		p < 0.0001		p = 0.03		p = 0.02		p = 0.72		p = 0.11	
APTT (seconds)	59	31	41	34	39	32	132	63	83	64	96	80	66	45	72	73	32	34
	p = 0.0006		p < 0.0001		p = 0.0008		p = 0.0005		p = 0.003		p = 0.008		p = 0.002		p = 0.38		p = 0.17	
Antithrombin (%)	107	106	127	107	128	110	111	108	127	113	122	98	121	106	112	113	102	104
	p = 0.39		p < 0.0001		p < 0.0001		p = 0.33		p < 0.0001		p = 0.004		p = 0.0005		p = 0.48		p = 0.23	

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The BIOTEL risk model is a strong predictor for thromboembolism in lung and GI cancer – further insights from corroborative studies with global assays and selected biomarkers

Nora Lee .Marliese Alexander[1], Ray Dauer[2], Cindy J O'Malley[3], Dimitra Savva[1], David Ball[1], Ben Solomon[1], Kate Burbury[1]
1 Peter MacCallum Cancer Centre, Melbourne, Victoria; 2 Eastern Health, Melbourne, Victoria; 3 RMIT, Melbourne, Victoria

Background
Cancer associated thromboembolism (CA-TE) is a frequent yet preventable cause of morbidity and death. TE risk can be effectively reduced by pharmacological thromboprophylaxis (P-TP). However, the heterogenous nature CA-TE and competing bleeding risk requires a more stratified approach. The BIOTEL-TE risk model (BRM)** demonstrates high sensitivity, specificity and potency for TE risk stratification and appears superior to existing models.
Aims
Assess the independent and corroborative value of global haemostatic assays and selected biomarkers in conjunction with BRM in patients undergoing chemotherapy +/- radiotherapy.
Methods
Biomarkers of interest: VWF-Ag, FVIII, PF1+2, FM, PPL, TAT and thromboelastography; ETP (BIOTEL only)
Timepoints: pre treatment, months 1,3,6,9,12 with clinical correlation.
Comparison of biomarker levels in patients with TE vs no TE. Uni- and multivariate analysis for TE risk prediction (Fine and Grey), death risk (Cox proportional hazards) and for overall survival. Significant predictive markers were compared to BRM for corroborative value.
Results
Prothrombotic profiles^^ were seen in over 60% of patients particularly in the first 3 months of treatment, persisting up to 12 months.
Correlation with BRM risk grouping (low vs high) was best seen with median baseline TAT (2.1 vs 4.2ug/L p = 0.0025). ETP levels according to BRM group did not reach statistical significance but risk stratified for TE. Significant thresholds included ETP peak≥254 nM+ ETP≥1930 nM*min (p=0.004), ETP≥1930nM (p=0.024), velocity index ≥69, peak ETP≥254 + α2m≥9 + VI≥71 + TAT Month1 ≥4.2ug/L (p<0.001) despite falling within normal expected ranges[++]. No biomarkers were consistently predictive across cancer groups.
Conclusion
The BRM is a potent risk prediction model that is rapidly accessible in a clinical setting. Selected biomarkers and global haemostatic assays tested in this study demonstrated a "thrombotic profile' concordant with BRM and predictive value for TE and death. Further investigation of BRM is underway in the TARGET-TP teletrial.


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Use of microfluidic chips in Thrombosis and Haemostasis

Freda Passam

Microfluidics is the use of devices that apply fluid flow to channels smaller than 1 millimetre in diameter. Microfluidic flow devices (chips) mimic the rheological conditions found in various physiological and pathological settings in vivo and have broad applications in thrombosis and haemostasis.

Firstly, they have become an integral tool in research to study the function of blood cells under flow and their adhesion to substrates or other cells. The channels can be designed to include features e.g. stenosis, that simulate the circulation in stenosed vessels. Microfluidic chips facilitate cell imaging and tracking for the study of cell-cell interaction such as in thrombo-inflammation.

Secondly, microfluidic chips offer the potential of diagnostic applications in thrombosis and haemostasis (lab-on-chip). Microfluidic devices can reduce reagent consumption, integrate and automate assays and lead to the development of point-of-care devices.

Thirdly, the vessel-on-chip offers a platform for testing new drugs in vascular biology and a tool for personalized medicine.

However, the diversity and ease of production has led to a wide range of chip designs, along with differences in laboratory protocols. International standardization bodies are working to standardize research protocols and clinical flow-based assays of haemostasis and thrombosis.


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Case Study – The Big M

Lisa Kaminskis

Coagulation Lab, Fiona Stanley Hospital, WA

The presence of a paraprotein (or M-protein) in patient plasma can be an important source of laboratory interference, presenting challenges to many departments throughout haematology due to their highly variant and unpredictable nature. One of the biggest challenges to overcome is recognition of the inhibitory M-protein in the troubleshooting process prior to exhaustion of patient sample or laboratory time. These paraproteins can be present in patients with multiple myeloma or MGUS (monoclonal gammopathy of undetermined significance) and there have been a number of these cases requiring extensive investigation in the Coagulation Laboratory at FSH over the last 12 months. The inhibitory M proteins can cause interference and prolongation in the coagulation profile, misleading results due to non-linearity in factor studies and heparin like effect in TCT and lupus screening. Deciding which results are accurate and how to report them can be time consuming and a challenge that is best handled in co-operation with your clinical haematology team.
This collection of case studies aims to raise awareness of these M proteins and suggest a guide toward how to approach them in the laboratory. This may allow the inhibitory proteins to be detected and dealt with more efficiently and with less uncertainty, with a hope of less lab time and patient samples being lost to The Big M.

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Acquired Haemophilia A – A laboratory perspective

Dianne Lovelock

Aim

Acquired Haemophilia A is one of the most challenging problems encountered in the diagnostic coagulation laboratory. We will discuss some of the difficulties in identifying acquired haemophilia A and in interpretation of results, and share some of our experiences with these patients.

Method

Case Review

Acquired Haemophilia A is a rare bleeding disorder which is characterised by autoantibodies directed against circulating factor VIII. Early identification of these inhibitors is key to optimising treatment and improving outcomes for patients. However, the presentation of these autoantibodies is heterogeneous, and detection and quantitation are often delayed. Interpretation of results can be difficult due to patient co-morbidities and conflicting laboratory results. We present three cases identified in our laboratory during a five-month period which highlight the diversity in clinical and laboratory presentation of patients with factor VIII autoantibodies. The first case is that of an 89-year-old male who presented with intracranial haemorrhage following a fall. The second case is an 84-year-old male who presented with a thigh haematoma. The final case is of a 69-year-old female with a history of chronic myelomonocytic leukaemia and historical lupus anticoagulant who presented with extensive bruising.

Conclusion

The laboratory diagnosis of and quantitation of antibody levels in acquired haemophilia A can be challenging due to the comorbidities of the patients, and the limitations of the coagulation assays used to identify these autoantibodies. Our cases highlight the difficulties faced by diagnostic laboratories in managing these complex patients.

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‘Can I have some Prothrombinex please?

John Balendra

Acquired Factor V Inhibitors are rare autoimmune phenomena with approximately 200 published case reports, seen previously with exposure to bovine thrombin used for operative haemostasis though now most commonly attributed to medications, malignancy, secondary autoimmune conditions and idiopathic cause. The clinical sequelae vary from an asymptomatic finding to fatal haemorrhage.

We describe the case of a 56yo female post-renal transplant for end-stage renal failure secondary to glomerulonephritis presenting with transplant failure due to progressive stenosis of the transplanted renal artery. Incidentally the patient was found to have a new finding of prolonged prothrombin time (PT) and activated partial thromboplastin time (APTT) which did not correct after 50:50 mix with normal plasma. The thrombin time (TT) was normal.

Further testing of common pathway factors identified a low Factor V assay (3%) and Bethesda Assay confirmed an inhibitor (6.5BU). Urgent balloon angioplasty of the stenotic renal artery was successfully facilitated with platelet transfusion.

The patient had resolution of coagulation findings and inhibitor on Bethesda Assay within two weeks without specific inhibitor eradication therapy (noting the patient remained on immunosuppressants including steroids) nor a significant bleeding event. Bovine thrombin exposure was excluded, and a definitive cause for the inhibitor was not determined.

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Acquired Factor XIII inhibitor causing a significant bleeding phenotype

Paul Zerafa, *J Beggs, P Wood.*
Pathology Queensland, Princess Alexandra and Royal Brisbane and Women's Hospitals, Brisbane, Q4102

A 35 year old Afghani refugee presented with an acute nephritis in May 2018. He proceeded to renal biopsy and developed a left perinephric haematoma post procedure. In July 2018 he developed spontaneous right psoas and retroperitoneal bleeds of uncertain aetiology. He developed a further right paracolic bleed and during investigation with CT scan showed a large thrombus involving both iliac veins and extending to the inferior vena cava. There was no personal or family history of a prior bleeding disorder.

Investigation for an underlying coagulopathy showed normal prothrombin and activated partial thromboplastin time. Fibrinogen was normal. An acquired factor VIII inhibitor was suspected but not found. Subsequent investigation revealed a factor XIII level of <0.01 U/L. The factor assay demonstrated non-linearity and a Bethesda assay demonstrated an inhibitor with an activity level of 2.9 Bethesda units.

As there was no active bleeding and despite the previous bleeding history he was anticoagulated with warfarin and developed no further bleeding sequelae. Based on the factor inhibitor, the patient was treated with prednisolone 1mg/kg and Rituximab 100mg for four weekly doses. He was given prophylactic cryoprecipitate for several weeks whilst on warfarin. Six months after the initial event the patient has had no further clinically evident bleeding, however the factor XIII level remained <1%. The patient subsequently acquired a high titre anti-nuclear and dsDNA antibody and a diagnosis of auto-immune nephritis was made. A factor XIII inhibitor diagnosis remains unclear with the auto-immune history suggesting an acquired inhibitor whilst the ethnic background is consistent with a hereditary deficiency in a high prevalence population.

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The Impact of Clot Wave Analysis for the Diagnosis and Treatment of Haemophilia

Prof Midori Shima

Department of Pediatrics, Nara Medical University, Kashihara, Japan

Some of the currently available coagulation analyzers have potential for continuous measurement of the changes in transmittance or absorbance during the clotting process in APTT or PT. By processing the data, clot waveform analysis (CWA) can be performed for the assessment of the global clotting function. In addition to qualitative evaluation by clot waveform, it possible to evaluate the clotting function quantitatively by various parameters. For example, the first derivative of the transmittance or absorbance reflects coagulation velocity and the 2nd derivative reflects coagulation acceleration. Clotting time (CT), maximum coagulation velocity (Min1), maximum coagulation acceleration (Min2) and maximum coagulation deceleration are common basic parameters. In 2013, we attempted to standardize the reagents, coagulation analyzers and parameters for CWA in the FVIII/FIX subcommittee of SSC (*Shima M et al. J Thromb Haemost 2013; 11:1417-20*). The CWA was useful for assessment of global clotting function of hemophilia patients. Furthermore, the CWA can be applied to the monitoring of the hemostatic treatment for hemophilia with inhibitor. We investigated the utility of the CWA for monitoring of bypassing therapies using modified trigger reagents with mixture of ellagic acid and small amount of TF (Elg/TF CWA). (*Haku J et al. J Thromb Haemost 2014;12:355-62*). We further attempted to apply CWA to the monitoring of FVIIIa mimicking bispecific antibody, emicizumab, which has been approved for the hemophilia A patients with inhibitor (*Nogami K et al. J Thromb Haemost 2014;16:1078-88*). Recently, we found that the coagulation velocity curve of hemophilia A is different shape from other clotting factor deficiency. Therefore, we established a new quantitative parameter, weighted center, of the coagulation velocity curve to assess the FVIII activity for prompt diagnosis of hemophilia, based on the APTT CWA. We defined weighted center in an area surrounded by the curve and baseline at X% of the peak height (Wx) and created 5 parameters. These parameters were used for template matching. We compared parameters of each plasma samples with those of 158 templates obtained from coagulation factor deficient plasmas of various severity and lupus anticoagulant positive plasmas. This template matching method using weighted center was useful for quick diagnosis of hemophilia A with single APTT measurement.

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Short and Long acting FVIII

Liane Khoo

Over the last 10 years, Haemophilia treatment has changed. Currently, in addition to recombinant standard half-life FVIII and FIX products, we now have a range of newer products to treat our patients. These include extended half-life products, using technologies such as PEGylating, Fc-fusion proteins, and albumin fusion. There are also antibody treatments (Emicizumab), non-factor replacement therapies (Fitusiran, anti-TFPI inhibitors) and gene therapy.

The paradigm has shifted from not only how we treat our patients, but also how these products are monitored in the laboratory. As such a “one assay fits all” approach to measuring factor levels is no longer applicable. This poses a challenge to the laboratories in obtaining accurate factor level measurements. This is because the modifications made to rFVIII and rFIX molecules to extend their half-life may alter the way they interact with the laboratory’s current APTT reagent and factor assay systems in unpredictable ways. When this occurs there may be unacceptably high or low estimates of plasma levels of replacement factor, so the laboratory must implement a strategy to achieve accurate product estimates to help guide clinicians in treating patients. These strategies may include use of alternative APTT reagents, use of chromogenic assays or maintaining existing methods coupled with use of correction factors.

This talk will provide an update on the results from some of the Australian wide field studies with the extended half-life factor products that was conducted in the various haemostasis laboratories across Australia. It will also provide a brief overview of some of the future laboratory studies planned.

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The Use of ROTEM and CAT to monitor new therapies

Stephanie P'Ng

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Platelet Function in non-severe burns injury

Matthew Linden

Background: Burn injury initiates an acute thrombo-inflammatory response leading to susceptibility to venous thrombosis and pulmonary embolism. Despite increased understanding of the impact of burn injury on haemostasis in the acute phase, little is known about long-term consequences of burn injury on haemostasis and thrombosis. Recent Western Australian hospital data has shown that all burn survivors, regardless of severity, are at an increased life-long risk of cardiovascular morbidity.

Aims: Here we investigate whether non-severe burn injury is associated with long lasting changes in platelet function.

Methods: In mouse studies adolescent CD57/BL6 female mice were given an 8% full thickness thermal contact burn (n = 6) or sham control (n = 6) and blood tested 28 days later. In preliminary human studies, blood was collected from adults with a non-severe burn injury (n=12) at 2 weeks and 6 weeks after non-severe burn injury or age/sex-matched controls (n=10).

Results: Collagen related peptide (CRP) stimulated platelet P-selectin expression was 1.2 fold higher in burn vs sham mice. In humans platelet response to CRP was higher two weeks (1.6 fold PAC1 binding, 1.3 fold CD62P expression), and six weeks (2.0 fold PAC1 binding, 1.7 fold CD62P expression) post burn. Formation of monocyte-platelet aggregates in response to thrombin receptor stimulation was reduced at 6 weeks (0.8 fold), but not at 2 weeks.

Conclusions: Using mouse and human data we show preliminary data to suggest that platelets remain hyper-responsive to collagen stimulation following a non-severe burn injury

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Utility of OpenArray platform for determining differences in microRNA expression profiles in critically ill coagulopathic patients with and without onset of thromboembolism

Yusra Harahsheh

[1]Department of Intensive Care Medicine, Royal Perth Hospital, Perth, Western Australia

[2]Medical School, The University of Western Australia, Crawley, Western Australia

Recent studies have investigated circulating miRNAs as potential biomarkers for identifying risk of thromboembolism with promising results. Critically ill coagulopathic patients are at risk of developing thromboembolism that cannot be predicted using standard clinical predictive tests or scores. Finding differences in miRNA expression profiles, however, may have utility.

Aim:

Our pilot study aimed to determine if differences exist between miRNA expression profiles in critically ill coagulopathic patients with and without the onset of thromboembolism.

Methods:

Blood was collected from 40 patients that developed an INR>1.5, aPTT>40s and/or platelet count >$150x10^9$/L, within 48-hours of ICU admission. RNA was extracted and converted to cDNA templates using TaqMan universal primers. 754 mature miRNA targets were analysed using TaqMan OpenArray plates. Targets with an AMP score <1.0 and a C_{RT} >30 were omitted from final analysis.

Results:

The miRNA expression profiles of patients who developed thromboembolism (n=10) were compared to those without (n=30). Twenty-nine miRNAs were upregulated and four were down regulated by greater than a 3-fold difference in the thrombosis group compared to the non-thrombosis group. Thirteen miRNAs (hsa-miR-423-5p, 302a-3p, 576-5p, 126-3p, 183-5p, 1200, 133a-3p, 515-3p, 646, 339-3p, 431-3p, 616-5p and 659-3p) were found to be significantly upregulated and none of the down regulated miRNAs were statistically significant.

Summary and Conclusions:

MicroRNA expression profiling using the OpenArray platform may be a helpful investigative tool for differentiating the risk of thromboembolism in critically ill patients with acquired coagulopathy. Larger validation cohorts are required to confirm these findings.

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Thrombosis and Haemostasis in Acute Care Unit of Study, University of Sydney

Jennifer Curnow

In 2016 the THANZ Education subcommittee began work on developing a post-graduate course in Thrombosis and Haemostasis. We have partnered with the University of Sydney to deliver our first Unit of Study in Semester 2, 2019. Thrombosis and Haemostasis in Acute Care (HAEM5001) is listed as an elective in the USyd postgraduate courses Master of Medicine Critical Care and Master of Surgery. A broad range of clinicians and scientists from Australia and New Zealand have contributed to the teaching modules. We have 48 students enrolled in this unit which is delivered and assessed entirely online.

An overview of the unit of study will be provided during this session. In 2020 we plan to offer the course to 'Short course students' which may be of particular interest to our Haematology trainees. Short course students study with the students enrolled in Masters degrees but are not required to attempt all assignments. They pay a reduced fee ($1500 early-bird or $1750 normal) and receive a Certificate of Completion.

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Posters

The Prevalence of Antiphospholipid Syndrome Criteria and Non-criteria Antibodies in Patients with Unprovoked Venous Thromboembolism
Joseph Rigano

Investigation of a prolonged APTT reveals a rare finding
Yvonne Brennan

Evaluation of the Nijmegen and CDC modifications to the Classical Bethesda Inhibitor Assay
Joseph Rigano

Evaluation of the Automated HemosIL® AcuStar HIT IgG Chemiluminescent Immunoassay for the Diagnosis of Heparin-induced Thrombocytopenia
Joseph Rigano

Bringing thrombin generation into the diagnostic setting: standardisation and establishment of normal reference intervals for a commercial thrombin generation system
Sarah Just

Do anti-β2GP1 antibodies increase shedding of platelet GPVI?
Yik Ho

Platelet function in paroxysmal nocturnal haemoglobinuria
Fathima Mohiyaddin Ayyalil

Clinical Outcomes in patients with Discordant Heparin-induced thrombocytopenia laboratory testing.
Zi Ng

Consumptive coagulopathy induced by Protthrombin Complex Concentrate (PCC) in Chronic Liver Disease.
Paul Zerafa

SAVE THE DATE

Blood 2020

The combined Annual Scientific Meeting of the:

HSANZ
Haematology Society of Australia and New Zealand

ANZSBT
Australian & New Zealand Society of Blood Transfusion Ltd

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